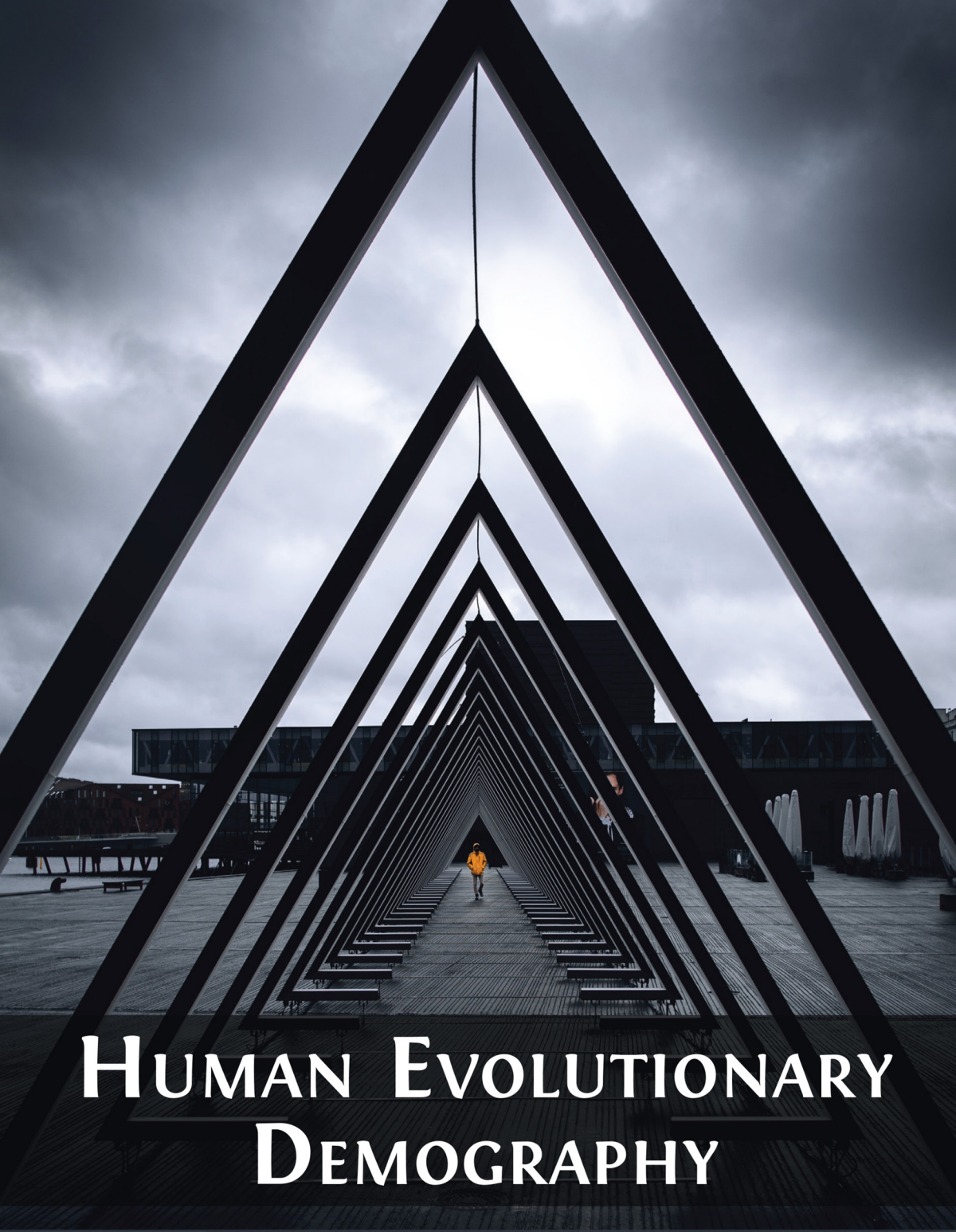

Human Evolutionary Demography

Edited By

Oskar Burger, Ronald Lee and Rebecca Sear

HUMAN EVOLUTIONARY DEMOGRAPHY

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We dedicate this book to James Vaupel, who did so much to promote the field of evolutionary demography

Contents

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We sincerely thank the many individuals who contributed to this volume by reviewing chapters; the book is undoubtedly stronger because of their efforts. We thank Gert Stulp for initial discussions. We thank all of our authors for being willing to contribute to this endeavour, for their fine contributions, and their patience as we put the volume together; their work is the reason we have been able to produce a volume showcasing the vibrant field of human evolutionary demography. Note that chapters were accepted for publication, and posted on the Open Science Framework (OSF), some time before the date of OBP publication, so references will reflect when the chapter was written. Date of posting on OSF is given in each chapter.

1. Human Evolutionary Demography: Introduction and Rationale

Rebecca Sear, Oskar Burger & Ronald Lee

Human evolutionary demography combines research in evolutionary biology with the study of human demographic patterns and behaviours. Evolutionary biology and demography share many conceptual features that give rise to a natural complementarity, such as a focus on the population as a unit of study and emphasis on aggregate processes that have implications for individuals. They also have distinct strengths that further this natural partnership. Evolutionary approaches are often top-down and theory driven, while demographic ones are more often bottom-up and driven by data and robust estimation procedures. We suggest that human evolutionary demography reflects these areas of overlap and complementary strengths while emphasizing at least two main objectives: understanding the role of evolutionary processes in shaping population-level demographic patterns (e.g., the evolution of age-specific patterns of mortality or fertility), and using an evolutionary approach to understand contemporary variation between individuals in demographic patterns (e.g., how and why does fertility respond to environmental influences, and vary between and within populations?).

Evolutionary demography is also inherently interdisciplinary. Interdisciplinary approaches are vital to furthering our understanding of the complex processes underlying demographic patterns, in part because such approaches can be a disruptive force challenging researchers to question assumptions and see the world differently.

The chapters in this volume demonstrate that the integration of demography and evolutionary sciences strengthens both. This recognition by an ever-growing number of researchers has resulted in such a successful body of research that we are now able to showcase this field in this edited collection, illustrating the vibrancy and diversity of research in human evolutionary demography.

Why does evolutionary demography matter?

> Dobzhansky famously observed that nothing in biology makes sense except in the light of evolution. Because evolution is driven by — and drives — birth and death rates, it is equally valid that nothing in evolution makes sense except in the light of demography. And to a considerable extent vice versa — much in demography, especially age-patterns of fertility and mortality, makes sense only in the light of evolution.
>
> — Vaupel, 2020

Why did we decide to create this collection? Because we share the opinion, neatly stated by Jim Vaupel (2020) above, that human evolution and demography are inseparable: evolution cannot

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be understood without understanding demography, and demographic patterns cannot be fully explained without evolution. Recognition of the gains that can be made by closer integration of these disciplines is steadily growing, particularly since the 1990s when several lines of research began to thrive which combined these disciplines (Carey and Vaupel 2005; Wachter 2008; Low et al 1992, see Figure 1). This volume aims to highlight to researchers interested in our own species what those gains might be, and to encourage further integration between disciplines.

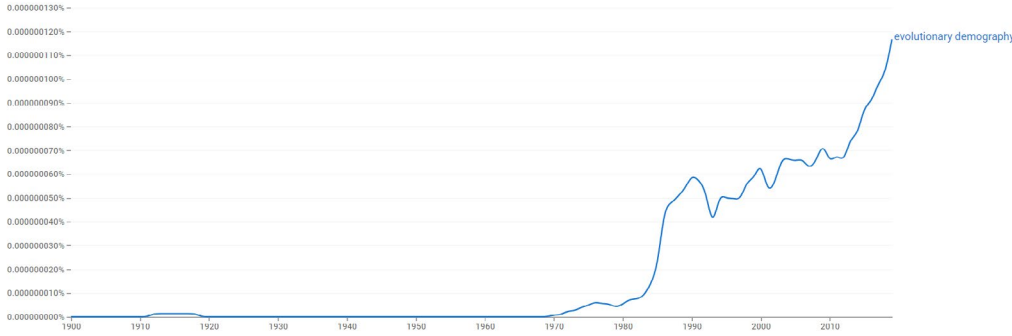

Fig. 1 Appearance of the term "evolutionary demography" in Google's NGram viewer, between 1990 and 2019.

Demography and evolutionary research are an obvious partnership because natural selection operates through differences in reproduction and survival, which are the two most fundamental drivers of population change. The role of demography in understanding aspects of natural selection and evolutionary processes is therefore clear, as the source of tools, techniques and insights into the analysis of demographic patterns. In addition, evolutionary researchers wishing to understand the multitude of social and cultural influences that underlie patterns of fertility and mortality, such as mating behavior, social organization, cooperation and competition, productivity, culture, investment in offspring, sibling rivalry and kin structures, can fruitfully draw on research on these topics in demography (as well as other social science disciplines).

Demography, too, benefits from evolutionary research, not least because an evolutionary approach necessitates crossing disciplinary boundaries. The processes underlying human demographic patterns and behaviors are highly complex. Lave and March (1993) consider the challenges of studying human behaviour to be so extreme that they lament '*God has chosen to give the easy problems to physicists*' [p. 2]. We believe that surmounting these challenges requires an interdisciplinary approach. Disciplinary silos impede progress because of the risk that researchers get stuck on particular tracks of theory, method, or ways of thought. Crossing disciplinary boundaries is a disruptive process, which has the potential to free thought, and is particularly important across the social and biological divide. Humans have evolved through the process of natural selection in the same way that every other species has. Acknowledging this is key to fully understanding our behaviour and demographic processes. Demography also provides a particularly fruitful arena for social and biological scientists to interact because of the 'biosocial' nature of fertility and mortality, involving not just the range of social influences mentioned above (and more), but also biological differences in skeletal structure, organs, endocrine systems, brain and immune systems. Uniting the detailed understandings of the

social sciences with insights from the evolutionary sciences about how our physiology, behavior and culture have evolved is a much more powerful way of analyzing and predicting human affairs than is doing social science without biology.

Yet the contemporary social sciences have typically shown little interest in applying research from the biological sciences to the study of human behaviour; sometimes strongly rejecting such attempts. This reaction has undoubtedly been influenced by the historical stain of eugenics and its link to human rights abuses, culminating in murderous Nazi racism. Interest in the application of biology to social affairs was in fact widespread in the early twentieth century because of the eugenics movement; a political ideology which argues that the biological inheritance of 'desirable' and 'undesirable' traits from one generation to the next is so simple that the human race could be improved through selective reproduction (Rutherford 2022). Several academic units for research on eugenic themes were set up during the early decades of the twentieth century; the establishment of the discipline of demography in the UK, for example, owes a significant debt to eugenic interests (Grebenik 1991; Langford 1998). But this political movement was based on faulty science and faulty social science, and began to fall out of favour in academia even before there was widespread condemnation of this ideology because of its human rights abuses.

Moreover, there are other reasons for the wariness of many social scientists to embrace biology. There is also concern that biological reductionism tries to explain human behaviour to the exclusion of cultural and social forces, and removes individual 'agency' from the equation. There are assumptions that biological explanations will simply have little power to explain much of the phenomena that social scientists are interested in, given that human affairs are so very variable over time and space, and therefore cannot be explained only with reference to changing gene frequencies (we discuss further below the misconception that evolutionary approaches are only about changing gene frequencies). Finally, there is concern that biological approaches are not sufficiently 'critical', in that they do not pay sufficient attention to biases introduced by power structures in academia that affect the production of research (though similar criticisms about a lack of critical thinking have also been levelled at some social sciences, including demography: Sigle 2021; Greenhalgh 1996).

All these concerns need to be taken seriously by those wishing to promote greater integration between the biological and social sciences, especially given that fears of a resurgence of eugenics have turned out to be valid (Panofsky, Dasgupta, & Iturriaga, 2021). Interest in this pseudoscientific endeavor never entirely left academia and has now edged back into the academic mainstream in the twenty-first century (Sear 2021; Saini 2019). Recent revelations about E.O. Wilson (a highly regarded scientist known for work on ants, conservation and other topics, who did so much to revive interest in recombining social and biological science in the 1970s) and his behind-the-scenes support for J. Philippe Rushton (who did so much to promote scientific racism) are a clear reminder of the impossibility of separating science and politics, and of the complex human interactions that underlie the production of research (Borello and Sepkoski 2022; Farina and Gibbons 2022). The solution to this resurgence is not, in our view, to reinvigorate calls to separate the social and biological sciences — such separation may have facilitated the recent resurgence in eugenic ideology. Instead, rigorous researchers from both sides need to work together to improve the quality of research that draws on both social

and biological research, in order to guard against the misuse of science and social science for political ends.

One of the aims of this volume is to highlight, with practical examples, how rigorous interdisciplinary research involving both social and biological science perspectives can further our understanding of human demography. It is hard to make sweeping statements about what contemporary applications of biology to human affairs look like, since there are now many ways of doing this, but this volume should also help dispel some misperceptions about 'social biology'. For example, it does not assume that the behavioural traits of interest to social science and policy are wholly genetically determined, nor that variation in these traits over time and space can be explained by genetic or biological factors to the exclusion of all other explanations. The study of links between genes and human phenotypic traits is still barely in its infancy, though we know enough to know that these links are typically very complex, so that it would be foolish to make confident statements about the over-riding importance of genes when explaining human behaviour or demographic patterns. Instead, genetic and biological research is considered complementary to social science, and evolutionary approaches often put significant emphasis on how environmental factors interact with genetic or biological factors to produce outcomes of interest in contemporary populations. There are also many different ways to apply evolutionary thinking to our species, some of which don't involve explicit consideration of genes at all (see Cully & Shenk's chapter), and some don't assume that natural selection is the only force that has shaped the evolution of human behaviour and demography (see the chapters by Orzack & Levitis and Colleran). Nor are evolutionary approaches confined to studying only traits that are currently adaptive; an evolutionary perspective can also be highly valuable when trying to understand patterns that don't appear to be easily explained from a fitness-maximising perspective, such as the demographic transition and contemporary low fertility (Borgerhoff Mulder, 1998; Stulp, Sear, & Barrett, 2016)

However, this volume is not only about the application of evolutionary biology to demography. Evolutionary demography encompasses a broad range of research, including the use of demography to inform evolutionary biology. This volume presents an overview of current topics of interest in evolutionary demography, and could be used as a higher-level textbook for illustrating questions of interest in the field, though it does not cover the basics of either an evolutionary or demographic approach.[1] There are also research areas relevant to evolutionary demography we do not cover — such as insights into evolutionary demography from the dynamic new area of ancient DNA research, or archaeological demography, and also contributions by economists on parental investment, research on the evolution of cooperation or the coevolution of human biology and culture. What we aimed to do with this volume was

1 For the basics of the evolutionary approach, see the first section ('Foundations') of Evolutionary Behavioral Ecology by Westneat and Fox (2010), and classic works in Life History Theory such as The Evolution of Life Histories by Stearns (1992) or Life History Evolution by Roff (2002). For demographic methods, the IUSSP's online teaching materials provide entry-level materials, and Preston & al's (2000) Demography is highly regarded for a more advanced approach; for excellent data visualisations of demographic patterns and trends, see the Our World in Data and Gapminder websites. For works that demonstrate how to combine biology and demography, Carey & Roach's (2020) recent Biodemography volume is an introduction to formal demographic methods with consideration of how these might be applied across species, including humans; and Hill & Hurtado's (1996) Ache Life History is an excellent introduction to the application of life history theory and demography to a human population.

to invite contributions from a range of researchers who have explicitly drawn on evolution and demography to inform their work. We hope the result gives an insight into what evolutionary demography is, and the wide scope of research within the field.

How did the field of evolutionary demography emerge?

As evolutionary demography is a merger of two meta-disciplines, we briefly consider each, in turn.

Demography is the study of population processes, which include fertility, mortality and migration (see Box 1 for definitions of terms used in demography). According to one of the most widely used textbooks in demography (Preston et al 2000): '*while the emphasis is on understanding aggregate processes, demography is also attentive to the implications of those processes for individuals*', a description that could also be applied to evolutionary biology. Demography is strong on statistical description, and is a discipline with considerable respect for data. Substantial investment has been made in developing data collection tools and techniques for accurately describing demographic phenomena: this is the 'core' business of formal, or technical, demography; sometimes contrasted with the 'rind' of social demography, which aims to understand demographic phenomena (Coleman 2000). To quote Preston (2020) again:

> demography maintains a well-deserved reputation for integrity & intellectual honesty that reflects a highly empirical orientation & closeness to process of data production. Demographic conversations are brief when assertions are based on flimsy evidence

Demography was closely linked to biology in the early days of the academic discipline (see Kreager's chapter for more detail on the historical connections between demography and evolutionary biology). Now, however, demography is primarily a social science, and social demography incorporates a wide range of conceptual frameworks from social science to understand why demographic patterns vary between and within populations. Demography has been referred to as an 'object discipline' or field of study, given that demographers are linked by an interest in fertility, mortality, migration and population structures, rather than united by any particular theoretical or ideological framework (Coleman 2000). One of the pioneers of evolutionary demography, demographer Jim Vaupel (2020), has said demography is an 'interdiscipline' due to its natural role providing a glue across fields. The fields that have contributed to demography are diverse, although some disciplines have affected demography more than others, notably economics (which has also influenced evolutionary biology) and sociology. Contributions from fields such as social anthropology are less embedded within the discipline, but nevertheless, calls have been made to incorporate both into demographic research in recent decades (Kertzer and Fricke 1997; Coast et al 2007).

Box 1: Defining terms within the demography side of evolutionary demography

Demography is the study of population size, structure and dynamics, and of the three components of fertility, mortality and migration that drive changes to population size, structure and dynamics. ***Mortality*** refers to deaths. ***Fertility***, somewhat inconveniently, is defined differently in biology and the social sciences. Demography, along with other social sciences, uses fertility to refer to the number of children born and fecundity to the capacity to conceive. Biology reverses the meaning of these two terms. Throughout this volume, the demographic definition will be used. ***Migration*** refers to population mobility, for moves over a relatively long period of time and distance. Little research in evolutionary demography has focused on migration (with a handful of exceptions, noted in Cully and Shenk's chapter) so we do not consider it further here.

At the population level, fertility and mortality are often measured by birth and death rates for each age and sex. From age-specific mortality rates we can calculate ***life expectancy*** at each age. e0 represents life expectancy at birth (the number of years a person can expect to live, given prevailing mortality rates), a commonly used summary measure of mortality. Fertility is almost always measured as birth rates to women, and if we add these up age-specific fertility rates over all ages we get the ***Total Fertility Rate*** or TFR (the average number of children per woman, given prevailing fertility rates), the most common summary measure of fertility. The ***Net Reproduction Rate*** (***NRR***), which incorporates both fertility and mortality, is also a key measure in demography. It is calculated by multiplying a birth rate which only includes *female* births to women at each age by the probability of surviving to that age. The sum of these products over all ages is the NRR, also known as R_0 (yes, the same R_0 that epidemiologists use to discuss COVID-19). The NRR tells us how many female births in the next generation will "replace" the initial female birth, taking both fertility and survival into account. We can also use the same information (those products) to calculate the rate at which the population will grow in the long run and ignoring migration, the so-called "***intrinsic rate of natural increase***", usually denoted r. An NRR > 1 tells us that in the long run, the population will grow ($r > 0$), and if NRR < 1 it will decline ($r < 0$), while NRR = 1 means that the population will in the long run be constant ($r = 0$).

These measures are also very important in evolution because typically either the NRR or r is used to define "reproductive fitness" at the population level, in both theoretical and empirical studies. Life history theorists sometimes study how sensitive these measures are to tweaks in fertility or mortality at each age, because that sensitivity may tell us how strongly natural selection acts for or against those tweaks. Fisher's measure of reproductive value (a measure of an individual's expected contribution to future population growth) is also calculated from those products.

In this chapter, we sometimes make a distinction between **formal demography** (the mathematical description and measurement of demographic patterns) and **social demography** (focused on understanding why demographic patterns vary within and between populations, often using individual-level statistical or qualitative analysis).

Evolution simply means change over time. 'An evolutionary approach' refers to a body of multiple models and theories to explain how and why the change happens in the natural world. Natural selection is the non-random aspect of this change that comes from differential survival and reproduction. Natural selection requires that: (1) there is variation between individuals in a particular trait; (2) this variation is linked with fitness (a function of abilities to survive and reproduce); (3) this variation is heritable. If these three things consistently apply, then traits will evolve via natural selection, meaning that those traits associated with the highest fitness in a population will be 'selected' and will spread through the population over time. Natural selection, acting through changes in gene frequencies, is an especially prominent and recognized component of what researchers in evolution study, but the majority of evolutionary research does not directly study changes in gene frequencies, nor is it widely appreciated how much work focuses on other aspects of evolution, such as the influence of random events ('drift') or non-genetic processes of inheritance (like epigenetics, gene-culture coevolution and cultural transmission).

Few of the chapters in this volume directly discuss genes (with the exceptions of the chapters by Wachter, and Mills & Tropf). Many instead focus on models for explaining demographic variation that are derived from the assumption of natural selection, such as life history theory,[2] but which rarely — when applied to humans at least — involve the direct study of genetic change. Some focus explicitly on non-genetic influences on demography, such as Colleran's chapter on cultural evolution, and the chapter by Orzack and Levitis, which suggests the shape of the relationship between age and mortality risk may arise from phylogenetic inertia; in other words, it might be inherited from our species' ancestors. This does not mean that humans have stopped evolving through the process of natural selection (see chapters by Moorad and DeLong), as is sometimes claimed in the media; it just means that evolutionary processes are complex, and their study requires a multi-pronged approach.

Evolutionary demography embraces an evolutionary approach to demographic patterns and behaviours. This incorporates a wide range of research on questions of interest to evolutionary researchers, demographers and those who straddle these disciplines, united only by the assumptions that evolutionary processes are important for understanding demography, and demographic processes are important for understanding evolution. Research in this area has arisen from the recognition from both demographers and evolutionists that greater integration between the two disciplines will improve both disciplines. This recognition resulted in some early groundbreaking work by researchers such as Caswell in the late 1970s and early 1980s (Caswell, 1978, 1983, 1985), followed by a few workshops during the 1980s, but perhaps really began to take-off in the 1990s, as a research programme involving pioneers such as Vaupel, Carey, Wachter and Finch (Wachter and Finch 1997; Carey and Tuljapurkar 2003). The work of these demographers and biologists coalesced around the study of patterns of mortality and aging. They used comparative cross-species work to improve predictions of human longevity — a line

2 Life history theory is a framework used in evolutionary biology to understand how organisms allocate energy across the lifecourse to growth, reproduction and survival. The framework assumes that naturel selection has 'designed' organisms to allocate energy in ways that will maximise their reproductive success, given particular environmental conditions and subject to constraints inherent to those organisms (e.g. Stearns 2000)

of research sometimes referred to as 'evolutionary biodemography'[3] (see Carey & Vaupel (2005) and Carey and Roach (2020) [pp. 2–4] for descriptions of the development and burgeoning of this work). A little later, at least two different groups became focal points of this work in evolutionary demography. One was led by Jim Vaupel at the Max Planck Institute for Demographic Research in Rostock. A second was centered in Northern California at Stanford, UC Berkeley, and UC Davis, led by Jim Carey (while of course a great deal of pioneering work was carried out by researchers at other institutions in many countries). For both, generous funding by the US National Institute of Aging was key.

Alongside these developments, behavioural scientists such as Low (see her chapter for a personal account of how this particular biologist came to the realization of the power of uniting biology and demography), and anthropologists such as Hill & Hurtado, Kaplan, Borgerhoff Mulder and Judge began drawing on demography to improve their understanding of patterns of human reproduction and life history e.g. Borgerhoff Mulder, 1992; Low, 1994; Kaplan, 1996; Clarke & Low, 2001). A key text here was Hill & Hurtado's 1996 book *Ache Life History*. This book united a theoretical framework from evolutionary biology with demographic methods, applied to data collected over many years of anthropological fieldwork, and demonstrated the power of this particular combination of 'top down' theoretically motivated research with rigorous 'bottom up', empirically strong research. While there was some overlap between this group of researchers and those described in the paragraph above (e.g. Carey & Judge, 2001), there were also notable differences. For example, unlike the work on aging, these behavioural ecologists and anthropologists were particularly interested in how the ecology or features of the environment (broadly defined to include the social and cultural environment) shapes demographic patterns, especially fertility and reproductive behaviour (Kaplan 2003). This line of research includes interest in how species-typical patterns evolved, but also the study of how features of the environment explain contemporary variation in demographic patterns (Sear and others 2016), so at least some of the work is aligned with social demography (see Cully and Shenk's chapter for an overview of this research area, which they refer to as evolutionary ecological demography, following Bobbi Low's coining of 'ecological demography' in the 1990s: et al 1992). Much of this work developed in anthropology departments in the US, though later a group was led by Ruth Mace at University College London (while again, pioneering work went on elsewhere across the world).

Many researchers in the evolutionary sciences are now realizing that demographic perspectives, methods and data are essential for furthering their aims. In 2007, Metcalf and Pavard (2007) even wrote an article arguing that 'all evolutionary biologists should be demographers'. The fact that such a paper needed to be written indicates that demographic training is not common in evolutionary biology, but there are growing signs of recognition for the importance of demography in evolution, such as the Evolutionary Demography Society,[4] established in 2013 (whose membership consists largely of biologists working on demography

3 Sometimes referred to simply as 'biodemography', though this term is also used to describe a separate area of interest in demography which also developed around this time. This latter version of biodemography uses biomarkers (biological measurements) to inform its approach but does not draw strongly on evolutionary theory, e.g. Crimmins et al (2010).

4 https://evodemos.weebly.com

in non-human species, but does include several anthropologists and human demographers as well).

Likewise, evolutionary approaches to human demography have grown due to demographers finding that evolutionary ideas help resolve puzzles that were not proving tractable using standard social science methods and theories. While demography is an interdisciplinary science, biology has not featured strongly as one of those disciplines that has contributed to development of the field in the decades after the Second World War. This means that demographers rarely receive training in evolutionary approaches, which can accentuate misperceptions about how evolutionary approaches work. Interest in greater integration with biology, however, has not come entirely from the biological side of the fence; the success of the reunion between demography and biology which began, slowly, in the 1980s, occurred because both social and biological scientists saw benefits in working together (e.g. Hobcraft, 2006); and there are now examples of successful research programmes that do just that (see Mills and Tropf's chapter on the genetics of reproductive behavior).

Who does evolutionary demography? The field is shaped by a combination of researchers who are, broadly speaking, either 'evolution-first' or 'demography-first' in terms of their disciplinary backgrounds. Evolution-first researchers are those who start their careers with training in evolutionary ecology and then gradually adopt demographic techniques and perspectives. Demography-first researchers start as classically trained demographers and then adopt theories or perspectives from evolutionary sciences.

This is certainly an over-simplification. There are researchers such as Caswell who follow in the tradition of Lotka and Pearl in making equally important contributions to demography and biology (e.g. Caswell 1978). Nevertheless, seeing the field as composed of researchers with these two varieties of background can be illuminating. For instance, researchers from both perspectives share an appreciation for the population as a unit of analysis, and for the vagaries of how to define population boundaries. Indeed, those who can talk at length on this topic are likely trained in demography, population ecology or genetics. In both perspectives, the key processes at work occur in aggregate, in that they are measured as emergent population-level outcomes. Evolution, for example, can only be observed at the population level, not the individual level. Outcomes of interest to demographers — such as life expectancy at birth or total fertility rates — are also characteristics of populations, not individuals. But these processes have implications for individuals. If some behavioural feature affects variation in life expectancy, like smoking, then we can make recommendations that individuals should weigh up the risks involved in smoking before engaging in this behaviour. Evolutionary theory can make predictions at the individual level, or at least the sub-population level, given that observable phenotypic outcomes are the product of the interaction between genes and environment. This means that individual, or sub-group, outcomes may differ within the same population if individuals or sub-groups experience different environments.

Each pathway, the evolution-first and the demography-first, also has its differences (Kaplan and Gurven 2008). Training in evolution tends to lead to more 'top-down' theoretical motivation and testing of causal hypotheses. Demography is much more empirical and builds understanding of patterns from the 'bottom up'. In evolution, ecology shapes demography (see Box 2 for more discussion of this among evolution-first researchers). In demography, demography shapes ecology (or ecology is not relevant). In demography, fertility and mortality

are distinct topics and many researchers will specialize in one or the other. In evolution, fertility and mortality are connected parts of a strategy, and the focus of a study is more likely to be on the whole strategy rather than one vital rate in isolation. Related to this distinction is that in evolution, demographic traits are products of an energy budget and any study of how tradeoffs affect demographic patterns is anchored in the concept of an energy budget. Indeed, the concept of an energy budget is prominent among 'evolution-first' evolutionary demographers because they likely encountered demography after learning about life history theory, the subfield of evolutionary ecology that applies evolutionary theory to demographic patterns, and which is key to the interface between evolution and demography.

Box 2: Defining terms within the evolution side of evolutionary demography

Many evolution-first researchers likely developed interests in evolutionary demography through the field of ***evolutionary ecology***, which is a highly successful theory-driven, predictive, and experimental enterprise focused on explaining how natural selection affects ***phenotypes*** (outwardly observable characteristics of individuals, which can be studied without immediate information on the genes involved), and how these phenotypes vary adaptively by ecological context. The shaping of phenotypes by ecology is therefore a key topic of study among many evolution-first evolutionary demographers. The assumption is that different phenotypes will optimize ***fitness*** — the propagation of genes in future generations — in different ecological conditions. Between species, natural selection shapes genetic variation so that species develop traits that are ***adaptive*** — that maximize fitness — in their particular ecology. Within species, natural selection has resulted in '***phenotypic plasticity***', the ability of the same genotype to give rise to different phenotypes in response to different ecological conditions (for example, age at menarche declines in human populations as nutritional conditions in that population improve). This plasticity is not entirely unconstrained — there is no population in which the average age at menarche is as young as 3 or as old as 30 — but nevertheless, for many traits, there is some inbuilt flexibility that allows them to vary according to external factors.

Like demography, which spans physiology and behavior, evolutionary ecology includes the study of both physiological and behavioural phenotypes; behavioural ecology is the sub-field that focuses on behavior. Evolutionary ecology seeks evolutionary explanation for any observable ***phenotype*** (any trait such as hair color, size, a distinctive birdcall, that results from the interaction of ***genotype*** and environment), while behavioural ecology would focus on the subset of those observable traits that are behaviours. Many evolutionary demographers interested in explaining variation in contemporary demographic patterns would consider themselves ***human behavioural ecologists***, though some will study behavioral and non-behavioral phenotypes.

Study design and analysis in evolutionary ecology often builds from an assumption that the trait in question will be close to ***optimal*** in terms of maximizing fitness for a given ecological context. This assumption then generates hypotheses about how variation in the ecological conditions affects variation in the trait, or how a specified change in circumstances might affect what trait values are optimal with respect to maximizing

fitness. In this way, optimization is used as a learning strategy; deviations from model predictions often help identify mis-specified costs for a behaviour or other factors crucial for explaining its variation. It is important to recognize that research in this tradition does not require that variation in physiology or behaviour between environments is driven by genetic differences between individuals. It also allows for individual and social learning to contribute to flexibility in the behaviour being studied (see Colleran's chapter for a discussion of how culture can be integrated into evolutionary demography).

Key to all of these approaches is that the utility being maximized by both behavioural and physiological traits in different ecological contexts is fitness (at the individual level, sometimes also loosely operationalised in empirical research as ***reproductive success*** — the number of offspring raised successfully to reproductive maturity — though this is only a rough approximation of fitness). Therefore, a key insight of the evolutionary approach is that our behaviour and physiology are not selected to maximize our health, wealth or happiness, but our genetic fitness, though in some cases maximizing health, wealth or happiness may be the pathway to maximizing fitness.

As examples of these pathways, of the three editors of this volume, two (Oskar Burger [O.B.] and Rebecca Sear [R.S.]) are evolution-first and one (Ronald Lee [R.L.]) is a demography-first researcher, perhaps a not-dissimilar ratio to the field as a whole. R.S. trained in zoology then in biological anthropology, developing a skillset as a human behavioural ecologist. Her PhD, with Ruth Mace (then a rare behavioural ecologist in the UK aware of the opportunities and benefits of working on our own species), involved applying the behavioural ecological approach to a demographic dataset, requiring her also to pick up some demographic methods. She was then hired for a job teaching demography, by a demographer — John Hobcraft — who was influential in promoting greater incorporation of biological thinking into demography, in a social science institution (London School of Economics). This immersion into demography and social science really brought home the benefits of uniting social and evolutionary science in understanding our species: an exclusively evolutionary approach is stunted, not just because of the unusually important role of social interactions and culture in explaining human behavior, but because there is just so much relevant existing work in the social sciences which it is simply inefficient to ignore. O.B.'s graduate school training was based in anthropology departments, starting in archaeology and gradually transitioning toward biological anthropology and evolutionary ecology. He took several classes in graduate school from well-known human evolutionary ecologists like Hilly Kaplan and Kim Hill, and was especially influenced by a forefather of the field, Eric Charnov. O.B. then received a much-needed education in demographic principles during a postdoctoral fellowship at the Max Planck Institute for Demographic Research, and gained tremendously from working with Jim Vaupel and his working group on the evolution of aging (including Owen Jones, Dan Levitis, Hal Caswell, and Kai Willführ, all contributors to this volume). The Evolutionary Demography Society formed during a workshop in Evolutionary Demography at the MPIDR, and O.B. was proudly one of the founding members (as were many of the contributors to this volume who were also at this workshop).

R.L.'s demography-first training began in demography at UC Berkeley (with Nathan Keyfitz among others) and then economics at Harvard. From the start he was interested in historical

applications of Malthusian theory and its counterpart in density dependence in non-human species. Later he developed mathematical models of intergenerational transfers in human populations and empirical applications through what became the National Transfer Accounts project co-directed with Andy Mason. He learned a great deal through collaboration or discussions with evolutionary anthropologists like Hilly Kaplan, Michael Gurven and Karen Kramer, applying the models to their hunter gatherer group data. Participation in workshops on evolutionary biodemography in the late 1980s and 1990s had an important influence. The Hill and Hurtado book on the Ache was a revelation (and also an inspiration for R.S. and O.B.). In 2002 he began reading evolutionary theories of senescence, starting a long process of self-education in cross-species evolutionary biodemography, informed and stimulated by a group led by Jim Carey (including Wachter, Tuljapurkar, and some honey bee researchers), and several joint workshops sponsored by the Carey group and the MPIDR group under Vaupel. R.L.'s particular interest, continuing today, is the integration of energy flows, intergenerational transfers, food sharing and cooperation with evolutionary life history theory, on which he has a chapter in this volume.

The commonalities of these three pathways indicate the importance of interdisciplinary training (all three started out with interdisciplinary training in biology/anthropology, anthropology/archaeology and demography/economics respectively), which then led to further explorations with other disciplines; as well as the importance of providing space for researchers to develop new skills, and to interact with a broad range of individuals and institutions, who are prepared to engage with one another to advance knowledge.

What does evolutionary demography look like now?

A glance at the table of contents of this volume illustrates the diversity of evolutionary demography. We have contributions that foreground evolutionary processes (such as selection and fitness), alongside many that foreground issues of interest to social scientists (on health, culture, household or intergenerational relations), as well as a range of perspectives on the field from biologists, anthropologists and demographers. Very loosely, evolutionary demography can perhaps be roughly divided into (1) research that focuses on describing evolutionary and/or population processes, which often draws most on the 'core' of formal demographic methods (we label this here evolutionary biodemography), and (2) research that focuses on explaining variation within our species in demographic patterns, often using individual-level analysis, and which is more aligned with social demography (evolutionary ecological demography). We do this not to create or solidify divisions within the field of evolutionary demography — especially given our arguments that evolutionary demography is important in its destruction of disciplinary silos — but as a convenient tool for crudely summarizing research in evolutionary demography. We are sure that others may disagree with this division, as well as finding research that doesn't fit neatly into these categories.

Evolutionary biodemography: This is a loose grouping of research, which tends to focus particularly on population-level phenomenon (compared with somewhat greater emphasis on within-population variation in evolutionary ecological demography), and is particularly well populated by biologists, with some demography-first researchers. It is the branch of evolutionary demography that first stimulated interest in merging the two parent fields, when demographers and evolutionary biologists came together to solve puzzles around mortality

and the aging process. Both human demographers and evolutionary biologists interested in the demography of non-human species had long been interested in how mortality rates vary by age, and had developed models to predict this variation. As human lifespans lengthened during the twentieth century, with increasingly effective medical care and other socioeconomic shifts that reduced mortality, it became clear that existing models did not seem to fit the observed data well at very old ages (Oeppen and Vaupel 2002; Vaupel 1997). In recognition of the problematic understanding of the origin of aging patterns, it became apparent that some new partnerships, such as those between demographers and biologists, were not just logical but also necessary. This led to a highly productive research tradition on the evolution of aging patterns and age-specific mortality rates, subsequently expanded to fertility patterns, involving cross-species comparisons. Cross-species comparison of mortality patterns has shown how remarkable human mortality improvement is, and has helped to demonstrate that a great deal more variation in age patterns across species is environmental, rather than genetic, than previously thought (Vaupel and others 1998; Jones and others 2014). In this volume, Jones and colleagues' chapter in this tradition applies a cross-species analysis to the study of life history strategies (how life events such as births are organized across the life course).

Another set of research questions in which this branch of evolutionary demography is interested focuses on the mechanics of evolution, and merges evolutionary biology with demography to answer research questions of particular interest to evolutionary biologists. We include several chapters in this volume that consider the mechanics of evolutionary processes and how they relate to demographic processes. For example, Moorad's chapter on 'Measuring selection for quantitative traits in human populations' is effectively a primer on quantitative genetics, providing guidance on methods intended to characterise natural selection on traits of interest but also highlighting the flexibility of this approach and its ability to deal with complications inherent to the study of human populations, including and social interactions. Ken Wachter, a pioneer of evolutionary demography, contributes a chapter on 'genetic evolutionary demography', focusing on mutation accumulation, and highlighting how 'with the rise of biodemography, evolutionary ideas have come to play leading roles in demographic thinking'. Hal Caswell and Silke Van Dalen focus on a neglected source of variation in fitness — demography — observing how demography can cause variation in fitness, which is stochastic and non-heritable.

Encompassed in this branch is work on developing techniques in formal demography, which is of interest both to evolutionary biologists, who need formal demography to fully understand reproductive fitness, and to demographers. Some of Hal Caswell's work fits in here, such as that on matric population models and the demography of kinship (Caswell 2001, 2019). In this volume, we have a contribution from Jim Carey, who describes his discovery of an identity in which the fraction of individuals x days old in a stationary population equals the fraction that day x days later. Carey highlights in this chapter one of the important benefits of interdisciplinary research — value brought by a fresh perspective because questions are asked that have not been asked before by each 'parent' discipline.

Evolutionary ecological demography: this branch leverages the fact that natural selection has shaped human physiology and behavior to help explain demographic patterns, typically focused on individual-level explanations. Anthropologists are well-represented in this area. Much of this branch has focused on reproductive outcomes, rather than mortality, taking as

a starting heuristic the assumption that reproductive behaviour has been shaped by natural selection to respond adaptively to changes in the environment (defining the environment broadly to encompass social interactions and culture). The field acknowledges that much contemporary human behaviour no longer functions to maximise reproductive success, given that the environment we live in today is different in many respects from that in which we spent most of our evolutionary history, but still argues that insights from evolution can help us to understand contemporary demographic variation. A lot of the research in this branch is about how variation in environmental or contextual conditions influences variation in demographic patterns across and within populations, given that it draws much inspiration from evolutionary (behavioural) ecology (Cully & Shenk provide an up-to-date overview in their chapter; see also Low, 1993; Mace, 2000, 2007; Voland, 2000).

Some early work here contributed to active debates in demography about the demographic transition (the shift from high mortality and high fertility to low mortality and low fertility that has happened, or is happening, worldwide). For example, Kaplan's anthropological studies with subsistence societies in South America demonstrated that children are always economically costly to parents (Kaplan 1994). This contrasted with some work in demography suggesting children were economically net producers throughout much of human history, so that part of the explanation for the demographic transition was that fertility dropped when children became a net economic cost to parents (Caldwell 1978, 1982). Kramer and Lee have also shown, however, that, despite being a net economic loss, children do contribute substantially to the household economy in pre-demographic transition societies (Kramer 2002) and that the high fertility maintained in pre-transition societies was underwritten by children's labour contributions (Lee and Kramer 2002). Evolutionary demographic arguments don't always contradict those from demography or other social sciences, however. Kaplan and colleagues (Kaplan 1996; Kaplan and Lancaster 2003), for example, have also produced models of the demographic transition that incorporate the shift from investing in child quantity to child quality — an important component of demographic transition models in demography, drawing on Becker's (1991, first edition published in 1981) work. Kaplan and colleagues' models add an extra layer of explanation to Becker's proposal by combining its economic foundation with an ultimate evolutionary function: ultimately our behavior is designed to maximize reproductive success, not household economic success or happiness.

The evolutionary demography of contemporary variation is currently a thriving area of research, and this volume includes many chapters in this tradition (see chapters by pioneers of this field Kim Hill, Nick Blurton-Jones, and Monique Borgerhoff-Mulder). A newer generation of researchers is keen to move the field towards applied research, combining evolutionary demography with public health and development (see chapters by Gurven et al and Gibson & Lawson). Again, this research fuses evolutionary with anthropological insights to suggest new avenues for applied demography, public health and development. Having mainly begun by studying the small-scale societies favoured by anthropologists, this field is increasingly moving in the direction of studying high-income populations (see chapters by Anna Rotkirch and Caroline Uggala), a welcome direction for many reasons, including increasing concern about 'helicopter' research by scholars from high-income populations working in lower income communities without sufficient community engagement (see Urassa et al).

Anna Rotkirch, for example, discusses the evolutionary demography of marriage in high-income populations; a refreshing perspective given that long-term relationships have been rather ignored by evolutionary researchers, despite a vast evolutionary literature on the mate preferences of students, as well as significant research on marriage in mainstream demography. Historical evolutionary demographic work also fits in this category and is well represented in this volume. Historical demographers have been particularly keen to apply evolutionary ideas to questions of interest, perhaps because they share a long-term perspective on our species. Evolutionary social scientists were also quick to realise the benefits of using historical data, which allows the testing of hypotheses across multiple generations. Chapters by Lisa Dillon and colleagues, Julia Jennings, and Kai Wilfuhr and colleagues all focus on how relationships between individuals within and beyond the household affect demographic patterns, a research area to which historical demographic analysis has made significant contributions. From a very different perspective, Jonathan Wells' chapter also discusses how relationships may affect demographic outcomes. His chapter sets relationships between those of different socioeconomic positions within the 'producer-scrounger' framework from biology, where 'scrounging' by the socioeconomically advantaged can affect the demography and life history of the socioeconomically disadvantaged 'producers'.

One consistent area of interest for evolutionary demographers, which perhaps does not fit neatly into either category above, is how our species' life history evolved (e.g. Hill & Kaplan, 1999; see the chapter by Tuljapurkar on the unusual trait of human menopause). Life history research explores how life events such as growth, reproduction and death happen across the life course. Evolutionary demographers have shown interest in how and why our particular life history pattern evolved — which includes relatively slow growth, late but then rapid reproduction, followed by a highly unusual cessation of reproduction long before death (in women: menopause). This research has often included building mathematical models of alternative scenarios, in order to explore how different factors may have influenced the evolution of human life history. Evolutionary demographers have suggested that part of the answer is our highly cooperative nature, which includes intergenerational transfers (Lee 2003, 2008; Kramer 2010). Humans engage in multiple cooperative activities, including extensive sharing of resources (referred to as a 'pooled energy budget', see Reiches and others 2009; Kramer and Ellison 2010), and substantial support for child-raising (Hrdy 2009). This help comes from many sources, including the older grandparental generation but also from older children and even unrelated adults (Sear and Coall 2011; Kramer and Veile 2018). It is these (largely) intergenerational transfers that have shaped our life history patterns, including our relatively rapid reproductive rate, at least compared to other apes, long lifespans and menopause, a trait shared only with a handful of other species, which also engage in intergenerational transfers such as certain whale species (Nattrass and others 2019; Johnstone and Cant 2019).

In this volume, Ronald Lee extends his work in this area with a microsimulation modelling exercise of how the size and relatedness of sharing-group arrangements affect the evolution of life history. This chapter not only reinforces the importance of intergenerational transfers in the evolution of human life history, but also shows variation between societies in how resources are transferred, notably in that contemporary high-income countries have reversed the wealth flows of subsistence societies throughout history. In high-income societies, net intergenerational transfers flow *up* generations, because of the public transfer of wealth to older

age groups through pensions and medical care (private transfers still flow down generations). This may well have significant implications for human life history and its future prospects. Our cooperative nature also means that humans are quite altruistic, punish cheating, enjoy the company of others, are lonely when isolated and develop elaborate cultures. All these are reasons why evolutionary demographers should draw on research on sociality from the social sciences, just as demography needs input from evolutionary frameworks.

What are the organisational frameworks of evolutionary demography?

Before concluding, we will briefly mention some important organisational frameworks that help to clarify the field.

Multiple levels of explanation: Particularly important conceptual frameworks in evolutionary biology, which are of relevance for evolutionary demography, are those which make the point that there are multiple different but mutually compatible explanations for traits, including behavioural and demographic traits. In evolutionary biology, Mayr (1961) introduced the concept of proximate and ultimate explanations for traits: proximate explanations are the immediate explanations for a trait, such as the mechanisms that bring about a particular trait (*how* is this trait brought about?); ultimate explanations refer to historical explanations for a trait, such as the evolutionary 'function' of a trait, i.e. what adaptive problem does it solve (*why* does this trait exist?). Taking the behaviour of eating as an example, one proximate explanation for why we eat is that we respond to the physiological sensation of hunger; an ultimate explanation would be that we eat because if we did not regularly take in food, we would die. As a very broad generalization, evolutionary researchers often focus on the 'why' questions, while social science typically focus on 'how' questions; note, this means that evolutionary and social science explanations are often compatible (not in opposition to one another, as is sometimes assumed).

A related framework for emphasizing that multiple levels of explanation can exist for the same traits is that of Tinbergen's (1963) 'four questions'. Two of Tinbergen's four questions relate to the historical explanations for a trait: 'functional' explanations are those that focus on the adaptive value of a trait (how does this trait maximise reproductive success?); 'phylogenetic' explanations consider the evolutionary history of a trait (how did this trait come to be over deep evolutionary time?). The other two relate to the more immediate causes of a trait. One relates to proximate explanation: what are the proximate (physiological or behavioural) processes which bring about this trait? The final explanation is ontogenetic: how does this trait develop during an individual's lifetime? One of the sections of our volume is a 'Tinbergen section', which uses this classic organizing framework to highlight different types of work in evolutionary demography. It illustrates how functional explanations can help understand demographic patterns (Mace's chapter, 'Why do we do what we do?'); how widely life history patterns are shared with other species (Jones & colleagues' chapter, 'My family and other animals'); how demographic outcomes are affected by what happens during childhood and adolescence (Sheppard & Coall's 'What has childhood done for us?'); and how physiological mechanisms bring about reproductive outcomes (Vitzthum's 'How it works'). This framework can be helpful for understanding both species-typical or population-level traits (as in the Jones & colleagues' chapter) but also variation in demographic traits at the individual level (which is the level Mace, Sheppard & Coall and Vitzthum discuss).

Life History Theory: if the proximate/ultimate distinction and Tinbergen's 'four questions' are organisational frameworks that focus attention on the importance of different types of research question, the theoretical framework most commonly used to guide evolutionary demography is life history theory. Life history theory is the application of evolutionary theory to understanding 'life history traits'. Life history traits include the demographic traits of mortality and fertility, in addition to indicators of growth and development such as the sizes of offspring, juvenile growth rate between birth and adulthood, age at sexual maturity and ageing. This means that research in life history theory has considerable overlap with evolutionary demography. However, there are a few differences in the styles of research and topics covered by each. Life history theory uses the concept of an 'energy budget', which is the food-derived energy that an organism obtains either by foraging (hunting, browsing, scavenging, etc.) or that is obtained via cooperative or exploitive relationships with other individuals. All of the energy that an organism obtains will be 'spent' on various goals. These include somatic growth, energy burned by the body's immune system or the physiological cost of repairing the body's tissues, as well as the energy that goes into finding and attracting mates, into producing children and caring for them.

This is a useful framework because energy that goes toward one end, such as immune function, cannot go to another, such as producing offspring, meaning that there must be ***trade-offs*** between life history traits. If evolution is 'shaping' this budget in non-random ways then we learn a great deal from studying patterns, within or across species, for how an organism 'spends' this budget. A key message from this section is that the importance of trade-offs in life history theory means that growth and the demographic outcomes of fertility and mortality are linked across the life course. A cautionary tale about what happens when this insight is ignored is provided by Mhairi Gibson's work on an energy-saving development project in rural Ethiopia (Gibson and Mace 2006), intended to improve the health of women and children, which had the unanticipated consequences of increasing women's fertility and possibly worsening child health, given that the energy saved by the development initiative was simply diverted into higher fertility (see her chapter with David Lawson on evolutionary approaches to population health for more detail on how evolutionary insights can be used in applied research).

Because of the influence of life history theory, in evolutionary demography it is much less common to study demographic traits in isolation from one another, because they are all linked together by the concepts of trade-offs and energy budgets. Demographic (or life history) traits, taken together, are seen as the solution to a problem. This problem is posed by the environment, including other organisms of the same and other species, and subject to constraints of the animal's physical make-up (how large is it, how fast can it move, or what kind of food must it eat). Indeed, life history traits are highly patterned across species. This is an important, active area of research in evolutionary biology and we examine it in more detail in the concluding chapter. Here we refer interested readers to the classic and foundational works of Hamilton (1966), which represents the dawn of life history theory (though Hamilton does not use that terminology); Charnov's (2001, 1997) classic work on mammal models and on the structure of life history tradeoffs; summary articles by Stearns (1976, 2000), and textbooks by Stearns and Roff (Roff 2002; Stearns 1992), which describe the field as it developed into maturity in the 1990s. Holland Jones has provided an overview of this literature (Jones, 2011).

Life history theory has been phenomenally successful at explaining and providing a structure for life history traits across species. It is very much a 'top-down' field which tries to make use of

explicitly derived predictions. It has also been applied to the study of within-species variation in life history, including our own, with work demonstrating trade-offs, for example, between growth and reproduction: we are one of those species in which, when reproduction starts, growth tends to stop, meaning that there are both within- and between-population associations between shorter height and earlier first births (see Hill's chapter and Uggala's chapter for explicit discussion of how life history theory can be applied to understanding demographic outcomes; several other chapters incorporate life history approaches, including Pavard & Metcalf's, Jones et al's, and Vtizthum's; Emery Thompson and Sabbi's contribution focuses on the life history of great apes other than humans).

It is worth noting here that there are debates within the evolutionary social sciences about the use of 'life history theory', notably a concern that many 'predictions' in life history theory in fact arise from empirical observations and/or verbally intuitive models that are rarely formalised using mathematical theory, and so are not predictions derived from theory at all (Nettle 2022). For example, a common assumption in the human life history literature is that high extrinsic mortality rates will lead to 'living fast and dying young', based on the intuition that when life expectancies are short, then it makes sense to get started on reproduction as early as possible, to avoid the risk of dying before successfully raising children (see Uggla's chapter). Such work often also assumes that this 'live fast, die young' strategy will be partly mediated by behavioural differences, such as greater orientation towards the present (rather than the future) or greater propensity to take risks.[5] Formal modelling, in both evolutionary biology and the evolutionary social sciences, suggests that this assumption may not necessarily hold, though it might under a certain restricted set of circumstances. This assumption has generated a lot of research, however, and many empirical studies at both population and individual level seem to find support for earlier reproduction in environments with higher mortality. Such findings may or may not be due to a 'living-fast-dying-young' strategy — and hopefully research will now turn to understanding the reasons for these empirical findings in more detail (Vries and others 2022) — but research drawing on ideas in life history research has nevertheless been influential in finding empirical regularities (unless of course the file-drawer effect has influenced this literature), which might otherwise not have been investigated. The concluding chapter of this volume discusses in more detail how a significant advantage of evolutionary demography is the ability of interdisciplinary research to throw up new research areas not commonly considered in the mainstream of a discipline.

5 Note: 'life history theory' in this volume refers exclusively to life history theory in evolutionary biology ('LHT-E' for readers familiar with Nettle and Frankenhuis' terminology: Nettle and Frankenhuis 2020, 2019). Here we do not discuss the conceptually distinct 'psychometric' approach to 'life history strategy' in psychology, which claims that 'life history theory' predicts that a large number of behavioural and cognitive traits cluster together into 'fast' or 'slow' life history strategies. This approach derives its theoretical framework from Philippe Rushton's 'differential-K' theory, which is scientific racism not science, and so this 'psychometric approach' should not be confused with life history theory from evolutionary biology (Sear 2021, 2020); see also the recent Evolution and Human Behavior special issue on 'Current debates in human life history' for more on the current state of human life history research: https://doi.org/10.1016/j.evolhumbehav.2020.09.005

Conclusion: A thriving and vibrant field

This chapter has introduced the field of evolutionary demography, some of its organizing frameworks, and provided examples of research in this field. It is a difficult field to define with precision, perhaps because of its interdisciplinary nature. Much of this chapter has focused on the field's ability to move forward our understanding of human demographic patterns, both at the population and individual level. This likely reflects the biases of the authors, given that not all research in evolutionary demography focuses on this endeavour, but instead, for example, focuses on questions of interest to evolutionary biologists, such as the mechanics of selection process. We invite the reader to draw their own conclusions about what evolutionary demography is by exploring the chapters in this volume. We have not organized the volume using our dichotomy between evolutionary biodemography and evolutionary ecological demography (for several reasons, including the difficulty of shoehorning all evolutionary demography into these categories); instead we have grouped chapters together on related topics (perhaps a more 'demographic' than 'evolutionary' classification given demography's orientation towards topics of interest).

We do here highlight one important feature of evolutionary demography, though: its comparative approach — comparative across both species and across all different kinds of human population, including throughout time, which enables new ways of thinking about demographic processes in our species.

There are challenges with any interdisciplinary endeavour, however, including differences in language, traditions of research, and a lack of interdisciplinary training, meaning that most demographers have little experience of evolutionary theory and that evolutionary social scientists have little training in demography. These challenges will require some effort to overcome. We hope that a volume such as this might help solve some of these challenges, but other steps could also be taken, such as improving training in the interdisciplinary field of evolutionary demography. This could incorporate both bringing in more demography content to evolutionary biology programmes, as recommended some years ago by Metcalf and Pavard, and incorporating more evolutionary training in demography programmes. The aim is not to turn all biologists into demographers or all demographers into evolutionary demographers, but to provide core training in both disciplines in order to supply early career researchers with a set of options about which direction to take their research and, hopefully, also to dispel the misconceptions that are still held in some of the social sciences about evolutionary approaches.

This cross-fertilisation of disciplines should be encouraged further, as such a broadening of skillsets in the social and health sciences can only strengthen our understanding of our species. Breaking out of our disciplinary silos has enormous potential to increase the efficiency of research, and to avoid the problem of disciplines constantly reinventing a wheel that another discipline has already put much time and effort into developing. Demography is also, in our wholly biased opinion, the most interesting of the social sciences. Demography matters to a huge variety of topics of interest in the social and biological sciences. Population processes — involving births, deaths and migrations — are also of great personal and policy significance. The news is full of population stories on a daily basis; such stories are not only of interest in their own right but because they are often used to promote particular political narratives. Rigorous, critical research on population is important to ensure we have a solid evidence base to inform policies and media narratives. Understanding how and why we live

and die, why we have children and the number of children that we do, and why these patterns vary between individuals and populations, is also key to understanding the human condition.

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SECTION 1: THE RATIONALE, MOTIVATIONS AND QUESTIONS IN HUMAN EVOLUTIONARY DEMOGRAPHY

To help extend the discussion from our introduction, the following trifecta of chapters comes from three distinctive viewpoints on human evolutionary demography. Across these three chapters, we hope that readers become acquainted with the connections between evolutionary and demographic approaches, realize their inherent complementarity, and see some of the big-picture topics that human evolutionary demographers are focused on.

Kreager is a classically-trained demographer who is somewhat unusual in recognizing both the contribution that demographic methods make to evolutionary research and the value of an evolutionary approach for contextualizing and explaining demographic patterns. Drawing from extensive cross-disciplinary research and expertise in population-oriented thinking, he provides an excellent jumping-off point for new readers to the links between classic demography and evolutionary science. In doing so, he presents many important concepts that are echoed in later chapters.

A champion of behavioural ecology, Low came to appreciate demography as an evolutionary biologist interested in population-level issues and questions. In the form of an engaging personal essay, Low provides her expert perspective on the value of demography for improving biology's ability to understand, for example, how evolution affects relationships (trade-offs) between fertility and mortality; but the implications of her argument apply across demographic behaviours.

Hill is an anthropologist who has done well-known long-term ethnographic fieldwork in the Amazon, with the Ache of Paraguay. Hill was one of the earliest anthropology adopters of life history theory, and has published life tables for small-scale societies and for chimpanzees. Along with Magdalena Hurtado, he pioneered the use of cutting-edge statistical techniques from demography and epidemiology in the discipline, recognising the importance of classical demographic methods for evolutionary anthropology. From this perspective Hill gives us the ten 'Interesting Issues in Evolutionary Demography' that should be of great interest to those who have been long active in this field, but also to newcomers interested in understanding the key issues we grapple with.

2. Evolution in the History of Population Thought

Philip Kreager

This chapter places evolutionary demography in the history of population thought, and more particularly in relations between demography and evolutionary population biology. Darwin conceived evolution as a dynamics of variation arising from the behaviour of populations at intra- and inter-species levels. While Malthus's principle of population was an important early stimulus, Darwin resolved the core problem in evolution — how mechanisms of variation combine to produce divergence of character — by analogy to Smith's account of the division of labour. With the benefit of hindsight, we can describe Darwinian population thinking as the first general methodology in which it became possible to combine bottom-up observation, including enumeration of local population dynamics, with top-down statistical methods. The two components entail different concepts of population, which may be characterised broadly as "open" and "closed". Their combination shows that evolutionary theory is rooted in the same sources of population thinking that gave rise to demography: the former lie in Classical population thinking and early modern population arithmetics, and the latter in nineteenth-century statistics and probability.

Hereditary influences remained a "black box" in Darwin's theory, which only began to be unpacked with the rediscovery of Mendel's research. The second half of the chapter traces the central role which demographic methods played in topical and analytical developments of the first half of the twentieth century, including both the formulation and critique of eugenics, the emergence of population ecology, and the rise of the mathematical theory of population genetics. There is an irony here: even as demographic methods came to play an integral role, mainstream demographers became less and less involved. The "separatism" of demography and evolutionary biology often remarked on in the post-war era thus has deeper roots. These lie partly in topical issues, like reactions against eugenics, but more importantly in a conceptual shift in how we understand relationships between ultimate and proximate mechanisms of population change, and its implications for analysis and modelling. Evolutionary theory entails a balance of methods and insights drawing on both population concepts, which demography has not yet achieved. The concluding section provides examples of how current evolutionary demography is now integrating these developments into demographic explanation.

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> By introducing population thinking, Darwin produced one of the most fundamental revolutions in biological thinking.
>
> — Ernst Mayr

Demography is generally considered the pre-eminent social scientific study of human populations. Its methods and practices embrace all the social sciences and adjacent medical disciplines of population health. By a convention widely observed over the second half of the twentieth century, quantitative inquiry on human subjects in population biology (inclusive of genetics, ecology and other fields of evolutionary biology) has been viewed separately from demography, even though there is often significant methodological and substantive overlap. Of course, the latter fields also address other species, but often with a view to resolving problems faced by human populations. While the importance of genetic and ecological knowledge has in recent decades gradually come to be accepted by many demographers in addressing topics like mortality, ageing, resource sustainability and the implications of fertility declines, what may be called the "separatist" view has continued to prevail more widely. Going beyond contributions to the substantive topics just mentioned, however, there is a larger issue which may be called the knowledge impact of innovative science. Even slight familiarity with the discovery of DNA, of genomics and developmental biology is sufficient to recognise that the growth of population-based knowledge and applications in the several fields of evolutionary biology over the last half century has been nothing short of phenomenal. At present, whether we consider volume or funding of research, population biology arguably now constitutes the considerably larger domain of population inquiry.

There is thus a strong *prima facie* argument for demographers to reconsider the separatist view. After all, if concepts and models of population have proven so fruitful in the development of evolutionary research, the advisability of intellectual exchange is, at least, indicated. Yet so pervasive has been the separatist view that it prevails widely as a given or unstated assumption in demography, thus becoming an obstacle to rethinking relations between biological and social scientific domains. Mayr's observation (1982: 487), above, is a case in point: that Darwinian population thinking *revolutionised* biology refers to developments quite unfamiliar in mainstream demography, and which might in consequence appear to carry no real importance for the discipline. History, however, shows otherwise. Darwin, in formulating the concept of natural selection, made population dynamics a central mechanism of evolution. To do this he relied heavily on population concepts foundational to population arithmetic and political economy in the seventeenth and eighteenth centuries that are also the sources of demography. There thus remains a common historical ground of population concepts, even though these fields have diverged subsequently.

The common ground is not of merely historical interest. The approach to population dynamics that Darwin initiated has remained truer to concepts and sources that first gave rise to quantitative research as a scientific approach to society. His population thinking achieved this by showing how core concepts of population prevailing before 1800 were fundamental to an evolutionary framework in which statistical methods were also key. As evolutionary biologists developed this combined approach in the late nineteenth and early twentieth century, demographic models and measures came to be seen as core components of population genetics and ecology; the greater explanatory power of this combined framework then underpinned the tremendous success witnessed in our era.

The purpose of this chapter is, first, to explicate Mayr's observation, and then to trace, going forward, the development of the concept of evolution as a locus of population thinking which has led to the recent revival of demographic interest in evolutionary research. The discussion proceeds in four steps. First, Darwin's population thinking is outlined. Here we follow its depiction in the influential "Evolutionary Synthesis" which Mayr, Dobzhansky and other evolutionary biologists put forward in the 1930s and 1940s, since their account remains the baseline from which contemporary population biology has grown. Second, Darwin's sources are reviewed, in order to establish the common conceptual ground of demography and evolutionary biology. This takes us back to eighteenth-century authors, notably Adam Smith, and to his early nineteenth-century followers (notably Robert Malthus), whose different concepts of population were brought together in Darwin's approach to concepts like variation and fitness. It is important to clarify how population thinking in demography and evolutionary biology are similar in major respects, but have differed in others. Two distinctions commonly employed in the literature (between open and closed population thinking, and between proximate and ultimate causes) are introduced for this purpose. It is striking that, although Darwin's own mathematics remained numerical, and the primary role of environment-organism interaction in his theory remained grounded in natural history, his recognition of the need for statistical inference in treating variation and fitness led to formal population models, like the life table, becoming a common ground of population genetics and ecology by the 1930s.

The third step considers developments in the late nineteenth and early twentieth century which took up the challenge Darwin's theory posed regarding how to integrate concepts and evidence from observational and field methods with concepts necessary for formal data collection and analysis. The Synthesis, which Mayr, Dobzhansky and their colleagues achieved, only occurred after several decades of controversy in which different ways of developing evolutionary population thinking were explored — with widely varying outcomes — in eugenics, public health, ecology and population genetics. The controversy over eugenics accounts in part for the hiatus that led demographers from the middle decades of the twentieth century to see their research separately from evolution. Yet there is a paradox: demographers turned away just as their methods were becoming core to mainstream population genetics and ecology. A more fundamental reason than aversion to the outlier of eugenics was the major factor in this turn; notably, whether a balance of open and closed population thinking was achieved. By way of conclusion, the final step in this story reviews problems related to scientific explanations that in recent decades have led demographers to contemplate their own methodological synthesis along evolutionary lines, and examples of promising research that are now emerging.

Population Thinking in the Emergence of Evolutionary Theory

Biologists' recent statements about the structure of evolutionary theory (e.g. Lewontin 2001; Gould 2002; Mayr 2004) emphasize relationships between three levels of population phenomena: genes (each individual's genome is a population composed of more than three billion DNA base pairs); organisms (each composed of populations of cells and organs that together form the several sub-populations, or demes, of which a species is composed); and environments (involving relationships within and between demic, and between species, populations, in the course of which environmental niches are occupied and constructed). As Darwin's theory gave a significant role to heredity, but was composed before the rise of genetics, these authors

take a historical approach that can be understood in three broad stages. The first begins with the logic of natural selection in the *Origin of Species* (1996 [1859]), noting unresolved issues that remained in Darwin's reasoning. The second then pursues subsequent developments: the rediscovery of Mendel's laws and ensuing controversies; the rise of population genetics; and the restatement of Darwin's programme provided by the Evolutionary Synthesis. The third then discusses the contemporary era of phenomenal growth in evolutionary biology opened up by the Synthesis, as well as limitations in the framework it has provided. This historical approach will be adopted in the inevitably much briefer summary given here.

Natural selection is a force or process in which variations that give an advantage to their bearers in the struggle for life are expected to accumulate in a species, and to have two major effects: firstly, they increase the adaptation of organisms to the environments in which they live; and secondly, they gradually modify the species. As Gould remarks, this process can be broken down into three components which provide the "syllogistic core" of Darwin's theory (2002: 125–41). *Variation* is arguably the most fundamental: the elemental fact that all organisms have unique characteristics requires not only that any species population is composed of a diversity of individuals, but that this population heterogeneity is continuously renewed. Natural historians before Darwin were, of course, familiar with individual uniqueness, recognising that such variation arises partly from adaptations to the environment, but also speculating that there must be a further internal process that guarantees the continuity of some traits characterising a species.

This second idea, *heredity*, was, until Darwin, normally accepted as consistent with Aristotle's founding natural history in which species are fixed, a view that resonated with later Christian teaching that all species were formed according to the original divine plan. Darwin sharply altered this picture, not only because his own extensive observation and compilation of evidence indicated that species are not fixed, but because he saw heredity as isotropic, i.e. a system that exhibits no preferred pathway of development. Hereditary sources of variation are, so to speak, the raw material of change, but impart no directionality. Copious small hereditary variations are observable in successive generations of offspring — i.e. much more variation occurs than is immediately advantageous in competition within or between species. In today's terminology, the additional variation is simply considered "neutral" — until, perhaps, environmental changes make a given trait a critical advantage or a liability. The key question, in any case, was how natural selection operates to promote certain hereditary variants, rather than others. Given the wealth of his own observations, and in the absence of a scientific account of the hereditary mechanisms now known as genetic mutation, recombination and drift, it is not surprising that Darwin's development of natural selection tended to focus on the decisive role of environment-organism relationships that vary across species and sub-species populations, rather than heredity.

The third syllogistic proposition of natural selection, *superfecundity*, further emphasized and reflected Darwin's primary concern to explain the force of variation. Referring directly to Malthus, Darwin observed that species tend to produce more offspring than can possibly survive (1996:54). Malthus's theory had postulated that, as over-supply would lead to competition for food between individuals making up a population, a positive check (i.e. mortality) would necessarily function to remove those members who were unable to compete successfully.

Gould brings these three propositions together in the following syllogism: (i) All organisms are characterised by internal (genetic) variation which is perpetually renewed in changing forms across generations; (ii) Only some offspring survive; (iii) Those organisms survive in which variation, by the action of environmental competition on inherited traits, yields traits enabling survival. Selection is a population dynamic in which species, and the sub-species groups that compose them, are formed and continually changed by the interaction of their members with each other, with other species populations, and with their environments. The deduction at the core of evolution is thus that selection is a creative force occurring naturally to favour the fittest organisms. As environments change, and individual and group actions proceed, and variations arise from this process, the characteristics of organisms and groups may diverge; this variation chiefly accounts for why a species is composed of several sub-populations with variant characteristics, but such divergence may also lead to the origin of new species.

In the course of later restatement of Darwin's programme, Mayr (1961; 1982: 67–72) introduced a simple formulation which helps to understand how this logic of divergence has shaped subsequent evolutionary thinking. His formulation remains widely employed although, as we shall see, it has come to be questioned in some respects that define current frontiers of research. Mayr contrasted the study of "proximate" causes of evolution to those concerned with "ultimate" causes. The former, addressed notably to characteristics of sub-populations within a species, has become the domain of molecular biologists (studying the recombination and transfer of genetic material) and physiologists (studying organic, cellular and sub-cellular mechanisms). Its role in explanation is to answer questions about how systems work, in which technical developments arising from laboratory methods and mathematical modelling since Darwin's time are pre-eminent. Of course, natural historical studies of individual and species adaptation in varying environments have long been concerned with proximate causes. Ultimate causes address why history in the long term has, for a given species, produced one system of adaptations rather than another. Research, for example in systematics (i.e. the natural history and classification of systems of speciation) and paleontology, retain a strong focus on Darwin's concern with variation arising from organism-environment interaction, in which causes are the product of the lived conditions of many thousands of generations of natural selection. Put another way, proximate causes are the immediate factors that determine the selection of genetic materials that occur in an individual and their physiological correlates; ultimate causes are conditions responsible for the evolution of genetic traits and correlates with which every individual of a species is endowed.

Variation and the Problem of the Renewal of Population Heterogeneity

If Darwin's reliance on Malthus is all there was to his population thinking, then evolutionary approaches would have little to add to demography. Indeed, Malthus's theory on its own would not have enabled population thinking in evolutionary biology to achieve its remarkable advances in explaining how and why the characteristics of individuals, and thence sub-populations, diverge. Superfecundity and the positive check remain, of course, key to the general logic of what limits population size and growth, but they are parameters that set only the outer limits towards which population increase in any species or sub-species may tend. The positive check is not in itself a mechanism of agency, only of restraint. It comes into play where environment-organism interactions reduce numbers by eliminating individuals and, ultimately, groups.

The idea of the positive check nonetheless contributed some important dynamic components to Darwin's population thinking, notably as a mechanism of stabilisation, and it also contributed to the centrality of intra- and inter-species competition as an ultimate cause underlying natural selection. The operation of proximate mechanisms, however, remained primary, since environment-organism interaction was crucial both to arbitrating hereditary sources of variation and to when and where the positive check might operate. In other words, evolution as a process of population change is not simply about mortality or fertility, i.e. *population renewal*. To understand how species evolve we need to identify mechanisms that ensure the *renewal of population heterogeneity*, i.e. what enables the continuing flow of new characteristics which can be transformed into adaptive advantages, thence leading to further adjustments in population memberships, composition, size and structure. The syllogistic core of evolution thus gives an incomplete account of a critical element in population thinking that concerned Darwin: how population variation functions as a *creative* force in evolution.

It will help, to begin with, to clarify how Darwin goes beyond Malthus. We can then turn to a key source of the "revolution" he initiated, which drew on a much older model of population thinking that prevailed in the era before the nineteenth-century rise of statistics and demography. More particularly, Darwin relied on analogies to Adam Smith's powerful restatement of the Classical model of population, in which the specialisation and interdependence of individuals — and the sub-populations to which they belong — in the division of labour provide the primary motor of social, economic and population change. Comparison of the two different conceptual approaches of Smith and Malthus as they shaped Darwin's population thinking allows the distinction between two fundamental modes of population thought — open and closed — to be introduced descriptively. We see, firstly, how Darwin brought them together tentatively as complementary components of evolutionary theory; and secondly, the tensions that nonetheless exist between them.[1] Section 3 then turns to the struggle to reconcile these tensions as Darwin's framework was developed in the later nineteenth and early twentieth century.

Darwin's "Malthusian Episode"

Malthus (1982 [1798]) considered that any population is constrained, sooner or later, by the limited carrying capacity of agricultural production in a given terrain. A population, in other words, exists in a fundamentally *closed* environment, and can only expand up to the limits of its productivity. Behaviour leading to population growth in excess of productive capacities, and a consequent and widespread positive check, is immoral, especially as it affects infants and children. He therefore argued that only one demographic response is legitimate: the regulation of fertility via the preventive check, i.e. the delay or foregoing of marriage so that fertility is restrained to levels at or below what agricultural production can support. As Wrigley (1986) has shown, Malthus conceived the operation of the positive and preventive checks as a system of feedbacks: a population as it grows may for a time expand production, but it will inevitably reach the limits of such adaptation, and the humane adaptive response of the preventive check is then necessary. Historical demography, and more recent population history, have shown, of course, that much more than nuptiality control is involved and many other factors may be

1 For historical background to the Classical Model and its subsequent development in population arithmetic, political economy and population renewal theory see Kreager 2008, 2009 and 2017.

important. As Wrigley also notes, Malthus was wrong about the natural limits of agricultural productivity, which was not a closed system, even in his own era (1986: 50–53). Yet the idea that the timing and extent of marriage function as feedback mechanisms that may serve to adjust mortality, fertility and population growth relative to the surrounding environment, has proven apt in some periods of European history, and conceptually fruitful.

Adopting Mayr's distinction, we can see that Malthus aspired to formulate a theory of ultimate causes. His admiration for Newton's law of universal gravitation as a model of explanation is well known (e.g. Flew 1982: 32). While aware, for example, that societies have diverse family, marriage and productive arrangements, and that the positive check may operate to a differing degree in them, Malthus considered such variation a secondary matter, i.e. such factors might delay, but could not fundamentally alter, the ultimate impact of superfecundity, the necessity of the positive check, or the single solution of nuptiality control. The principle of population put forward in his *Essay* thus propounds an absolute, closed and concise model of limits to population to which all must in the end conform.

Such a dismissal of the central importance of variation was obviously of no help to Darwin. Indeed, if the positive check, as the sole and ultimate mechanism of selection, continually removed less successful individuals — with no account being given of how variation renews population heterogeneity — then the long-term evolution of populations would see only the progressive reduction of sources of variation, leaving populations composed of increasingly perfectly fit members in each species. In Darwin's view, however, the diversity of environmental adaptations, together with the isotropy of heredity, guaranteed that natural selection has no such foresight or drive to perfection. Indeed, the reduction of heterogeneity effected by the positive check on its own would have the opposite, disadvantageous, effect by leaving populations vulnerable to circumstances in which environments change.

Darwin's evidence, in any case, showed the contrary: environment-organism adaptation rested on the specialisation of individuals to suit the environment, and as individuals faced competition and colonised new niches, then new specialisations and sub-populations characterised by them were found to emerge. Changing symbiosis with other species also occurred in this process, enabling ever denser development and habitation of a given setting. In this process, population heterogeneity was continually renewed, and this became possible because sub-populations making up a species are not actually closed, but *open* — i.e. they have mating, migratory, and other relations with species members. Both intra-species variation (whether arising, e.g. from mating within a given deme or species sub-population, or between them), and changing competition between species, are entailed. Population heterogeneity and openness are thus jointly critical mechanisms of evolution.

Historians of biology have found that Darwin left notebooks, letters and marginal comments in texts he had read which enable them to trace the development of his population thinking in considerable detail. Schweber (1977: 231–32, 286–96) provides a detailed account of Darwin's "Malthusian episode", and of his subsequent development of biologists' reading of Adam Smith, which gave form to his account of heterogeneity and openness. The Malthusian episode came early in the conceptual development of Darwin's theory (in 1838). At that time, he, like Malthus before him, was strongly disposed to the theoretical ideal in which laws define ultimate determinants. When quantitatively formulated, such laws reveal central tendencies that ensure the stability of natural systems while allowing for many surrounding random

and other fluctuations. He was greatly interested in this form of theory, and not only because the pre-eminence of mathematically defined physical laws was accepted as canonical in the intellectual milieu in which he lived. Of more immediate concern to Darwin was the complexity of his natural historical evidence, which led to the view that, amidst the copious variation that heredity made possible, the process of evolution via environment-organism interaction worked to produce only small and gradual changes within demes and species, normally over long periods of time. This gradualism, together with the uncertainty of the exact nature of the hereditary component (which he assumed to act randomly), led him to the view that divergence of character could only be established with the help of a statistical conceptualisation of change. In other words, the creative agency of environment-organism interactions acting on the flow of hereditary variation should be expressed in terms of predominant frequencies amongst a vast array of different outcomes. In this way it might be possible for biological theory to emulate the general law-like mathematical formulation of the physical sciences.

More particularly, Darwin's interest in Malthus was kindled by accounts of the latter's theory given in Quetelet's (1869 [1835]) social physics, and in contemporary reviews that discussed Quetelet in relation to Malthus, which Darwin studied closely. Quetelet, arguably the foremost European exponent of a new science of population statistics, drew on his experience as an astronomer to propose the idea of "*l'homme moyen*", or the statistical normality of the "average man". Linking this to Malthus's account of superfecundity and the positive check appeared to open up the possibility of formulating deterministic or ultimate laws of society analogous to those of physics. The often-cited passage in Darwin's *Autobiography* (1958: 120), where he remarked on the epiphany that the *Essay on Population* represented in the development of his theory, directly follows the 1838 notebook passages in which he considered Quetelet (Schweber 1977: 293). In short, what Darwin derived from Malthus was not only the ultimate constraint of the positive check. This constraint provides an ultimate causal mechanism for evolution in so far as the will to survive or avoid death becomes the premise on which competition for existence rests. More than this, Malthus's theory appeared as an exemplar of the whole view of scientific theory in which quantitative systems are governed by deterministic laws that allow variation within long-term tendencies to stabilisation.

As Darwin quickly recognised, however, Malthus's checks and Quetelet's statistics of normal tendencies unfortunately left out the critical explicandum of the proximate mechanisms of variation.[2] There is, put very simply, much more going on in the lives of species members, or individuals and groups in society, than competition for survival and the average outcomes of such a process. Not everything that heredity and environment-organism interaction generates is telling for the divergence of demes and species, and even if significant for divergence the effects may only become important later in history. Darwin therefore turned his attention concertedly to the problem of how to formulate a cohesive theory of the creative process of variation.

The logic of the division of labour, which already existed as a model embracing population heterogeneity and openness, and had been remarked by natural historians (Kreager 2015: 76–77), became the focus of his attention. His familiarity with this logic as applied to biological processes emerged by the 1840s in his detailed notes on Milne-Edwards' *Introduction á la zoologie générale* (1851 [1834]), and other writings to which he had access (Limoges 1968,

2 Schweber notes that the review of Quetelet's book which Darwin annotated concluded with remarks on this inadequacy (1977: 293).

1970; Schweber 1980: 249–57). Milne-Edwards worked in an established natural historical approach known as "animal economy", and employed the phrase "division of labour" to explain how organs in the body become progressively specialised. His development of this analogy closely followed the line of reasoning in Smith's *Wealth of Nations* (1976 [1776]). Just as Smith describes how society and economy evolve from hunter-gatherer groups in which all individuals carry out the same productive, military and other functions, Milne-Edwards describes how, in simple organisms like polyps, bodily functions are not differentiated; just as agricultural and commercial societies advance beyond the simplest level of social organisation by developing specialised personnel for agriculture, defence, manufactures, transport and so forth, so species become more complex by developing specialised organs for respiration, digestion and reproduction (cf. Milne-Edwards 1827 and Limoges 1968 to Smith 1976 vol. I: 689 et seq. and Kreager 2017).

Darwin, like Smith, readily came to view the idea of the division of labour as of major importance at the population level. Both authors considered the renewal of the many and heterogeneous groups, and the emergence of new populations with specialisations productive in changing environments, as key motors of historical and evolutionary change. Darwin's elaborate development of the analogy between the creative force of variation and the division of labour in human society is extensive and detailed, and a few examples will have to suffice here to give the reader an idea.[3]

Transferred to the population level, Milne-Edwards' account of the development of specialised organic characteristics becomes an account not only of physiological development in individual members of a group, but of how such greater or lesser divergence characterising species sub-populations translates into their greater or lesser adaptive capacities for expanding into environments available to them, and the heterogeneity of groups that comes to characterise such sites. As these capacities become manifest, accompanying changes in population composition, size and growth follow suit. Darwin, citing Milne-Edwards (1996: 92–98), illustrates his argument by many examples drawn from competition amongst *flora* and *fauna*, leading to his famous diagram of species divergence.[4] Such specialisation, as in Adam Smith's

3 Comparison of Darwin's and Smith's population thinking draws on Schweber (1977, 1980) and Kreager (2017). Schweber further remarks on the status of the division of labour as a widely employed metaphor and model in the mid-nineteenth century. That said, Darwin's debt to Smith's account of population specialisation, interdependence and the renewal of heterogeneity as fundamental elements of the dynamics of evolution should not be overstated. As with his incorporation of Malthus's positive check, Darwin sought analogies that would enable him to think cohesively about observed processes recorded by natural historians — *not* a systematic reduction of biological phenomena to principles supposed to regulate political economy. This is evident merely in the fact that most types of feedback in environment-organism interaction differ from those in the division of labour, and Darwin did not pursue analogies to Smith's population thinking further than its general logic. As we shall see, in providing a place for formal, statistical analysis of variation in his theory, Darwin's logic marked a major advance on Smith, which is central to the "revolution" remarked by Mayr.

4 Thus, "In an extremely small area, especially if freely open to immigration, and where the contest between individual and individual must be severe, we always find great diversity in its inhabitants. For instance, I found that a piece of turf, three feet by four in size, which had been exposed for many years to exactly the same conditions, supported twenty species of plants, and these belonged to eighteen genera and to eight orders, which shows how much these plants differed from each other. So it is with plants and insects on small and uniform islets; and so in small ponds of fresh water. Farmers find that they can raise most food by a rotation of plants belonging to the most different orders: nature follows what may be called simultaneous rotation." (1996: 94).

account of the division of labour, is closely bound up with the interaction or interdependence of sub-populations in a given productive environment. For Smith, the specialisation of tasks in the division of labour both develops individual capacities and characteristics, and requires many productive groups to work in close interdependence; expanded capacities for individual agency and production enhance general living conditions for the several sub-populations involved in a given productive niche, affording them competitive collective advantages in their wider environment; this entails not only enhanced economic but social agency. Specialisation and interdependence of constituent populations making up a society are thus a principal motor of their own and general social change, and the integral role of population composition, size and growth in the evolution of economy and society is explained by Smith in these terms. In essence: "the number of workmen in every branch of business generally increases with the division of labour in that branch or, rather, it is the increase of their number which enables them to class and subdivide themselves in this manner" (1976 vol. II, Introduction, p. 277). Population, in other words, tends to increase in sub-populations working in tandem in a given economic sector or sectors, and this becomes a motor of population growth in society more generally. By analogy, Darwin "chose the principle of optimalisation of the amount of life per unit area as the overall explanatory principle" (Schweber 1980: 288).

Thus, both Smith and Darwin considered (contra Malthus) that the growth of a population was not only key evidence of its competitive success, but that such growth was itself a principal mechanism of improvement. Competition at the individual level may ultimately be decisive, but is conditioned by the structure of interdependence between populations, which conditions the circumstances in which an individual acts. Darwin therefore, like Smith, considered the positive check as functioning in *proximate* terms, that is, as conditional on environment-organism interactions and on the nature of relationships within and between *local* populations — rather than, as in Malthus, an *ultimate* or universal mechanism to which all populations and all members must sooner or later answer in a particular way. Rather than fundamentally and ultimately closed, population dynamics are by nature open, as groups exist in manifold relationships and interdependencies with other groups. These interdependencies, as in Smith's analysis, give Darwin's account a much more extensive set of organism-environment feedbacks than Malthus's singular stress on the positive and preventive checks in an ultimately closed environment. As Schweber remarks, Darwin's whole approach reflects a critical difference between Smith's account and Malthus's: individual species members have much more agency in dealing with proximate causes than is possible under Malthus's emphasis on the ultimate necessity of his two checks on population (1977: 283; Kreager 2017: 531).

This emphasis on the agency that diverse group members exercise in producing variations followed directly from the much more extensive body of direct observation of adaptive processes that characterised Darwin's natural history, in contrast to Malthus's political economy. For Darwin, explaining processes of population change rests first on empirical identification of proximate causes, as these arbitrate the possible operation of ultimate positive checks. Put another way, the inter-relationships between groups in a given environment requires a bottom-up perspective: explaining population dynamics begins in observation at lower levels of aggregation, since changing group compositions and interrelationships carry implications for higher levels of aggregation, both in the short and long term.

Two Concepts of Population

Darwin's quantitative skills remained those of a botanical arithmetician, employing methods similar to those of seventeenth- and eighteenth-century population arithmetic; his notebooks make clear that he was not adept at higher mathematics.[5] Yet, as we have seen, his population thinking was prescient in understanding that the different conceptual approaches to population underlying Smith's and Malthus's works are both necessary to explaining how populations evolve. From the former Darwin took the idea that populations are by nature open and heterogeneous, variation arising in them from the interdependence of groups and their members, which he saw as analogous to the way specialisation functions in the division of labour. Such open population thinking was a breakthrough in showing how the vast body of his natural historical evidence could be generalised at the population level.[6] It did not, however, satisfy the scientific criteria expected of theory in the milieu in which he wrote. As remarked earlier, the middle decades of the nineteenth century were an era in which the rise of population statistics led to its proposed formalisation as a social physics (Porter 1986).

Lacking close familiarity with the new methods, Darwin nonetheless responded to this second idea of population by drawing on contemporary views of Malthus's *Essay*, in which the impact of the positive check was understood as imposing absolute limits on population suitable to developing methods of social physics. The mortality of the positive check could be used to define limits to growth for any population given the particular environment in which it is found. Darwin hoped such an approach would enable statistics to demonstrate changes in the frequencies of specific evolutionary traits.

As we shall see, this proposition proved very difficult for Darwin's followers to develop in the late nineteenth and early twentieth century. Before turning to their several formulations — some brilliant and of enduring importance, whilst others have come to be recognised as not only dubious but dangerous — we can at least try to state succinctly the fundamental problem posed by Darwin's dual approach to population: his open population thinking, grounded in natural history and the role of environmental constraints in shaping evolution, is analytically distinct from, and may even seem opposed to the closed populations on which formal modelling depends. The extent to which Darwin was himself aware of this difference remains uncertain. It was brought out at least as early as the 1920s by the doyen of twentieth-century formal demography, Alfred Lotka, whose mathematics of population ecology (1925) was one of the earliest evolutionary formalisms to be established, and then extended to human fertility and mortality (1934; 1939). What Darwin appears to have been the first to recognise, at least implicitly, is that the two concepts, even if radically different, are nonetheless complementary. How, then, did he bring open and closed population thinking together? What problems then remained, that generated such variously seminal and flawed approaches amongst his followers?

5 For example, in making an estimate of relative frequency, Darwin made multiple calculations each based on different ways of proportioning a population, and then compared the results — in effect, reinventing a method Graunt had devised two centuries earlier (cf. Browne 1980; Kreager 1982).

6 Darwin adopted Classical population thinking not only because it was a model that was integral to Smith's account of the division of labour and early quantification of human society, but because it was also established practice in natural history. Thus, in his extensive comparative study of barnacles (1851), he drew on the large body of data available in natural historians' plant catalogues, and the established field of botanical arithmetic, in which counting and comparing physical characteristics was a standard practice (Browne 1980).

The closed character of formal population analysis and data systems needs, of course, no introduction to a twenty-first-century audience of demographers and population geneticists. Lotka used his training in physical chemistry to argue the generality of this scientific methodology.[7] Whether in thermodynamics, census-taking, life table construction or the theory of population renewal which was Lotka's own contribution — the individuals making up a population are treated as identical subject to the system of classification employed. Just as the behaviour of atoms and molecules conforms to the rules of the periodic table, so the population movements of human beings conform to the fixed set of statuses given, for example, in a census schedule. Censuses, like the periodic table, have the considerable advantage of being effectively comprehensive, thus enabling exhaustive and purely formal analysis of all changes of state between recognised categories. Once born, an individual can only move between classificatory statuses: he or she gets older, marries, establishes a household, has children, changes occupations […] and eventually dies. Closed units, whether of the total population under analysis, or of any of its component sub-populations, enable aggregate states of population change to be calculated precisely: age and sex structures, gene frequencies, life expectation, trends in fertility, mortality, labour force participation and so forth. This approach, which Mayr and other contributors to Evolutionary Synthesis referred to as "typological" or "essentialist" (1982: 47), is immensely powerful once species and demic populations have been identified.

Darwin's *Origin* was, however, concerned not only with the renewal of existing populations but *the renewal of population heterogeneity*, since evolution proceeds by continuing adaptation and consequent variation in and between populations. To begin by treating populations in nature as closed is artificial. As natural historians had long recognised, species rarely present themselves as discrete groups in nature. Sustained observation is a first necessary step, to identify the role of environmental factors in shaping variation at local levels. Such open inquiry decides which characteristics should be tracked, and in which environments. Identifying the relationships between individuals that appear to constitute membership of a species involves repeated hypotheses and continuing observation to test them, until the unity of a proposed species can be considered established. Of course, the rise of genetics since Darwin's time has provided further laboratory methods of observation that greatly assist identification. Nonetheless, the primary questions necessary to track variation and possible divergence remain: "What is a population (i.e. for the purpose of differentiating organisms in the process of variation)?"; "What set of sub-population units comprise a species?"; and "What relationships account for their differences?"

Natural historians up until the late nineteenth century employed enumeration as part of open population thinking, i.e. accepting that an exhaustive or complete counting was only exceptionally realisable. Similar to Graunt and other early modern population arithmeticians, totals could be compared without formal mathematics and without comprehensive census inventories. Where characteristics appeared to clearly differentiate groups, they were accepted

7 Lotka sets out the analogy between species populations and those of molecules carefully and elaborately, emphasizing that his analysis is confined to "isolated systems" (1925: 26) and that, as in all probabilistic models, possible events and relations are limited to those specified by classifications in advance (1925: 35; 41). When he later came to develop the model for human fertility and mortality, he was able to say more simply that analysis by definition is confined to "closed populations" (1939:11). The following very brief summary can scarcely do justice to his extended presentation.

as indicating the presence of distinct phenomena whether the entity was a plant or animal in the wild, a human being recorded as dying of a specific disease in the "mortality bills" that were the only record of causes of death before the modern census era. Darwin, in effect, moved on from his predecessors by the simple step of accepting that a carefully observed body of numerical evidence at the population level — however provisional — was effectively complete. This appeared to be a substantial improvement on previous practice that was not population-based, in which whole species and higher types might be proposed from merely a few specimens.

Darwin's reasoning, as we have seen, addressed variation and the divergence of species in terms of optimalisation: the amount of life that could be supported in any setting would become greater, more diverse and complex as competition intensified; larger genera, species and demes would tend to produce more hereditary variation, in which those offspring with more diverse characteristics would have additional advantages to adapt and increase their numbers. Such a local "division of labour" in this way provided a plausible account of how particular adaptive advantages could accumulate at higher levels of aggregation, further encouraging Darwin to think at the level of populations. As Schweber remarks (1980: 288), Darwin's premise that the quantity of life is gradually optimised in local environments effectively bypassed the difficulties of integrating different levels of description: local observation of open populations, in which characteristics are gradually differentiated as inquiry proceeds, seems to flow seamlessly into later analysis in which units of population might be defined formally as distinctive demic and species populations, i.e. treated as closed for purposes of statistical analysis. Indeed, as an empirical procedure for generating and testing hypotheses, this logic appears straightforward. Hence Darwin could hope that emerging emerging statistical techniques could be applied to variation and divergence, even if his own understanding did not extend to how formal models and data systems are actually constructed.

What this way of thinking assumes, however, is that the role of open population thinking — sustained local observation of proximate relationships in order to differentiate units of population — has been carried out prior to statistical modelling. With the benefit of hindsight, we can see that, for a scientist of long natural historical experience like Darwin, this "bottom up" approach was so elemental it could not ever be questioned. Formal analysis, however, can take place whether or not sustained observation has given an empirical ground to hypotheses, and whether or not local processes are translated accurately into specifications of closed population units entailed in large datasets. We turn now to subsequent developments in evolutionary theory, which proved to be fraught with controversy. Apparently powerful arguments claiming to establish ultimate causes were built on the basis of classifications and units of measurement not grounded in observation of proximate causes. Arguably, one of the critical lessons from the emergence of evolution as a population theory is that confusion and ambiguity proliferate where the different roles of the two concepts of population, and their engagement at different stages of analysis, are not recognised.

The Early Struggle to Incorporate Population Genetics and Demography into Evolution

In this short account, we will consider two contrasting approaches of the late nineteenth and early twentieth centuries which illustrate this issue: Galton's attempt to use evolution to build a science of eugenics; and the several contemporaneous movements in early-twentieth-century

population thinking that led to the consolidation of demography as a discipline, and gave its methods a fundamental role in population genetics as a core component of the Evolutionary Synthesis. This period stands as something of a paradox for demography: as the discipline gradually took its contemporary shape, its formal methodologies made significant contributions to population ecology, to the critique of eugenics and to the formulation of the Synthesis — yet its professional stance became increasingly separate from evolutionary biology.

Galtonian Eugenics

Darwin's need to treat heredity as a "black box" in his theory led to an immense amount of speculation and exploratory research (Provine 1971). As Porter (1986:280) remarks, Darwin's own later ruminations on this problem were "virtually a complete failure amongst biologists", although they attracted the attention of the biometric school developed from the 1870s by Galton and Pearson. Both men were remarkable polymaths whose life work focussed on developing advanced statistical techniques to track the evolution of hereditary and racial differences, which they then put forward to legitimise highly controversial public policies. Darwin had speculated that hereditary took the form of particles or "gemmules" that circulate in all parts of the body, transmitting specific traits particularly in the course of embryo formation (1868). He was led to this in part by questions concerning the role of evolution in shaping human society. Galton, for whom the latter concern was paramount, redeveloped the gemmule hypothesis not as a matter of embryology, but as a demographic phenomenon. For this purpose, he prioritised the Malthusian component of Darwin's theory — superfecundity — as the primary force in evolution. Reproduction, Galton argued, arbitrates the role of heredity in human and social development, since varying levels of fertility within and between groups in a population determine which, and how widely, certain hereditary characteristics rather than others come to predominate; fertility differentials constitute "reproductive selection", the importance of which is vastly greater as a factor in natural selection than environment-organism interaction. He coined the term "eugenics" to refer to an ostensibly scientific and statistical practice that would ensure that only the best babies could be born. Not only should those judged the most fit members of a society be encouraged to reproduce, steps should be taken actively to restrict the fertility of less desirable groups. As Galton repeatedly emphasized, the majority of offspring were being produced by only certain (lower-class) groups in society. Eugenics rapidly became highly topical in an era in which European reproductive levels had for the first time begun to decline radically.

To understand the impact of reproductive differentials on the quality of human populations, Galton needed a statistical method that could discriminate between more and less powerful influences on genetic transmission. This led to his famous conceptualisation of statistical correlation and regression, and their formal mathematical development by his associate, Karl Pearson. Both concepts, of course, have come subsequently to play a widespread role in population research. Their use in eugenic argument, however, relied on institutional and popular definitions of social class differences, ascribing ultimate causes to them without examination of their empirical basis.

While insisting on their allegiance to Darwin's and Malthus's theories, Galton and Pearson argued that there were crucial flaws in their reasoning. Darwin, as noted above, did not give sufficient attention to reproductive selection. Malthus did not go far enough in his criticism of a supposed lack of sexual restraint among the poorer classes; while recognising that their superfecundity leads necessarily to the action of the positive check, he did not ask whether that

check would actually be sufficient, i.e. whether there would still be a great majority of lower-class children relative to those of higher classes. The normal function of mortality Galton and Pearson termed "the selective death rate", i.e. the ultimate mechanism of natural selection in weeding out the less fit. Average family size had, nonetheless, remained higher in lower-class "degenerate and pathological stocks" (Pearson 1912: 27). Hence the dire prospect, if the positive check did not remove the greater majority of lower-class children, of their superfecundity of surviving children to greatly outnumber those of "the cultured and highly sensitive upper and middle classes"; the outcome would be "race suicide" which, "in the inmost recesses of history [...] explains the fall of great world-civilisations" (Pearson 1912: 10, 39).

The central issue, for eugenic argument, was thus how to demonstrate this calculus. Correlation, Galton remarked, provided the method demonstrating "the closeness of the relation between any two systems whose variations are due partly to causes common to both, and partly to causes special to each" (1907: 174). Pearson's mathematical development, appearing in his note on reproductive selection to the Royal Statistical Society (1896), begins with statistical demonstration of the correlation between fertility and organic characteristics across generations. For this purpose, he employed a classic measure in social physics — height — in this case of mothers, daughters and wives in "1,842 families of Danish race". Pearson showed a regular percentage change in height across generations; he would later describe such variation as an instance of "the law of ancestral heredity", i.e. the change of any organ or physical or mental characteristic that typifies its spread in a large population over time (e.g. Pearson 1912). The question, then, was what part of the Danish population was contributing most to such changes. Analysing net fertility (i.e. allowing for infant and child mortality, and for non-marriage) in artisan and professional classes, Pearson concluded that while the former represented only 27 per cent of the population, its greater fertility produced over half of the younger generation. In short, on this account reproductive selection is the much greater factor than natural selection (i.e. as defined only in terms of the selective death rate) in population replacement and change.

Pearson's "The Problem of Practical Eugenics" (1912), is one of many articles in which he developed this mathematics of correlation as a basis of demographic policy, particularly in the context of the fertility declines now commonly known as demographic transition. Anticipating later demographic interests, he was particularly concerned with the economic value of children, notably the impact of factory legislation which had removed the value of child labour as a component of working-class family incomes. His analysis assumes the "law of ancestral heredity", and is directed particularly to showing that well-intentioned government policies supposed to improve the environmental conditions of factory populations are much less important to national development than their impacts on heredity. He traces fertility declines in the Registrar General's data for a number of manufacturing towns and rural areas, particularly in the period 1870–1905, in relation to the several Acts that prohibited child labour. He notes not only the steep decline in birth rates, coupled with the still relatively larger family sizes of the working classes, but levels of tuberculosis, insanity, deafness and other conditions he considers pathological, calling attention to their incidence by birth order. As these conditions are markedly more common in the first, second or third child a woman bears, Pearson concludes that not only are working classes producing a higher percentage of the population, their reproduction ensures a higher percentage of "cacogenic" stock overall. Meanwhile, the upper and middle classes have come to have an "artificial birth rate" in consequence of their inclination to lower fertility in the

context of changing economic conditions. Pearson then traces the implications of these several developments in relation to demographic topics that have proven of long-term interest, notably contraception and ageing. More immediately, Pearson advocated major changes to taxation, (raising rates on income, estate and inheritance for the childless); while factory legislation should not simply be repealed, its continuing impact on the "racial efficiency" of the population could only be countered by amplifying the numbers of "well-born children".

Both Galton and Pearson played major and respected roles in scientific organisations of the time, and both were offered knighthoods.[8] Their eugenics is a reminder that distinguished authors claiming to be followers of Malthus and Darwin may, in fact, be promoting theories that are hardly consistent with such claims (cf. Kreager 2014). On the statistical side of population thinking, there can be no doubt that their work constituted a serious and imaginative attempt to address fundamental problems of conceptualising and measuring structural changes in frequencies across generations, of the logic of population stabilisation given incomplete genetic data, and of the incremental or "small steps" by which genetic variation influences population change. Their technical insights, however, were vitiated by two radical departures from the evolutionary structure of population thinking that Darwin had carefully developed: their predilection for arguments based exclusively on ultimate causes; and their sole reliance on closed or typological population thinking.

In eugenics, heredity displaced Darwin's emphasis on environment-organism interaction in the study of variation. Pearson considered heredity "more potent", adding acerbically that population policy makers should know that a stud productive of Derby winners does not rely chiefly on improved stables (Pearson 1912: 36, 38). Eugenicists' pursuit of heredity as the seat of ultimate causes was, moreover, built upon incomplete and ambiguous definitions of the human sub-populations treated in published statistics, and the more or less complete exclusion of sub-population interactions that, as proximate causes, were crucial to Darwin's view of evolution. The populations Pearson employed were drawn from standard institutional sources in which classification rested on criteria not informed by observation of how groups are formed, sustained and related over time. Given Pearson's "cacogenic" arguments, it is also evident that the classifications selected were in consequence all the more susceptible to powerful class and other biases. Pearson in effect extended closed population thinking to human heredity in ways that run counter to Darwin and Mendel: all genetic and physiological characteristics other than those mentioned above were taken to be identical for all individuals in each given population type; all genealogical or other links that show members' involvement with other populations were not considered; and change over time was always directed, i.e. not isotropic.[9] In the end, the eugenic exercise excluded a vast array of sources of variation, and was strongly tautological: those groups with higher birth and death rates were categorised from the start in closed classifications as "cacogenic" or "degenerate".

8 Pearson refused, being a socialist.

9 Pearson admitted in a footnote (1896: 398–39, n.4) that his statistical approach via correlation puts aside Darwin's central concern in the *Origin* with how variation can give rise to new demes and species. Pearson reduces fitness to progressive change in extant species defined as composed of homogeneous social classes.

Vital Statistics, Population Ecology and Genetics: Some First Steps toward an Evolutionary Demography

As we have seen, Darwin's conviction that statistical evidence is essential to understanding evolution as a process of population interaction embraced both numerical observation at the local level and the potential for modelling aggregate frequencies at higher levels of analysis. In evolutionary biology, the famous breakthrough that swept away eugenic and many other arguments came at the local level: the rediscovery in 1900 of Mendel's experiments on genetic variation in peas. Mendel's work differed sharply from the eugenicists in the careful observational method used to establish sub-populations and the nature and structure of their relationships.[10]

With the benefit of hindsight, the early biometricians' eugenic project may be said to have occupied a kind of median position between the continuing research of evolutionary biologists and the much wider period concerns about the potential impact of declining birth and mortality rates, race, and migration on national population composition and replacement. Controversy over the role of reproductive selection embraced vital statistics, public health, biological anthropology, sociology and a great many essays (variously of socialist, conservative, feminist and other persuasions) written for general audiences (Soloway 1982). The issue was one of general public concern. Eugenics, with its technical claims and dramatic highlighting of demographic differentials as simultaneously social and genetic determinants, attracted widespread attention, and was without doubt a major stimulus both to controversy and to recognition of the need for more critical, observation-based approaches. Developments in social and vital statistics were, of course, for the most part of a fundamentally different kind from Mendel's work, since they relied on closed-population datasets established during the nineteenth century with the founding of national statistical offices and professional statistical societies, and the dream of a social physics.

These data provided the foundation for several environmental reforms, including those Pearson attacked, and for declines in mortality related to these reforms at all but the youngest age over the later nineteenth century. They also, as we have seen, provided primary evidence of fertility declines. Vital and social statisticians thus felt a strong need to respond to the eugenicists' arguments, but they also faced uncertainty regarding the specific mechanisms underlying differentials in fertility and mortality between social groups. Their response was to tighten and extend the actuarial approach on which demography rests. This response was

10 Although outside the immediate topic of this chapter, Mendel's method independently encapsulated the combined open and closed population reasoning that Darwin pioneered. Peas of seven seed types were selected, merely on the basis of visible distinctive characteristics (smooth, wrinkled, white, etc.). This selection amounts, in effect, to a pragmatic hypothesis that such features indicate genetic variants. The seven types were planted, and numbers of offspring consistent or variant with the original types noted in the outcomes for each planting. Self- and cross-fertilisation of offspring were then carried out in regular combinations across a succession of generations, and the outcomes enumerated. In this genetic demography of peas, the question 'What constitutes a population or sub-population?' is left open, and the specification of the several sub-populations emerges as a key result from observation, including the ratios that give the regular proportions of dominant and recessive forms that arise from the relationships between them. In effect, the behaviour exhibited in the experiments sorts the population into recurring groups defined by their observed qualitative and quantitative properties (of which the most famous is Mendel's is 3:1 ratio expressing the incidence of dominant versus recessive traits); such regularities then become properties that can be tracked and modelled in wider surveys and in other populations.

characteristic of three major innovations in which demographic methods became fundamental to addressing problems in population biology in the early decades of the twentieth century. The first emerged in part as a response to eugenics, while the second two were driven by problems in evolutionary biology.

The first development, reflecting concerns over differentials in declining fertility, led the General Registry Office (GRO) in England and Wales to put the need for a comprehensive social classification scheme on its agenda. As Szreter's (1986) study of the GRO's programme has shown, its class schema was designed to refute eugenic arguments, although the alternative mechanism put forward to explain fertility declines (the rise of contraception) remained inadequately documented. In addition, a detailed family census was conducted, in 1911, which included more variables, such as parity, than existing censuses. As Szreter (1986: 538–40) remarks, the GRO social class scheme, which remained largely unchanged until the 1970s, continued to reflect several problematic eugenic assumptions which reduced the forms of variation that could be tracked. In short, the immense improvement in data and measurement techniques remained dependent on statistics that track sub-populations defined by occupational, provincial and other conventionally pre-determined, closed administrative units. Relations within and between such groups that involve, for example, gender, labour sectors that combine several occupations, and regional cultures and economies, may not be captured accurately in standard administrative units. Subsequent research reanalysing closed data to reflect non-standard units has revealed major fertility differentials and patterns of variation that conventional classifications missed (Szreter 1996; Garrett et al. 2001; Pooley 2013). As evolutionary biologists would expect, population heterogeneity remains strongly characteristic of modern fertility and mortality trends, including the great diversity in patterns of decline. An approach based on *a priori* closed classifications and units has, by itself, not succeeded in establishing the several theories put forward to explain demographic transition, and this problem continues to this day (Cleland and Wilson 1987; Pollak and Watkins 1993; Demeny and McNicoll 2006).

A second major demographic development of the early twentieth century, Lotka's stable population theory, was conceived as a new foundation for the mathematics of evolution. Lotka carried social physics a step further, reasoning that stabilisation in human and molecular populations is analogous, so that the second law of thermodynamics can be used as a model for formal demography. In the *Elements of Physical Biology* (1925), Lotka successfully applied his approach to relations between species, leading to what are now called the Lotka-Volterra equations which provide the basis for studying predator-prey relations. While providing a central and fruitful framework for population ecology, such models address species-level phenomena without attention to intra-species variation, leading Lewontin to remark that they "are both overly specific and arbitrary in their mathematical form so that they may not catch the important reality of interactions" (2004: 15).

More generally, the approach shared some important limitations with Galton and Pearson's work, which have kept it from becoming the general mathematics of evolution that Lotka had hoped to provide. First, because Lotka sets aside the role of intra-species divergence in the renewal of population heterogeneity, his work remained marginal to central debates in evolutionary theory after Mendel, i.e. the problem of how to integrate genetic variation into population thinking, in which heterogeneity arising from environment-organism interactions

remained fundamental. Second, Lotka largely ignored actual processes and variation in organism-environment interaction. Biologists have more recently remarked that the assumption in which the environment acts on the organism as an autonomous force is simply unrealistic: such a view implies that fully formed niches exist waiting for organisms to come to live in them. This assumption is conducive to closed population thinking, since nothing beyond the premise that self-contained environmental units exist in nature is required. Such a view is, however, deeply troubled by evidence that organisms play an active role in constructing niches, so that organisms and environments co-evolve (Lewontin 2001; 2004: 13–16). The same lack of realism arises in human populations if considered in conventional Malthusian terms in which there is a fixed carrying capacity for any environment (Odling-Smee 2015). Reconciling these more recent criticisms with the continuing utitlity of Lotka's work for population ecology appears to be an ongoing subject of debate.

In the first volume of his principal demographic work, the *Théorie analytique des associations biologiques* (1934), Lotka reiterated the biological foundation of his approach as stated in the *Elements*, together with his careful emphasis, noted earlier, on the purely formal nature of closed analysis. The second volume of the *Théorie* (1939) then developed an extensive application to human populations without reference to other species. Lotka showed how his theory enabled demographers to integrate fertility into the style of analysis used in stationary, or life table, methods, yielding intrinsic growth rates in which purely formal population units, regardless of variation in their initial age/sex structures and vital rates, tend inevitably to stabilise over different time periods. Lotka's later work remained subject to the limitations consequent on exclusively closed population units, just noted.[11] Although post-war social demographers (e.g. Ryder 1964) expressed considerable interest in the possibility of developing Lotka's method as a basis for a general sociological theory of population, its limited focus on population renewal, rather than the renewal of population heterogeneity, and its insensitivity to environment-organism interaction, have meant that many sources of variation cannot be integrated into his formal analysis. These commonly remain "independent" economic, cultural and other variables, often analysed via correlation and regression techniques. Thus, although Lotka greatly clarified and subtilized the formal nature of demographic analysis, and did not reduce variation solely to Queteletian normality, the problem of explaining diverse mechanisms of variation and integrating them into models of population stabilisation has remained.

The third major development combining demography and population biology arose in central evolutionary debates over the implications of Mendel's genetic research for Darwinian population thinking. Demographic models were integrated into genetics in Fisher's *Genetical Theory of Natural Selection* (1930), in which he postulated a species in which reproduction occurs continuously in stable age distributions so that, as in (but independently of) Lotka's formulation, life table probabilities and probabilities of birth in a given interval can be combined in a single equilibrium model. Parallel contributions by Wright (1930) and others (see Provine 1971; Lewontin et al. 2003)) moved this approach toward later population genetics by demonstrating the importance of gene or allelic interactions in local populations, encouraging a return to the Darwinian view of species as aggregates of sub-populations (i.e. effective breeding populations, or demes), and of hereditary influence as a consequence of complex interactions

11 In order to treat human populations without reference to their environment, Lotka made a number of further assumptions which have subsequently been disproven (Kreager 2009: 474n)

or combinations of genetic material. This research put a final end to the eugenic quest for simple demographic laws of fitness; rather, while many demographic parameters may combine to shape fitness (e.g. population density; the relative frequency of genotypes, or the mixing of genotypes, in a population) they do so in many different, shifting combinations with other adaptive factors.[12]

Hiatus: the "Separatism" of Demography from Evolutionary Population Biology

All four of the above developments marked an increasing focus of research on fertility and its place in the transmission of characteristics — whether social or genetic — across generations. Without doubt, there was a growing intellectual convergence that brought early twentieth-century demography into closer alignment with evolutionary biology. Yet only the latter participated in the Evolutionary Synthesis that emerged in the 1930s and 1940s, the culmination of half a century of research that brought mathematical modelling of demographic, genetic, cellular and ecological processes into alignment with Darwin's theory (Mayr and Provine 1998). As the 1950s and 1960s proceeded, no comparable synthesis emerged in the demographic study of human populations, and even demographic followers of Lotka eschewed his evolutionary arguments and applications. Instead, demographers' growing preoccupation, as is well known, was with theories of demographic transition, in which population biology attained only a secondary role in the biomedicine of mortality and fertility control, and related "proximate determinants". Evolutionary biology as a major conceptual source of theory and method was strikingly absent when demography's central post-war concerns came to be established, a neglect that largely continued up to the 1980s (Sear 2015a). The irony, as Lewontin (2004:10) observed, is that once Fisher had put demography at the centre of the genetics of natural selection, evolutionary biology and demography went their separate ways.

Historical accounts have attributed the emergence and powerful influence of separatism to demographers' aversion to eugenics in the aftermath of national socialism, together with the pressing agenda of post-war reconstruction and fears of rapid population growth. These were indeed important factors, and have been discussed elsewhere.[13] A more important consideration,

12 Lewontin (2004: 13) describes this as "a lack of transitivity in fitness": "Competing genotypes can play a game of 'scissors-paper-stone' in which genotype A is superior in competition with B and B is superior to C but C is superior to A, because in each competitive interaction a different set of attributes is involved: A is stronger than B, B is faster moving than C and C is more aggressive than A."

13 The view that separatism arose largely from post-war demographic aversion to eugenics and its pre-occupation with rapid population growth, for example in Kreager (2009), neglects four key factors, of which three are evident in the preceding discussion. One is that the GRO's extensive work to refute eugenics shows that early twentieth-century demographers were already strongly critical. Secondly, eugenicists' claims that their work was a contribution to Darwin's theory were unfortunately not refuted adequately by Galton's contemporaries even though, as we have seen, the fundamental premises of eugenics were a travesty of Darwinian theory. In the absence of such clarification, the confusion of evolutionary approaches with eugenics continued to influence some demographers over the whole first half of the twentieth century, e.g. Pearl (1925). Third, while demographers took Lotka's mathematics seriously, they jettisoned its evolutionary rationale. We may wonder whether they understood clearly that his biological application concerned ecological issues marginal to central issues in evolutionary debates; again, separatism occurred on the basis of limited awareness relating to a biological sub-field, not with reference to mainstream evolutionary population thinking. In short, the separation of demography and

however, is that the long struggle to construct population genetics and integrate it into Darwin's concept of natural selection had a major impact on how ultimate and proximate causation in evolution are understood. This, in turn, changed the role of mathematical modelling in evolutionary theory in ways consistent with Darwin's work on the divergence of character, but counter to the old Newtonian ideal of theory as a mathematical formalism of ultimate physical relationships. Demographers' non-involvement in the Synthesis meant that few were cognizant of these developments, and that mainstream approaches to population theory and methodology remained, as Hauser and Duncan noted, aligned to physics (1959: 15).

As we have seen, ideas about scientific theory, from Malthus through to Quetelet and Lotka, gave pride of place to the goal inspired by physical sciences of mathematically formulated, general-law-like systems. Darwin was from the beginning sympathetic to this view, and continued to leave open the possibility that statistics could provide methods for modelling the frequency of intra- and inter-species variation. The complexity of open population dynamics, and the "black box" of heredity, however, meant that no formal statistical laws could be put forward in his account of speciation in the *Origin*. In the period from the rediscovery of Mendel to the Synthesis, population genetics employing mathematical methods and closed populations became mainstream in evolutionary thinking, even while commonly seen as opposed to natural historical approaches and the more traditional, predominantly open, Darwinian logic of population thinking. Increasingly, however, the methods developed by Fisher, Wright and others were brought into closer alignment with the observational approaches of natural history and physiology via laboratory research. Species selected for experimentation, like *Drosophila* and small mammals, were chosen because they appeared to open up comparative research on promising hypotheses arising from natural observations (Kohler 1994). Once Mendelian features were established, demes and species populations could then be raised in lab conditions as closed populations for testing purposes, many trials becoming possible because such populations could be reproduced quickly. The role of mathematics in tracking the changing frequencies of genetic characteristics under different mating patterns was to build local models that indicated further hypotheses and tests in which genetic traits and changes could be isolated. The results increasingly moved natural historians and mathematical genetics closer together. On one hand, models such as Fisher's and Wright's established key natural historical arguments, notably that Mendel's results were consistent with Darwinian population thinking. Experiments in natural, as well as laboratory conditions became possible. On the other, mathematical approaches were freed from the nineteenth-century dogma that biological theory should be built primarily along the lines of a physics of ultimate causes. Population genetics could be modelled once mathematics was applied to proximate mechanisms, further removing the dangers that eugenics had exposed in trying to postulate ultimate demographic and genetic laws of evolution. The convergence of approaches also removed, at least for a time, any suggestion of genetic transmission of environmental characteristics, thus helping to focus attention on molecular structure as key to the chemistry of genetic transmission. Watson and Crick's DNA model followed in 1953, and with the rise of genomics, the mathematics of gene sequences can be used to hypothesize and model combinations of genetic chemistry in local parts of the genome that enable laboratory observation and exploration of proximate causes of gene expression.

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evolutionary theory was established over the early decades of the twentieth century, and then reinforced by post-war demographic concerns.

Emergent Evolutionary Demography

The approach to general theory in population biology that has emerged from the Synthesis thus remains a methodology that combines insights from closed and open population thinking, not a quest for a universal formalism of evolution. The fundamental open population question, "What is a population?", still has to be answered whenever the quest is to identify mechanisms of genetic, environmental and phenotypical variation, and this usually requires observation or laboratory construction of local populations (Kreager et al. 2015). The role of formal mathematical approaches, however, has greatly expanded, for example via models that simulate the implications of particular genetic or environmental variations for population composition, structure and change. This is most obviously necessary in the context of genomics: with billions of base pairs, and even more possibly significant combinations of them than persons to which they can belong, the "What is a population?" question becomes "*Which* population?", i.e. which set of genetic and other parameters, out of the many possible combinations, can be observed to function as proximate causes leading to expression of characteristics that define a population?[14]

As Wachter (2015) observes, the route to defining actual populations increasingly proceeds via hypothesized populations. Thus: hypotheses arising from incomplete evidence at higher levels of aggregation in the genome are used to model "local population spaces" in which tests may be carried out, and this activity is likely to precede and accompany successive hypotheses/empirical trials in which key sub-population characteristics are gradually isolated (Lewontin 2004: 17–18). Specifying the population is a critical step in research, and the approach as a whole combines top-down and bottom-up research strategies, as models specifying population characteristics are revised on the basis of each round of evidence.[15] The Synthesis, in short, is not a static paradigm, but has continued to evolve. Evidence, for example, questioning the idea that environmental niches can be modelled simply as closed entities given in nature, has led to reconsideration of Mayr's ultimate/proximate distinction so that it may better allow for feedback processes (Laland et al. 2010, 2011; Huneman et al. 2017). Such developments are of obvious interest to demography, as they encourage study of how social and cultural relationships are integral to natural selection as part of feedbacks with the genome and the environment.

The recent renewal of demographic interest in evolutionary biology as a source of concepts and models has grown up in this dynamic situation, where the critical role of collaborative research is once again recognised as necessary. On the demographic side, an impetus has also undoubtedly come from the huge problem, noted earlier, of the unexplained heterogeneity of demographic transitions. The "*Which* population?" question here is broadly analogous

14 For example, a population of haemophilia sufferers can be identified on the basis of a single gene, but in the study of cancer or multiple sclerosis the genetic component is much more complex, and there remain serious questions as to environmental influences across the life course which vary between individuals.

15 Spencer (2015) considers the importance of not grouping population members on *a priori* criteria as a concern in current genomic research. While the iterative approach to modelling just described is commonly employed, he remarks on "the unease we have with describing the continuums of diversity of organisms like humans as discrete groups" (2015: 502), and continues by pointing out that if, "in fact, genotype data are available for each individual within the sample […] why not model each individual as a 'population', and let the covariance in alleles between individuals capture the population structure?" (2015: 512). In such a local model space, use of an individual-level correlation matrix avoids having to define populations other than as individual genomes; the set of principal components thus established constitute clines of genetic variation, which may then be explored in a wider sample of individuals. As Spencer says, "every man is an island (or at least a population)" (2015: 512).

to that described for genomics, above. A theory of transition was initially assumed to be universal: modernisation would explain how social, cultural, economic and other proximate causes combine consistently to produce one sequence of reproductive and mortality declines everywhere (allowing, of course, for secondary variations). Instead, an immense heterogeneity of trends within and between societies has been documented, the diversity of which is not consistently explained by the matrix of modernisation variables (see references given in the sub-section on vital statistics, above). In demographers' exploration of alternative approaches, two remarkable parallels to evolutionary biology may be noted.

One is the much greater interest in open population processes, that is, functional links between individuals and between sub-population memberships that are unobserved in standard demographic classifications and closed population units. These include: the impact of hierarchical relations on inequalities in demographic outcomes; inter-generational relationships and variation of generational roles across the life course as they affect reproduction, family formation and longevity; migration and changing cultural identities as adaptive strategies; and network transmission of ideas and practices between sub-populations as they shape varying reproductive choice and health outcomes within and between groups. The second and related development is increasing attention to sub-population variation at levels below, or that cut across, conventional national and provincial administrative population units. Current problems of demographic explanation, in other words, have drawn the field toward the kinds of issues that long ago, in Classical population thinking, gave relationships between sub-populations and their members a determinant role, and which likewise shaped Darwin's account of how demes and species are formed and change.

By way of conclusion, two brief examples drawn from recent evolutionary demography can be used to illustrate how the methodology of local population spaces described above is now being used to address central problems of demographic explanation. As Kaplan and Gurven (2008) reiterate, combined top-down and bottom-up population thinking is necessary. Bottom-up approaches may, for example, relate physiological variables (e.g. mothers' energy reserves as indicated by body mass index (BMI); dietary constraints; local environmental disease risks to infants) to demographic measures (mothers' age at first birth, parity progression, infant mortality) in order to identify proximate causes as they vary health conditions and changing vital rates in different sub-populations. The top-down element is provided by life history theory: reproduction entails trade-offs in which available parental energy and resources for childrearing must be balanced against the increasing demands that a succession of children inevitably makes; natural selection occurs as interactions between physiological constraints and the incidence of births and infant deaths alter this balance in ways that regulate continuing parental investments and the survival of certain children. Note that this approach, rather than treating fertility and mortality separately, focusses on feedback mechanisms between them in specific environments. Modernisation variables act not as external forces that sweep away traditional arrangements, but through this proximate process, and they may be more or less important depending on which aspects of environment-organism-genetic interaction they influence.

Longitudinal research on Tsimane communities in Bolivia provides an example of a "bottom-up" study, addressed to lowland, subsistence farming and foraging communities whose way of life and demography remain substantially traditional (Kaplan et al. 2015). A combined ethnographic methodology incorporates continuing comprehensive census data

collection, reproductive history interviewing and annual medical examination of a wide range of physiological characteristics. This combination provides local population data from several observational techniques which can be compared and analysed as an effectively closed population. Total fertility is at very high pre-transition levels (8.8 births per woman), with modestly lower rates in communities somewhat more exposed to Bolivian towns. Although the latter communities now have greater access to public health facilities, infant mortality levels have risen, even while women's BMI has improved. The authors show how this rather counter-intuitive pattern can be accounted for by linking reproductive histories to women's improved energy circumstances: births have come at earlier ages in marriage, and closer together, both of which are facilitated by higher energy resources, but which normally carry added health risks. Variables that might be expected indicate modern impacts, like education and greater facility in speaking Spanish, appear to have at most minor influence. In the authors' view, this finding shows the operation of natural selection as maximising the production of surviving offspring (i.e. not maximised fertility) in balance with the realities of parental investment.

The study is prospective in the sense that the Tsimane communities are at a pre- or initial stage of demographic transition: the authors, expecting fertility declines to ensue, have established a baseline of current proximate mechanisms and their relationships on which subsequent variations in familial, physiological and community-level factors and their interactions can be assessed. Such baselines have been notably absent in most transition research. Their finding that mothers' age declines at first birth, associated with higher overall levels of fertility, is already indicative of a central mechanism of "pre-decline rise" in the region, and is one of the main lacunae to have been found in transition theory (Dyson and Murphy 1985). Their approach, in considering feedbacks between fertility and mortality via physiological factors, also runs counter to conventional transition and Malthusian arguments that higher fertility is a homeostatic response to higher mortality. While community variables like education and bilingualism are not yet important influences, as components of social learning they are likely to become a potentially major environmental force in social and genetic change (Sears 2015b). The authors underline the importance of ethnography at sub-population levels in evolutionary demography, noting that subsequent research will need to identify the social networks in which health information associated with these variables may spread more widely.

The second example takes up the question of how such social relationships can be integrated into formal modelling of evolutionary change. As noted earlier, one of the problems demography has faced is how to bring variation in social and economic relationships, or "independent" variables, into core demographic analysis. The issue is thus one of preparing new top-down approaches. Lee (2003, 2008), for example, has addressed the role of inter-generational transfers as a mechanism of evolutionary demography, with particular reference to ageing and juvenile mortality. Conventional evolutionary models, following Hamilton (1966), rest on a purely demographic analysis in which, under a stable population growth rate, mortality increases at older ages in inverse relation to expected fertility by age. Put very simply: as older people do not have babies, their contribution to group fitness may appear to be marginal; further, if they have no proximate functions supporting fertility, and are susceptible to the complications of age-specific deleterious mutations as they reach later life, there would seem to be no serious evolutionary advantage to their increased longevity.

As Lee remarks, this formulation leaves the human capacity for long post-reproductive survival unexplained.[16] A considerable body of natural historical, ethnographic and historical evidence has for some time made the conventional view untenable: elders, particularly female relatives, contribute substantial support to raising their grandchildren, and in many cases to others in younger generations that are not direct descendants. As proximate functions of support contributed by elders to the survival and growth of groups are evident in many species, selection for their greater longevity (including differences for the sex contributing most to transfers) is logically indicated. Likewise, the uniform progression of mortality with age in the conventional model, by not taking account of transfers, fails to recognise that early death, e.g. in infancy, incurs much less physiological and support cost than deaths at juvenile ages, by which time much greater investments have been made.

Life history theory, in which a balance between fertility, mortality and investments in children is fundamental to evolution, again provides the elemental logic. Lee's model is addressed to the long period from prehistory in which the human race depended on foraging, so again a kind of baseline is being established. As the objective is to show what difference transfers make to levels of fertility, mortality and natural increase at each age across the life course, a complex set of variables is entailed. Since production varies with a group's relative success in competition for food and resources, the capacity to make transfers depends on population density and size. Production also depends on feedbacks from consumption, since it depends on the growth, size and strength of individual members, which have been shaped by the food and resources available to them. The net transfer that becomes possible at each age can therefore be modelled as estimated production minus consumption (assuming no wastage); this will vary according to the composition of units or groups involved, and Lee's model may be applied to a range, from individuals and mother-offspring sets to larger family groups and cooperative breeding groups. The implications for natural selection then turn on how changes in fertility and mortality, and resulting age structures, interact with intergenerational investment supported by transfers. Lower mortality at the youngest ages increases population growth, and, if coupled with lower mortality at older ages, profits from feedbacks via transfers that also optimise longevity, further stimulating population increase. Greater surviving reproduction thus increases fitness at both the top and the bottom of the age pyramid; transfers become the key to understanding longer post-reproductive longevity characterising more successful and numerically dominant competitive groups. For these groups with greater capacity to invest in children who are then more likely to survive, older adults over time will come increasingly to be selected genetically for greater longevity — this not only helps to ensure continued transfers, but opens up the possibility of reducing fertility (i.e. increasing the quality and quantity of investment per child), thus avoiding the Malthusian trap of high density groups becoming subject to too much competition. As every unit must be in transfer balance (whether successfully, or via loss of

16 Beginning in the 1960s, two distinguished British evolutionary biologists, William Hamilton and Brian Charlesworth, developed models of ageing that relate genetic variation to life history, and which have substantially reshaped understanding of variation in longevity and its relation to fecundity. Further discussion of Hamilton can be found in Ronald Lee's chapter, 'Sociality, Food Sharing and the Evolution of Life Histories', and of Charlesworth in Ken Wachter, 'Genetic Evolutionary Demography', both in this volume.

members to mortality at younger and older ages), the sum of all units, or the total population, will also be in balance.

In its early formulation, Lee's model made a number of abstract assumptions, for example only applying to single-sex transfers of food in stable populations. Later iterations have reduced some of these, and also included more variables, but an account here would extend discussion greatly beyond the scope of this chapter. In each case the model has been developed with evidence from the ethnographic background on foraging populations in mind, and applied to population data on them. Simulations utilising the model enable a 75,000-year prehistory of foraging groups to be constructed, a picture of environment-organism-genetic interaction in the long term. This may, as further developed, serve as a baseline indicating possible ultimate evolutionary parameters within which proximate mechanisms — introduced in the relatively short and recent 2,000-year period of more complex agricultural, urban and manufacturing societies — can be understood.

Concluding Note

In view of the historical development of population thinking traced in this chapter, it is clear that research has moved on from the hunt for ostensible laws of natural selection based, for example, on Malthus's positive check in closed populations, or the eugenics of reproductive selection. The Evolutionary Synthesis, in reasserting Darwin's dual conceptualisation of population thinking, has facilitated a closer relation between formal modelling and local population data, whether in laboratory or field settings, and increasingly in the later twentieth century with reference to proximate processes observed in human groups. Stepping back from this long and complex history, the importance of evolutionary population biology to demography may be summarised broadly on two counts.

One, as we have seen, is to remind demographers of the substantial body of population theory on which social and biological population research *jointly* rest. Darwin, in building his theory of evolution on analogy between observation-based natural history and Smith's account of the division of labour, enabled evolutionary biology to remain truer to the long tradition of open population thinking than has been the case in demography, with its overriding focus on closed methodologies of population statistics. Demography over the nineteenth and twentieth centuries remained primarily the study of population renewal, whereas evolutionary biology has addressed both renewal and structural change by explaining the dynamics that renew population heterogeneity. Yet Darwin, in also insisting on the crucial role of statistical demonstration of the variation and divergence of characteristics, opened the door to applications of actuarial methods, which early-twentieth-century analysts like Lotka, Fisher, Galton and Pearson then began to develop. Recognition of the complementarity of the two concepts of population was one of the main achievements of the Synthesis.

A second wider implication of this history follows from the fact that mid-twentieth-century social and economic demography did not undergo a comparable synthesis. Its approach to theory, notably in attempts to explain demographic transitions, remained focussed primarily on the evidence of closed population methods, often viewed in terms of stylised macro- and micro-levels. This methodology has undoubtedly proven very fruitful in tracking aggregate trends at these levels. The central finding of a vast body of research on demographic transitions has been to demonstrate the immense heterogeneity of fertility and mortality declines in the modern

era, taking place in a vast array of environments — exactly as Darwinian population thinking would lead us to expect. However, in its reliance on closed units, often based on institutional compilations rather than sustained observation of groups in society, and without a primary focus on evolving interdependence and divergence amongst constituent populations, demography has encountered great difficulty in providing a scientific explanation of its central finding. Evolutionary demography, following on from population biology, recognises that heterogeneity requires explanation on several levels, from genetic and cellular processes up to the diverse ways in which social groups are distributed and redistributed in social structures over time.[17] Formal population analysis remains by definition addressed to closed units, but its development is shaped necessarily by increasingly complex bottom-up configurations of observed proximate mechanisms — the "division of labour" within and amongst local populations which Darwin recognised as the locus of environment-organism-genetic evolution.

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17 Exploration of alternative levels of analysis and their implications for explanatory models are a subject of the many contributions to Kreager et al. 2015.

18 Note this chapter has been posted on the Open Science Framework website since 13/06/2019, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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3. A Biologist's Perspective on Human Evolutionary Demography

Bobbi S. Low

Human evolutionary demography has produced striking advances by applying the lens of fitness maximization to demographic data. This approach has strong parallels and links to life history theory, which concerns life patterns (e.g. age at first reproduction, age-specific fertility and mortality) and behavioural ecology, which examines ecological and social influences on behavior. Both those fields focus primarily on non-human species. In addition to clarifying fitness thinking within demography, human evolutionary demography is helping those of us in related fields to deeper understanding of our own disciplines, partly because we know so much in detail about human lifetimes and their diversity. Evolutionary demographers often can bring multiple scales of analysis and multiple kinds of data to bear on research questions, enriching our broader understanding. In the past, those of us who studied non-humans have not typically been able to do this — but seeing the value of such work, in at least some cases, for some species, today we may be able to do better. Finally, there is some potential for this cross-disciplinary approach to have real, and real-world, value in terms of making sensible and realistic policy.

I am a biologist who stumbled into human evolutionary demography. When my son was two months old and I was a single mother, my field work was on digger wasps that hunt fast-flying robber flies. Schlepping my son, a portable crib and gear to the field site was awful (mostly my son screaming, reflecting how he hated this). I had an epiphany: I needed something to work on that I could do on the computer, after he was asleep. This led, in the 1970s, to my first work on humans. Then, six years later, at the birthday party of a colleague's nephew, the grandfather — an evolutionary scholar — said to me "with your interest in resources and reproductive success, you should meet another parent here who has worked with the Swedish Demographic Database." This was invaluable advice: the Database, originally designed so that schoolchildren could trace their lineages, had never had someone bring a set of testable hypotheses to explore, and I found a gold mine! An analyst there, who became a good friend, was amazingly helpful in getting the files organized. I discovered something I had never suspected about data gathered without reference to one's hypotheses: they can't be biased by one's approach to questions — but they also sometimes fail to be useful for one's important questions.

That I should shift into demography is not as odd as it might seem, because my focus has long been on life history theory and behavioural ecology; evolutionary demography (with some language shifts) encompasses both. Life history focuses primarily on non-human lifetime patterns — demographics like age-specific fertility and mortality — which are shaped by the trade-offs all organisms face.

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Behavioural ecology takes analysis to a finer level, focusing on how environmental conditions shape both demographics and behaviour. And *human* evolutionary demography tackles all of these concerns for the species about which we have, arguably, the best and most detailed data.

All three of these approaches examine the costs and benefits of different life history/demographic patterns under varied environmental constraints: age at first reproduction; trade-offs of current versus future reproduction; semelparity (one-time reproduction)/degree of iteroparity (how often reproduction is repeated); clutch or litter size; trade-offs in offspring size versus number; and more. The languages used differ across fields somewhat, as do the emphases, but cross-fertilization across perspectives has been fruitful — and I think has become even more useful today. I attempt nothing like a complete literature review; other chapters will do that admirably.

Here I hope to highlight facets of evolutionary demography that help those of us in related fields to deeper understanding of those fields: the importance of multiple scales of analysis, and of multiple kinds of data; the value of really deep knowledge in a particular species for enriching broader studies, and the value of evolutionary demographic analyses in the wider, applied policy world.

Organisms invest time and energy in growth, maintenance, finding mates, raising offspring. For many expenditures, what is spent on one endeavor cannot be spent on another: energy invested in an offspring, for example, cannot be used to improve one's own condition. Which expenditure is most effective at any moment depends in large part on environmental conditions (Stearns 1992; Roff 1992). It is worth noting that both Stearns and Roff included human data for comparisons when available.

The three approaches — life history theory, behavioural ecology, and evolutionary demography — are strikingly parallel; their evolutionary and ecological bases are deep and clear. As noted by Sear et al. (2016), all three seek *ultimate* explanations for the variation we see in life history variables. They contrast, in that life history comparisons tend to be broad in scope, comparing multiple species; behavioural ecology tends to focus on ecological influences on behaviour, often for one particular species. However, until recently (Borgerhoff Mulder 1991; Cronk 1991a), neither of these considered human patterns. Human evolutionary demography produces rich and detailed data on past and present populations within one species — humans. Evolutionary demography not only uses this perspective, but also commandeers the social science "bottom up" approach in examining variation — looking at proximate triggers or cues for behaviour. The combination is powerful.

Human life histories and demography may show more intraspecific variation than we (think we) see in other species. We have, for example, broad cross-cultural data for more than a thousand societies (many of which are traditional), and we have modern transnational data for about 175 nation states. This complexity and variation within a single species suggests, I think, that evolutionary demography can both enrich and refine life history theory and behavioural ecology.

Although scholars in the various fields were not well connected when pioneer evolutionary demographers began to apply the lens of fitness maximization to demographic issues, these pioneers converged on problems central to life history theory and behavioural ecology — which were then still developing as well. The work accumulated was revealing (see Sear et al. 2016 for an excellent review). Alice Clarke, then a doctoral student, and I were struck by the commonalities, leading us to write a review of papers testing evolutionary hypotheses with demographic data (Clarke and Low 2001). It was rewarding to find real progress.

Sometimes, in those early years, papers read like 'standard' demographic papers. The evolutionary and ecological hypotheses that drove the questions might be hidden — but they were there, and were important in beginning to infuse demography with evolutionary thinking. At the time, I was dubious, but I now think such 'stealthy' approaches were really helpful: do outstanding demography, link results to things evolutionary scholars think are important, but do not "lead with your chin" by aggressive labeling (further, I am hearing from colleagues today that stealth is still useful in getting published and in changing minds). I think the new lens helped shift the thinking of "classical" demographers. I remember showing a "box" in Daly and Wilson's (1983) *Sex, Evolution, and Behavior* to a demography colleague who was methodologically expert. The box took an evolutionary lens to an excellent paper by the colleague; the writing was aimed at people already using an evolutionary perspective. He became agitated, and forcefully shut the book. We said no more about the topic, but in a very few years, as evolutionary demography papers accumulated, he was moved to write about an evolutionary perspective arising from his own data (Knodel et al 1997).

The work Dr. Clarke and I found focused primarily on traditional and historical societies; it covered basic topics in life history, and included work on strategy-environment matching both in the past (historical forces) and in the present (current utility). The authors we reviewed were anthropologists, economists, demographers and biologists — all expanding horizons in demography by examining human demographics through an evolutionary lens — what today might be called part of behavioural ecology. I still have a preference for the term "ecological demography" (Low, Clarke, and Lockridge 1992, Low 1993) rather than "evolutionary demography" because almost all extant work is about how well particular strategies perform under specific ecological and social conditions — that is, behavioural ecology. We seldom have the relevant genetic information to infer evolutionary change over time. In biology, the term "evolution" often concerns changes in gene frequency over time — and even now, few studies on human adaptive responses can meet that criterion.

Many of the topics Dr. Clarke and I found in 2001 tended, unsurprisingly, to be rather straightforward parallels to the sorts of questions raised by life history theory and behavioural ecology papers on non-human species:

- age-specific fertility (Daly and Wilson 1997);
- relationships between resource control and/or status and reproductive success, especially for males (Chagnon 1979; Irons 1979; Hill and Hurtado 1996; Kaplan et al. 1995; Betzig 1986) and family lineages (Hughes 1986; Turke 1989);
- quantity-quality trade-offs in fertility (Becker and Lewis 1974; Mace 1998, 2000a);
- the rarity of twins (Lummaa et al. 1998; Haukioja, Lemmrtyinen, and Pikkola 1989; Gabler and Voland 1994);
- infanticide (Daly and Wilson 1984, Hrdy 1992, Hill and Hurtado 1996) and child abuse and neglect (Daly and Wilson 1984);
- infant and child survivorship as a function of parental socioeconomic status and a child's sex (Mace 1998, 2000a);
- optimal birth spacing (Blurton Jones 1986);
- sex-biased investment (Trivers 1972; Charnov 1982; Cronk 1991b);
- the impacts of migration on lineage success (Clarke and Low 1992).

In each case, the issue of optimization — finding the most reproductively effective strategy, given environmental constraints — was paramount, as it remains today. These papers, and more, represented new and fertile investigations in anthropology and demography. They connected previously separate fields, and had strong ties to work by biologists on other species. We found scattered, less concentrated work on additional topics: sex differences in remarriage; impacts of illegitimacy on survival and reproductive success; and alloparental care.

Another ubiquitous concern in these early papers was that of trade-offs, imposed not only by ecological conditions, but also cultural practices. Even that long ago (2000–2001) there was well-grounded work that, while focusing on important life history topics, integrated these with cultural practices (e.g. optimal fertility and inheritance (Mace 1998, 2000a) and the impacts of marriage system on child mortality (Strassmann 1997)) that can affect the relative advantage of alternate strategies. The issue of trade-offs is as old as Darwin. Like Lawson and Borgerhoff Mulder (2016) and others, I have argued that demographic transitions are really about how much investment in children matters in improving their competitiveness, and that increased per-child investment usually results in fewer children because of the trade-offs. This is simply a re-phrasing of the quantity-quality trade-off raised by Darwin (1871, I: 319):

The only check to a continued augmentation of fertility in each organism seems to be either the expenditure of more power and the greater risks run by the parents that produce more numerous progeny, or the contingency of very numerous eggs and young being produced of smaller size, or that are less vigorous, or subsequently not so well-nurtured.

Of course, evolutionary anthropologists and demographers (e.g. Borgerhoff Mulder 2000) and some economists (e.g. Becker and Lewis 1974; Becker and Tomes 1976) have recognized and highlighted this quantity-quality trade-off; though I have found no other reference as old as Darwin.

The costs and benefits of trade-offs may differ for different kinds of individuals. Suppose there is a trade-off between offspring size and number: a beetle female cannot make as many large eggs at a time as small eggs — but really large beetles can nonetheless make a lot of very large eggs (biologists call this the "phenotypic correlation"). That is, specific individuals with extraordinary resources may not be so constrained as others (e.g. Lessells 1991). Similarly, what you spend on your house, you cannot spend on a car (you have finite resources) — yet really wealthy individuals can afford both a fancy house and a fancy car. In modern societies with high inequality (in wealth, health access and more), this may mean, for example, that wealthy individuals can have many children *and* invest fully in all. There can be circumstances in which familial wealth (or other contributions) reduce the trade-offs (as above: when more resources mean more, still highly-invested, offspring). As Easterlin and colleagues (Easterlin 1978; Easterlin and Crimmins 1985) argued, we may be back to: "more resources leads to higher fertility."

Further, it is clear that such cultural influences as religious and legal rules (pro- or anti-fertility) and individuals' assessment of their status relative to their parents, their cohort and others in their current environment influence fertility decisions. Thus, we will continue to see great variation. The task now is to understand that variation (e.g. Macunovich 1998) — and here, I think evolutionary demographers lead the pack.

Though we attempted a thorough review, Dr. Clarke and I missed some important papers (e.g. Mace 2000b); we even missed analyses of clearly ecologically-driven issues like optimal foraging (e.g. Smith and Winterhalder 1992). We weren't alone; unintentional biases in citations were common, often reflecting difficulties in covering literature, for example, from other countries, or different

communities of scholars (see Sandstrom 2001). This problem has been ameliorated over the years by services like Google Scholar, Academia.edu and Research Gate, so that today, thorough coverage of papers on a topic is easier to achieve, and failure to be complete is more easily discovered.

What Is Exciting Today

Early work in evolutionary demography drew on principles from biological theory, particularly optimal foraging theory, life history theory and behavioural ecology; the concept of (biological) fitness maximization was then largely unknown in classical demography. As a result of the progress and explosive expansion over time of evolutionary-minded papers, we are at an exciting juncture today in many ways.

Human evolutionary demography is in a position to inform and deepen our understanding in several fields, from some presumably 'simple' and unquestioned principles in life history theory to fertility policy. Current evolutionary demography can draw on more kinds of data than earlier work. Often, it can provide analyses at several scales, from transnational analyses to analyses of individual patterns within a single population or sub-population. Models are more sophisticated today, and potentially more useful. It is not my intention to review modern advances: again, other chapters in this volume do that. But I will explain why these advances excite me.

Finding the Right Scale of Analysis: It Depends on the Question

Because scholars are interested in human data for many reasons, and demography is a broad subject, papers' emphases can vary greatly. In non-evolutionary demography, both single-population and large-scale comparisons were common, but in some cases, we would say today that the match between scale of analysis and the questions asked could be improved. An example is work by Birdsall (1980; see also Birdsall and Griffin 1988); these papers were broad comparisons examining fertility across nations and completed fertility within nations by wealth category for four countries. Birdsall's analyses were important, in the service of understanding fertility and poverty in the developing world. Yet as a behavioral ecologist I was struck, and initially confused, by the emphasis on transnational comparisons. The transnational data looked (loosely) as though fertility was higher when resources were more limited — the opposite pattern from that found in other species, in traditional societies and in historical societies.

I eventually realized that (as you will find obvious) these were spurious patterns for the questions that interested me: the reproductive patterns of families in such strikingly different ecologies, as it were, were simply not comparable for evolutionary questions, though they clearly had policy relevance (Birdsall's focus). Similarly, Vining (1986) and Pérusse (1993), who took similar approaches, argued that cultural success and biological fitness were unrelated or negatively related. Stulp and Barrett (2016) have noted that such cross-sectional comparisons were inappropriate for analyzing wealth-fertility patterns.

Birdsall's within-country comparisons by wealth quintile showed patterns that by now will be familiar to many of us: in some nations, wealthier families had more children than less wealthy families, in others, mid-income families were larger. Today we recognize that there is no single pattern; results depend on conditions (more below). In Birdsall's work, there was no real conflict in the empirical data, but the conclusions drawn more or less ignore the within-population results — yet these are important for questions of interest to us.

It is important, then, to recognize that different scales of analysis are appropriate for different questions. How does one decide on the appropriate scale for a question? In comparative analyses from both non-human life history/behavioural ecology and human evolutionary demography, both large-scale and small-scale comparisons can be useful. Early on, there were occasional mismatches that confused at least some of us. As biologists sought to make large generalizations, they typically compared across species. Sometimes evolutionary demography papers do too, with important results (e.g. Galdikas and Wood 1990). Most comparisons of human populations today do a good job of matching scale to question.

Comparisons across human populations, or across individuals within a population, may highlight complexity that is masked by cross-species comparisons. These approaches offer rough parallels to general life history theory and behavioural ecology, which similarly look at variation among individuals that belong to a category within a population or across conspecific populations in different environments. I suggest below that the detailed understanding brought by more localized studies can usefully inform the broader arguments.

The lack of an evolutionary lens in non-evolutionary analyses of demographic transitions led, I think, to a relatively narrow focus. For example, the well-studied historical particulars of the Western European transition led non-evolutionary demographers at first to imagine that industrialization was *the* driving force in lowering fertility; this confusion of correlation with causation led to consternation when developing nations such as Thailand (e.g. Knodel, Havanon, and Sittitrai 1990; Knodel and Wongsith 1991) underwent rapid demographic transitions without industrialization. In Thailand, the important proximate factor turned out to be the benefits of secondary education (which was not free) in getting good stable jobs so one could marry and have children. Parents discussed how many children they could afford to put through secondary school, and fertility fell dramatically, from eight to ten children per couple to roughly two, in about ten years.

This importance of the reproductive utility of particular statuses or resources, and the requisite costs to acquire them, are widely recognized in evolutionary demography. Even early classic evolutionary anthropological or human behavioral ecological studies of traditional societies routinely found that even for ostensibly egalitarian groups lacking any formal currency, a man's skill (e.g. hunting among the Ache: Hill and Hurtado 1996), social/political status and power (e.g. among the Yanomamo: Chagnon 1979) mattered to reproductive success. When physical resources exist (e.g. cattle, sheep or goats among the Kipsigis: Borgerhoff Mulder 1988, 1990; or money from market transactions e.g. among the Turmen: Irons 1979) they are used.

The bottom line is simple: whatever resources, tangible or intangible, can improve reproductive success, they will be so employed. Because these societies (and most traditional societies) are polygynous, it is males who are mostly affected. Even apparent exceptions actually follow the rule. Among the Mukogodo, parents invest more in their daughters than their sons (e.g. in food distribution or trips to the clinic: Cronk 1991b) — is this an exception? No. In this case, the Mukogodo are the poorest and least powerful people in the region they inhabit. Mukogodo men are seen as undesirable by families from other groups in the region; plus, Mukogodo men can rarely manage the bride wealth demanded. But Mukogodo *daughters* can marry into families from higher-status groups, so it pays reproductively to invest more in daughters for Mukogodo families.

Certainly, in traditional societies for which we had data, increased resource control (and/or status) typically led to increased reproductive success, primarily for males (just as for other

species studied). Historical data reinforce this pattern, even in socially monogamous societies. Wrigley and Schofield's (1981) detailed non-evolutionary work found that marriage and birth rates in England historically fluctuated, tracking the economic environment. I similarly found in nineteenth-century Sweden that fertility and marriage rates fluctuated in response to the ecological pressures of shortage and abundance of resources — transitions were local and reversible; I could find no evidence of a monolithic, irreversible transition in Sweden (e.g. Low and Clarke 1983). Nonetheless, even though divorce was unknown, wealthier men had, through remarriage after a wife's death, higher lifetime fertility than poorer men (see summary in Low 2015). Voland (e.g. 1990) similarly found that wealth enhanced fertility for Germen men. The variation we observe actually clarifies the fact that fertility is, in fact, influenced by ecological factors.

Some of my biology colleagues may well recoil from placing humans in this "bin" with other species. We all agree that natural selection operates on humans as well as other animals, but for complex behaviors such as fertility timing, humans can be and are influenced by cultural factors (religion, cultural norms) and other group-level influences (e.g. policies at governmental levels, which shape individual costs and benefits). Surely this invalidates any comparison? I would argue that what we are asking is: are responses, however mediated, ecologically appropriate (e.g. does fertility fall when resources constrict)? The major difference between genetically-dictated responses and "cultural" (etc.) responses seems to be that responses arising from phenotypically plastic conditions (whether "if-then" genes or conscious, culturally-mediated decisions) is that phenotypically plastic responses can react quickly (e.g. Cavalli-Sforza and Feldman 1981). So whether we are asking about genetically or culturally mediated responses, the core question remains valid: do responses make ecological sense? There is one caveat, however: cultural responses that are ecologically inefficient can persist for some time in humans, because humans have such a long generation time. All we can really say is that cultural norms that are costly in terms of dramatically reducing reproductive success will never become and remain the most commonly-observed behaviours, and will tend to be replaced over time — e.g. the Shakers.

So far, what I know reinforces my understanding that, because the real question is about selective appropriateness, we are finding that resilient human responses are, indeed, typically selectively sensible norms. What a pleasure today to see how evolutionary demographers with broad and deep data on post-transition societies (e.g. Stulp and Barrett 2016; Stulp et al. 2016) tease apart the relationships between wealth and fertility (generally positive), finding that considerable variation makes ecological sense.

Again, the scale of focus must be appropriate for the questions asked, and evolutionary demographers are able to integrate data across multiple scales. They continue to demonstrate that work at multiple levels, with well-matched levels of analysis to questions, generates real understanding.

Integrating Multiple Kinds of Data Brings New Insights

A particularly fine development has arisen today because evolutionary demographers are using multiple approaches that complement each other. Empirical field data (analyzed through an evolutionary lens), large secondary data sets and lab work each bring different dilemmas to a researcher, but, I think, each also offers unique insights. Together, they enrich our knowledge at multiple levels. Over the years, I have found that field work (I have worked on non-human species from kangaroos to digger wasps) leaves one at the mercy of field ecological conditions, but can

also lead to new insights simply because you are watching intensely, and ecological conditions can change. Lab work (e.g. calcium metabolism in a number of species) allows a more targeted analysis, from biochemical and physiological aspects to DNA analyses, but can sometimes be tedious, and (especially in shared labs) vulnerable to unforeseen contamination. And secondary analyses of existing large data sets, such as the nineteenth-century Swedish demographic data, can give us windows into worlds we otherwise could not approach. However, because the data were likely gathered for questions other than yours, they can be frustrating to work with.

Evolutionary Demography Can Enrich Biology

I think the multi-faceted findings from evolutionary demography can inform and deepen biological analyses that exist at the broad, multi-species level of comparison. Here is an example. Biologists have understood for a considerable time that life expectancy at birth or hatching (e_0) predicts much about reproduction, and that it is the impacts of *extrinsic* mortality (not especially related to individual behaviour) that matters. The shorter the life expectancy, the (relatively) earlier reproduction (AFB, age at first birth) will begin; this typically means that total fertility rate (TFR), and resulting age-specific fertility, will be higher for populations, or lineages, experiencing short life expectancy at birth.

The classic paper (Harvey and Zammuto 1985; see also Figure 5.10 in Stearns 1992) cleverly transformed data to compare life expectancy at birth and age at first birth for warthogs and rabbits, chipmunks and meadow voles and more; the results were striking. Of necessity, they used existing data, which were (and are) hard to come by. As a result, the comparisons made two crucial assumptions. Firstly, any population will adequately represent the species: a chipmunk is a chipmunk is a chipmunk. Secondly, relationships are at equilibrium and do not change rapidly. These are relatively common assumptions in ecology.

But are these assumptions accurate? I can hear evolutionary demographers chuckling already. The wealth of data on human populations can help to clarify the complexities sometimes overlooked in big generalizations. The basic relationship (above) holds for humans as well as other species: the lower the life expectancy at birth ($\boldsymbol{e}_0$), the earlier reproduction begins, and the higher fertility is likely to be throughout life (Low et al. 2008; Low et al. 2013; see also Daly and Wilson 1997). However, across human populations, the relationship is non-linear, and there is extraordinary variation. Further, it is clear that neither of the assumptions made by Harvey and Zammuto holds for humans (Low 2013, Figures 11.2 and 11.3).

Low et al. (2008, 2013) found that no single population would adequately represent the relationship between $\boldsymbol{e}_0$ and AFB for humans as a species. We could not capture all the variation, because the samples were national averages. Even so, the variation is dramatic: in a sample of 130 nations for which there were data for both $\boldsymbol{e}_0$ and AFB, AFB ranged from 18.2 to 29.6 years, and $\boldsymbol{e}_0$ ranged from 31.3 to 82.2 years.

The wealthiest, longest-lived populations fit the generalization well, and had we only examined them, we could have produced a graph much like that from Harvey and Zammuto. Life expectancy at birth was a good predictor of age at first birth (Low et al. 2008: when life expectancy was >60 years: regression results were β=0.757, R^2=0.58, p<.0001). In part, this reflects the strong influences of cultural norms (more below) on reproductive practices.

In contrast, the greatest variation in AFB was in the poorest countries (called by the United Nations Development Programme "Human Development Index 3": "HDI-3" in the data used).

These societies approximate those termed the "Bottom Billion" by Collier (2007). In these countries, one would likely predict constraints to be greatest, just as appears to be true for other species: we expect life expectancy at birth to be short. Yet AFB could vary strikingly within a narrow range of $\boldsymbol{e}_0$. Rwanda and Chad, for example, had almost identical life expectancy, but AFB was 18.2 in Chad (which was extremely poor and suffered drought), and 22 in Rwanda, which endured genocide and civil war so severe it may have led to state collapse (Low et al. 2013). Here, the sources of mortality are important: early childhood deaths as in Chad affect life expectancy differently from adult deaths (principally HIV as well as genocide in Rwanda: Low et al. 2013, especially Figure 3).

What about the second implicit assumption: that life expectancy is in equilibrium? If life expectancy is not stable over time, the relationship between $\boldsymbol{e}_0$ and AFB may also be in flux, and mismatched. We found (Low et al. 2013, Figures 1, 2) that, indeed, life expectancy varied dramatically from 1955 to 2000 in essentially all countries, though the particular patterns of changes fell into several different groups. There were nine strongly different patterns of changing life expectancies (2xBIC ranged from 22.1–313.4; when this measure is >10, differences are considered very strong, so these patterns are extremely strong: Jones, Nagin, and Roeder (2001)). Here is a clear example of the ability of evolutionary demography to refine older, broader generalizations. And note that this analysis still lumps non-comparable populations together, as if 'nation' represented one population. We still have a lot to learn.

The lesson for biologists here, I think, is that just as picking one population at one point in time is inappropriate to represent "human fertility", it is almost certainly true that not all populations of chimpanzees or chipmunks are identical, although I recognize that the logistic difficulties can be extreme in studying multiple populations of non-human species. And, in fact, we do know about some inter-population variation in a few species, such as chimpanzees and orangutans (e.g. Whiten et al. 1999, Watts 2008, van Schaik et al. 2003). Such fleshing out of the selective inferences from specific inter-population variance can enrich our understanding. Here, evolutionary demographers have much to offer biologists.

In the Wider World

Often the work of life history, evolutionary demography and behavioural ecology can seem remote and academic. I think the opposite is true: only by focusing on the ultimate pressure of fitness maximization — the driving force shaping behavior — as well as fleshing out the proximate details of just *how* environmental pressures shape behavior, is it possible to get beyond our past of simply seeking correlations, to uncover what actually influences behaviour.

Human fertility is related in complex ways to a considerable number of factors such as wealth, income, education, certain kinds of female labor force participation and more. These relationships differ in least-developed and more-developed countries: 'development' typically involves more female education and more paid female participation in the labour force.

Once constrained largely by ecological influences, fertility today may be most strongly influenced by cultural factors: norms and religious beliefs (as in fights over abortion rights), and human oddities that probably count as 'ecological factors' because they contribute to an environment in which arguments succeed or fail (like policies such as tax structures that affect the costs of children, and more). In nations around the world, policies are being made constantly that affect women's lives, both directly (e.g. access to health care, or rules about fertility control) and indirectly (e.g. policies affecting the costs and benefits of women's individual choices). But many, perhaps most,

such policies have unintended consequences, both in their direct and indirect effects. Who better to analyze proposed policy than scholars who not only measure and understand proximate drivers of behaviour (child-care availability, etc.) but who also understand evolved human biases — which can contribute to the failure of the best-intentioned proximate policies? I suggest that evolutionary demographers are ideally positioned to make a positive difference in this arena.

Historical events can leave their footprints, complicating matters. Once (e.g. in the 1960s and 1970s), fears of overpopulation (e.g. Ehrlich 1968) were intense. Governments at various levels, and individuals, responded in various ways, again with unintended consequences. South Korea, in response to concerns about population growth, established policies that today have resulted in arguably the world's lowest total fertility rate, and there is concern about ageing and loss of workers resulting from this policy overshoot.

Fertility rates and total fertility vary around the world today. The UNDP's Human Development Index rankings assess health (life expectancy at birth), education (years) and living standards (e.g. GDP per capita). As we would expect, fertility is strongly patterned with the HDI measures of development. The TFR for the most-developed nations averaged below replacement, but is well above replacement for the least-developed nations. Of course, this does not reflect within-nation variation; it does, however, suggest that governing bodies will have quite different interests in influencing fertility."

The most developed nations comprise much of the HDI rank 1 and they have below-replacement fertility, as well as delayed ages at first birth, and high levels of women's education: the correlates you would expect. The proximate causes of declines vary. In contrast to the Korean experience above, Japan's low fertility appears to have been driven first by individual choices rather than formal policy: after World War II, women appeared to shift to stop childbearing well before menopause (lowering TFR), and later to delaying both marriage and fertility. This resulted in a marked decline in fertility among women in their twenties, and a slight increase in fertility among women in their thirties (Tsuya 2015). Japan has, from the mid-1990s through the 2000s, implemented a number of policies aimed at helping parents of preschool children balance their domestic and work responsibilities, for example increasing child care opportunities and more. But the problems remain: despite pro-natalist policies, Japan's TFR remains below replacement.

Policy has typically addressed proximate influences: e.g. expanding educational and professional opportunities for women. But gendered divisions of labour, a major force in our evolutionary past, persist as an influence: in Japan, even though women are employed, they continue to do roughly five times as many hours of household work per week as do men (Tsuya 2015).

Many low-fertility countries hope to implement pro-natalist policies, but the complexities of fertility and its covariates suggest that more unintended consequences are likely to follow. Getting input from scholars who understand both proximate and ultimate influences on our behavior should help to reduce unintended negative consequences. Further, not only fertility rate, but other important demographics, such as sex ratio, can be affected. Well-meant Indian efforts to empower women, with the further goal of improving treatment of their daughters, backfired: the more educated and wealthy were the families or mothers, the more daughters suffered discrimination (Mahalingham 2007; Das Gupta and Visaria 1996).

Here is another difficulty in making policy: implementation of policies assumes agreement on policy goals — but from the level of individuals and communities to government institutions, agreement is often lacking. Perhaps the United States is an extreme in terms of polarization today, but consider the fights, at all these levels, about funding for Planned Parenthood, which

provides information, outreach and reproductive services for almost five million people a year (https://www.plannedparenthood.org/).

Information, we hope, can help alleviate disagreements in which the difficulty is that facts are lacking. Policies affecting fertility, if successful, will have downstream impacts that benefit different actors. Lee et al (2014) brought a demographic lens to the questions raised by low fertility: who benefits, and who does not? Because there are multiple economic interdependencies that link both public and private intergenerational transfers across age, the impacts of transfers, and the policies that affect them, can differ. Thus, low (and falling) fertility can drive rapid population aging. Lee et al noted that almost half (48%) of the world's population then lived in countries in which the total fertility rate (TFR) is below replacement.

In their analysis of forty countries, Lee et al. found that fertility well above replacement would benefit government budgets; fertility near replacement would raise standards of living (when factoring in the effects of age structure on families); and fertility below replacement would maximize per capita consumption (so long as the cost of providing capital for a growing labor force is included). Age structure and dependency ratios, for example, affect the outcome. Such analyses should be welcome in the policy world: many policymakers recognize that lack of data creates serious obstacles to effective policy (e.g. Takayama and Werding 2011). In sum, fertility and family patterns are influenced both by current costs and benefits, and by deep-seated influences that may derail the best-designed policies that affect current costs and benefits. Since Tinbergen (1963), in biology we call these current utility and phylogenetic or evolutionary influences. Human evolutionary demography is uniquely positioned to make a positive substantive difference in the real world. It sets what we learn in a context both broad and deep: the patterns we are uncovering give a rich context to what might once have been thought of as patterns peculiar to humans.

Human evolutionary demography adds enormous depth of information about life history patterns and ecological influences. It sets human data in the broadest context, creating important connections to evolutionary anthropology, demography, life history theory and behavioural ecology. It lets us examine what influences fertility patterns both broadly and in detail, at multiple levels and in a manner consistent with what we know about other species. If we want to influence fertility, for example, understanding how it is shaped by ecological or evolutionary patterns is crucial. Human evolutionary demography is a true, and important, nexus.

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4. Anthropological and Evolutionary Demography

Kim Hill

Demography was once a subfield of the social sciences dedicated to the statistical study of birth and death rates, and the mathematical description of these vital rates (function fitting). This also included an empirical examination of proximate factors that affect vital rates. Anthropological demography focused mainly on small-scale (non-Western) societies, and employed interpretations drawn from so-called "anthropological theory" (e.g., Howell, 1986; Campbell and Wood, 1998; Kertzer and Fricke, 1997; Bernardi, 2007). Cross-cultural comparisons were a mainstay of the field. In the past thirty years, however, anthropological demography changed significantly to become a theoretically informed study of mortality and fertility, and other age-related biological features. The theory is based on an evolutionary perspective that can unite human demographic studies with those of other primates, mammals and vertebrate species (e.g., Hill, 1993; Kaplan, 1996; Vaupel, 2010, Blurton-Jones, 2016). This transition expanded the field from the study of vital rates to one including research on growth, development, ageing patterns, etc. (physiological, cognitive, emotional mechanisms) that are strongly theoretically tied to mortality and fertility schedules (e.g., Ketterson and Nolan, 1992; Rickleffs and Wikelski, 2002; Kaplan and Gangestad, 2005; Kirkwood and Austad, 2000). These important changes in the field emerged primarily from the injection of life history theory from biology into the social sciences. A fundamental proposition of evolutionary biology is the recognition that fertility and mortality are the two components of individual fitness. Hence, all phenotypic adaptations that act on one or both of these components will evolve via natural selection. From this view, it is clear that the mechanisms of fertility and survival are key biological adaptations and can only be fully understood in the context of evolution.

Mammalian Life History Theory

The area of biology that focuses on mortality and fertility is called life history theory (LHT). LHT is a field that examines phenotypic traits whose expression at one age has implications for fertility and mortality rates at other ages. Temporal tradeoffs are therefore central to LHT. The goal of LHT is not just to describe demographic rates (fit them with mathematical equations), but to explain and predict the shape of mortality and fertility functions as adaptive outcomes of natural selection (Stearns, 1976, 1992; Charnov, 1991, 1993; Charlesworth, 1994). Natural selection produces organisms that effectively convert limited resources into gene copies at the highest possible rate in competitive environments. LHT, therefore, concerns the optimal timing of developmental events, investment in growth, somatic maintenance and reproduction, such that living organisms

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maximize their genetic contribution over time. In short, LHT predicts the fitness maximizing combinations of mortality and fertility investment trajectories that should emerge via natural selection, and the optimal timing of related phenotypic investments *given the unavoidable mortality risks of an environment in combination with the ecological opportunities for nutrient capture*.

Charnov has described the adaptive LH problem as "growth confronting landscapes of death" (Charnov, 2011). Both nutrient capture profiles and mortality risk are considered to have "extrinsic" components, or "constraints" that determine optimal life histories, just as constraints determine optimal phenotypes for all biological features (Parker and Maynard Smith, 1990). Specifically, some risks of mortality and opportunities for nutrient capture cannot normally be changed with reasonable investments (given species' general phenotypic design and their econiche); hence populations of organisms can be expected to adapt to those facts as if they were "extrinsic" determinants of optimal investment patterns.

The general design of each mammalian species means that each has a living and feeding niche to which it is adapted. As they grow, mammals are able to harvest more total energy (advantages of body size and strength), but they become less efficient at biological "throughput" (absorbing, transporting and utilizing that energy for growth or reproduction) of that energy (universal metabolic and growth scaling laws). *Proportional growth* slows with body size (change in weight with time unit is proportional to body weight to the 3/4 power in mammals). This is possibly due to the branching nature of energy transport and distribution through the body (Case, 1978; Kleiber, 1932; West et al., 1997). Since energy for growth is diverted to reproduction during adulthood (see Charnov, 1991, 1993), growth laws imply that the proportional total energy expended on reproduction per unit time also declines with body size in mammals, both across individuals and species. Note, however, that while *proportional* energy throughput declines, absolute energy harvest and throughput increases monotonically with body size, such that larger females can produce more of *the same size offspring* per unit time than can smaller females. Finally, for a given feeding niche, there is often an optimal body size. As organisms approach that size they obtain fewer productive advantages from continued growth. The absolute cessation of growth at reproductive maturity in determinant growers such as mammals defines adulthood. At sexual maturity, growth ceases and reproductive function activates. The regular relationships between weight, growth, energy harvest and potential reproductive output are the reason that body mass, and not height or some other anthropometric measure, is the most important life history variable. Importantly, since growth itself is a function of the ecology of energy capture, ecological variation in environmental quality will change optimal life history trajectories.

Given the distributions and availability of food resources and the way that body size effects energy for reproduction, the mortality landscape of an animal's living environment is the other major determinant of optimal life history. For simplicity, mortality can be divided into two types of hazards: those that can be reduced substantially with reasonable investment (disease, ageing, exposure, etc.), and those that cannot be easily avoided even with reasonable levels of investment (predation, accidents, etc.). The unavoidable hazards in an animal's environment constitute what is termed "*extrinsic mortality*". The two types of mortality hazard are mainly conceptual, because most causes of mortality can be partially avoided with some investment. By appropriate investment, some mortality reduction can be achieved, but a baseline hazard remains that is essentially "unavoidable" (e.g., conspecific violence is probably an unavoidable cause of some death in human societies, yet certain investments can make individuals less susceptible to becoming victims of violent aggression).

While growth in body size increases total energy throughput available for reproduction in adulthood, there are also other potential gains that come from a longer juvenile development period, body growth and delayed reproduction. These gains come from increased "embodied capital" (Kaplan et al., 2000). Some of the most common gains from delaying reproduction include greater safety from predators through increased body size, time to grow and program larger brains, building effective social alliances and the advantages that can be gained through learning and experience prior to adulthood. This then sets up the most basic life history trade-off: reproducing earlier or reproducing later.

How long should a mammal grow before diverting energy to reproduction (age "α" in life history terminology)? Since there are gains from extending the development and growth period, but there is also some probability of death with each interval that is pre-reproductive, natural selection should favor an "optimal juvenile period" that maximizes gene contribution. In general, when mortality is high, or growth to an optimal adult size is rapid, earlier reproduction is favored. If lifetime fitness (w) can be simplified as the product of survival to age α ($l\alpha$), and the reproductive value at alpha ($V\alpha$), the optimal age at first reproduction is precisely when proportional gains in $V\alpha$ from growing and developing one more time interval are precisely matched by the proportional decrease in probability of survival to the age of first reproduction by waiting one more time interval.

$$w = l\alpha\, V\alpha \qquad \text{(eqn. 1)}$$

$$w(\max), \text{ when } \quad \log dV\alpha = -\log dl\alpha \qquad \text{(eqn. 2)}$$

Since the right-hand term in eqn. 2 is simply the instantaneous mortality rate, this means that the end of the juvenile growth and development period should take place when the proportional increases in body size and other multiplicative components of reproductive value are exactly matched by the yearly mortality rate (proportional loss in probability of survival to age of first reproduction). Higher mortality around the age of sexual maturity will favor earlier maturity.

While the primary life history traits are yearly survival and reproductive rates, LHT can best be thought of in economic terms with LH investments allocated to "embodied capital" rather than material capital (see Kaplan et al., 2000). The LH that allocates energy in a way that results in greatest inclusive fitness is the one that becomes prevalent over time. Hence, LHT is a biological investment theory analogous to optimizing investment strategies in micro-economics. The fitness-maximizing problem for living organisms that can invest temporally in different life functions is analogous to the problem facing a hypothetical financial investor, endowed with an initial factory that extracts resources from the environment (e.g., mining, logging, fishing, etc.), and who must strategize to maximize total productive income over time, derived from that starting endowment (see Figure 1). Such a factory owner could invest all resources in immediate short-term profit (harvest and sell as much as possible now), or instead invest in growth of the facility and replication of other factories at the expense of maximum short-term productive gain. Most importantly, our hypothetical investor's time and resources are always finite and divisible such that investment in one facility or function directly reduces the amount that can be invested in alternative operations. Hence, economic investment theory and LHT are both about the study of optimal trade-offs in investment patterns to maximize productive gain over time.

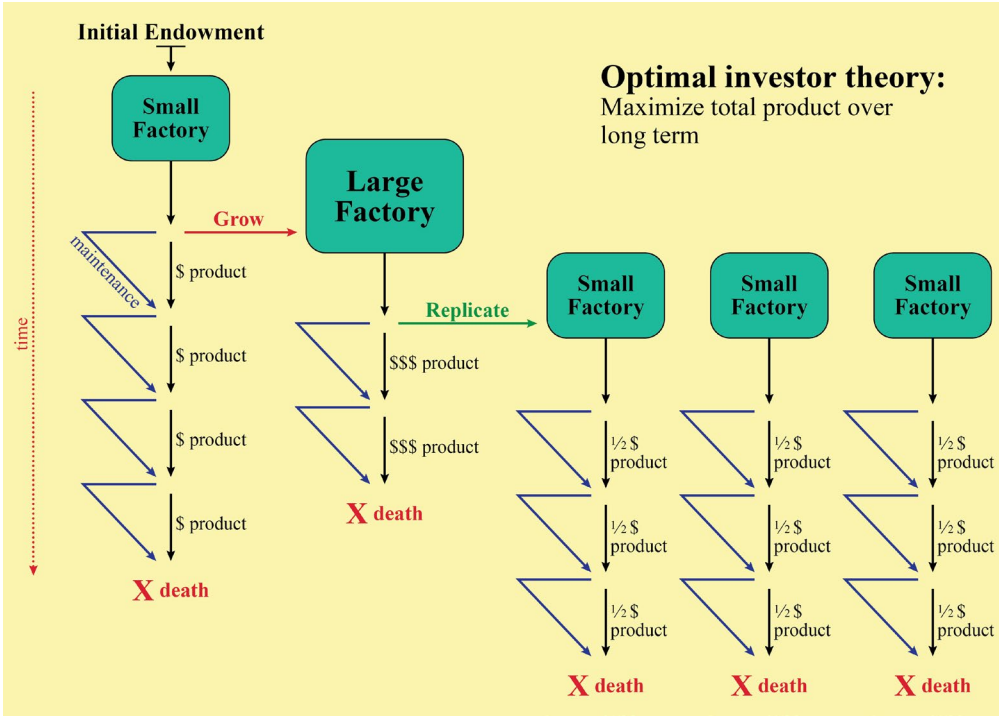

Figure 1: Optimal investor theory suggests that the initial founder of a small factory can either invest in growth, in factory maintenance, or take profits (product) after each production interval. Large factory owners can also convert some production into multiple small factories in order to ameliorate the risk of total loss if the large factory is destroyed by unavoidable circumstances.

Continuing the economic analogy with LHT, our hypothetical factory owner can either extract maximum profits in the short run, or re-invest some resources in expanding the size of the original facility for longer-term profits. Sometimes, he or she will do better to delay taking profits now and instead expand his or her operation, if a larger factory size will substantially increase mean productive harvest per unit time. In our hypothetical scenario, imagine that there is also some finite chance that any factory might be destroyed suddenly or shut down by an unavoidable or unpredictable natural disaster. That threat means that a wise investor should not keep reinvesting into a single expanding factory indefinitely. To do so might risk losing the whole large factory before anticipated long-term profits could be extracted. Instead, it may be wiser to establish several smaller, dispersed factories that will all continue to produce income, even if one of the factories were destroyed. Likewise, an old facility might ultimately deteriorate so much over time that upgrading all the defects would cost more than simply building a new factory from scratch (readers who have purchased a new car rather than continue to pay higher and higher mechanical service costs to repair a failing older vehicle will recognize this dilemma).

Continuing this analogy, our hypothetical investor might discover that the marginal increases in productive harvest with increasing factory size diminish progressively as the factory grows larger (perhaps due to logistical problems of supplying and transporting goods within large factories). This means that higher returns on investment (% gain per dollar of capital investment) are expected if the investor builds several new small factories, growing them to medium size,

rather than continuing to expand the size of the original factory indefinitely. In our example, the establishment of new facilities is analogous to biological reproduction, if we stipulate that new factories are always built with a partner who splits the profits 50:50 (analogous to the genetic relationship between parents and offspring). Given the curve of factory productivity with size, deterioration with age and the chances of being destroyed by a natural disaster, the investor must calculate how large to grow his or her factory before investing profits in building a string of smaller factories (and then expanding them in turn). The ultimate goal is to maximize the net worth and total productive harvest capacity of all factories over time. The optimal investment trajectory should maximize total income over time indefinitely.

To finish our analogy between economics and LHT, our hypothetical investor must determine how much to reinvest on repairing and maintaining each factory that he or she builds. The optimal solution must take into account that maintenance costs reduce immediate factory output but allow existing factories to continue producing for longer periods of time. Of course, too much maintenance would be foolhardy if a disaster is likely to destroy any factory in a statistically known time span, but too little maintenance is wasteful of the investment to build the facility in the first place.

The investor analogy illustrates that LHT is an investment theory, just as other biological theories are similar to already developed microeconomic theories (for example Optimal Foraging Theory and models in microeconomics). The investment perspective of LHT allows us to organize trade-offs into major categories that are experienced over time. For humans and all mammals characterized by determinant growth (where captured energy devoted to growth during a juvenile phase is diverted to reproduction in the adult phase), life history trade-offs can be divided into three major categories: (1) growth vs. somatic maintenance; (2) growth vs. reproduction; and (3) reproduction vs. somatic maintenance. The tradeoff perspective also implies that adults face a trade-off within their reproductive budget: using available resources to produce more offspring per reproductive bout, or to produce fewer offspring of higher reproductive value (the quantity vs. quality trade-off) over time.

The entire suite of LH trade-offs can best be conceptualized as a single basic fitness trade-off between present and future reproduction (Bell and Koufopanou, 1986; Harshman and Zera, 2007). This trade-off implies that higher survival or higher fertility (for self or offspring) can only be achieved at the expense of the other (Gadgil and Bossert, 1970). Higher vital rates at one point in time come at the expense of lower rates at another point in time; or increased fitness of ego comes at the expense of decreased fitness of offspring and other close kin. Specific LH tradeoffs can often be detected empirically with careful research. Some nice examples are recent research with wild chimpanzees showing that maternal fertility and offspring growth trade-off against each other (Emery Thompson, 2016); field evidence from traditional human societies showing that fertility often trades off against offspring survival (Strassman & Gillespie, 2002); and studies showing that childhood growth trades off against activity (Urlacher and Kramer, 2018) or investment in disease resistance (immune function) (Urlacher et al., 2018). Finally, studies of ageing in a large number of living organisms strongly suggest that longer lifespan and fitness benefits earlier in life directly trade off against each other as well (Austad and Hoffman, 2018). The laws of conservation of matter and energy imply that investment trade-offs should be ubiquitous in living organisms.

Within each major life history trade-off category there are many sub-trade-offs as well (e.g. invest in immune function vs. invest in DNA repair; grow in mass vs. grow in height; invest more in helping current offspring A vs. current offspring B, etc.). Early life history tradeoffs in

humans include: evolutionary decisions such as how long to grow in utero, when to shift from lactational dependence to other foods (decisions made by mothers about optimal gestation time and age of weaning), how much to invest in somatic maintenance and repair during childhood, how long to learn before using knowledge to produce resources or engage in social competition with adults and when to stop growing and start reproducing. Later trade-offs in human adulthood include: how frequently to produce an offspring, how long and how much to invest in each offspring based on age, sex and other qualities, how much energy to dedicate to avoiding illness and injury, when to stop reproducing and focus on helping close kin, how much to invest in somatic repair (anti-senescence) vs. assisting descendant kin. This short list can be expanded into an even larger set of specific trade-off decisions between virtually all energetic investments in survival vs. reproduction, for individuals and their close kin.

Fast and Slow Life Histories

Organisms sometimes experience high "extrinsic" mortality due to causes that cannot be fully avoided (e.g., accidents, predation, extreme variation in food availability or weather) even with reasonable investment. This favours speeding up the timing of events in the life history of the organism in order to complete more reproduction before the opportunity is lost forever through death. Species in such circumstances are said to have a "fast life history", because they evolve to grow fast, reproduce early and expend greater reproductive effort in each adult time period. The initial difference between high and low extrinsic mortality risk is further compounded due to differential investment in survival. High extrinsic mortality favours less investment in somatic maintenance and repair (less DNA repair, anti-oxidant activity, cell repair, etc.), which results in earlier degenerative death and amplifies the extrinsic differences between species with high and low mortality risk. Because all organisms face natural risks that cannot be avoided by reasonable investment, the overall pace of a life history must be adapted to the chances that reproductive capacity will not be fully realized before the organism is destroyed.

Since optimal investment in somatic maintenance (lifespan), age at sexual maturity and rate of reproductive effort are all strongly affected by extrinsic mortality, we can talk about "fast life histories" as those characterized by short lifespans (early onset of senescence), early age at sexual maturity and high reproductive effort per unit time in adulthood (Promislow and Harvey, 1990). It is well established that a correlation between these LH traits is found across mammal species, hence the "fast-vs-slow" model outlined above is strongly supported empirically (see Purvis and Harvey, 1995; M. Oli, 2004). It is also generally true that smaller animals have faster life histories than larger animals, because predation risk is generally higher for smaller animals (also growing large takes time, so large body size is already an indicator of a longer juvenile period and hence slower LH). However, it is important to note that high or low reproductive effort per unit time does *not* always mean higher or lower observed fertility rate. The number of offspring produced per unit time (fertility rate) is determined by a combination of how much investment is put into each individual offspring (the "quality-quantity trade-off" in LH theory), and how much energy is invested in reproduction during each time period. The optimal level of investment per offspring, however, is determined by the marginal impact of parental investment (change in offspring reproductive value with additional investment) not by the chances of the adult reproductive dying each year. Since it is possible for parental investment to be efficient (large gains in reproductive value with increased parental investment) or inefficient, the observed fertility rate is determined

by the ratio: [Total Reproductive Effort/Investment per Offspring]. It is quite possible for an organism to evolve a long lifespan and a very *slow* life history (due to low extrinsic mortality) but have a high annual fertility rate (because low investment per offspring is favoured). Examples are found among some large reptiles (e.g., alligators, Lance, 2003) and fish (e.g. ocean sunfish, Pope et al., 2010), which grow large and slowly, have long lifespans, but produce hundreds to thousands of small offspring each reproductive bout (this is also common in large trees). Only when there is low adult extrinsic mortality, and the effect of increased parental investment on offspring reproductive value is large, should we find slower life histories also being characterized by lower fertility rates.

Finally, fast-slow LH adaptations are expected to vary predictably with mortality landscape only when "all else is equal". Sometimes other factors determine age at maturity, lifespan, or reproductive effort. For example, in mammals (and humans) males universally show higher extrinsic mortality than females at every age, yet they also grow longer and achieve sexual maturity and first reproduction at later ages than females. This is because the gains in reproductive value from waiting an extra year are much steeper for adolescent males than females (the effect of greater body size or social "experience" in intra-sexual competition). Hence, the difference between males and females in the onset of reproduction is the opposite of what a simple "fast-slow" view of life histories might predict.

Likewise, there are no simple fast-slow life history predictions related to changes in long-term or short-term resource abundance. When resource availability increases, mammals do not respond with slower life histories (cf. Baumard 2015, 2018). Instead, they grow faster, reach sexual maturity at younger ages but with larger body size, and show higher fertility rates (these are evolved reaction norms). Lifespan generally changes very little, and, if it does, it changes in the direction of an increase. Hence the reaction to resource abundance is a mix of faster (early maturity, higher fertility) and slower (longer lifespan) LH traits.

Finally, how evolved life histories react to "harsh" and "risky" conditions depends on what we mean by "harsh" and "risky". It is not possible to generalize that harsh environments will result in fast life histories (cf. Brumbach et al., 2009). High mortality landscapes favour the kind of fast life history described above. However, when "harsh" is used to refer to resource shortage or variation, it is often the case that age at maturity will be delayed, because body size remains small and greater proportional gains can be achieved through further growth. This is most likely the reason why human populations that experience food shortage show delayed onset of menarche worldwide (Thomas et.al., 2001). In that scenario, "harsh" conditions lead to components of a "slower" life history (delayed sexual maturity). With food shortage we also get lower fertility rates, because the total energy budget for reproduction is decreased. Once again, some would interpret this as indicative of a slower life history (lower fertility). Finally, if a "harsh" environment refers to a "variable" or "risky" environment, the adaptive LH response will be determined by exactly what kind of variability or risk is experienced (high risk of food shortage may lead to delayed maturity and lowered fertility, but high risk of injury or death may lead to a faster LH), and whether the risk can be ameliorated by more somatic investment in self or offspring. If an organism can survive temporal variation by investing more in energy storage, for example, then a variable environment might favor slow growth and reproduction, and a longer lifespan. On the other hand, if variation is frequently lethal, despite strategies to minimize the impact of fluctuation, then populations may evolve very fast life histories, even including semelparous reproduction (a single reproductive episode followed by death) if there is a poor chance of surviving until the next available breeding

season. The key issue is whether variation is likely to be lethal, not the environmental fluctuation *per se*. "Risk" can favour either *faster or slower* life histories depending on the details of the risk.

By mammalian standards, human beings have a very slow life history due to exceptionally low mortality risk across much of the lifespan compared even with other large slow-growing mammals (Harvey and Zammuto, 1985). This is due to a series of cultural and behavioural traits that emerged during hominin evolution, such as the use of fire, projectile weapons against predators and food transfers during illness and injury that lead to exceptionally high survival (Kaplan et al., 2000; Hill and Hurtado, 2009). As a result, human children grow slowly, adolescents reach sexual maturity at a late age compared with other mammals, investment in anti-ageing mechanisms is outstanding (e.g., Hart and Setlow, 1974; MacRae et al., 2015) and onset of senescence takes place at a later age than other great apes (Emery Thompson and Sabbi, this volume). On the other hand, alloparental provisioning allows for exceptionally high female reproductive output in early adulthood despite the slow human life history (somewhat like queen ants, who produce large numbers of offspring but have very long lifespans). Note again that slow life history does not always imply low fertility rate.

The Derived Human Life History

People living in traditional societies (and probably ancestral Homo sapiens) exhibit a series of life history traits, reflected in demographic parameters, that are unique and different from those of our closest phylogenetic relatives (chimpanzees and bonobos). These differences are especially notable because mammalian life history traits tend to be strongly correlated with body size (Western, 1979; Promislow & Harvey, 1990; Charnov, 1993), and many chimpanzee populations are very similar in body size to many tropical hunter-gatherers (Walker et al., 2006; Emery Thompson and Sabbi, this volume). A quick comparison of human and ape life histories suggests four human characteristics: (1) a longer juvenile period; (2) a longer adult lifespan; (3) high early fertility that ends long before the lifespan; and (4) assisted reproduction by post-reproductive adults (Kaplan, 1997). Here I present a detailed comparison of human and ape life histories that suggests many other interesting differences as well. In my view, the derived human life history includes the following:

1) Human beings are born at greater birthweight and after longer gestation despite a smaller maternal pelvic opening (Emery Thompson and Ellison, 2018). This is favorable to human infant survival, but dangerous to mothers, and is possible only because of assistance for human mothers during childbirth. A side effect of this trait is reasonably common death in childbirth among human beings but not apes (no deaths in childbirth have ever been reported among chimpanzees, Emery Thompson, personal communication).
2) Earlier age at weaning for humans due to allomaternal provisioning, extensive food processing and low-fibre, high-protein lipid nutrient harvest by adults.
3) Infant motor skills are more slowly developed in human beings because intense allomaternal caretaking allows for extremely altricial offspring to thrive. Early energy allocation mainly goes to brain growth and learning rather than physical activity and motor skills (Kuzawa et al., 2014).
4) A long post-weaning period of juvenile food dependence among human beings (typically >15 years before food independence- Kaplan et al 2000).

5) Lower proportional body growth by human beings after weaning and until adolescence. Primates have low growth constants (this means little yearly weight gain each year relative to initial body size) among mammals. But human beings have exceptionally low growth constants even for primates (Walker et al., 2006). Childhood energy is used for other functions: not growth (e.g., neural development, cognitive function, immunological competence, etc.). Since juveniles are provisioned by adults, slow growth is not due to their own food production capabilities. There is good evidence that slow childhood growth is partially due to the metabolic costs of brain growth and function (Kuzawa et al., 2014). However, the adolescent growth spurt (and sex differences in its timing), despite no corresponding age-specific increase in energy harvest (Walker et al., 2006), demonstrates that children probably *could* grow faster, and provisioners *could* subsidize faster growth if it were fitness maximizing. Human children might have evolved slow body growth to save energy during juvenile years while the brain is being programmed (some birds show programmed weight loss between reproductive seasons to save energy not needed for immediate reproduction: Norberg, 1981).

6) Humans require much more brain growth after birth than do other primates. This is a required side effect of a large brain, but possibly allows more post-natal programming of the central nervous system as part of the process.

7) Human beings experience a long and intense juvenile learning period that may determine (rather than body growth) the optimal age of reproductive maturity. Since the marked adolescent growth spurt subsidized by others suggests that juvenile growth could be higher at an earlier age, perhaps human children have evolved delayed sexual maturity until proportional gains in Vx from learning (rather than body size) are matched by the proportional losses due to mortality.

8) Human age at sexual maturity and first reproduction are only slightly later than chimpanzees, with some population means almost overlapping (Hiwi menarche at 12.6 years, Pume first birth at 15.5 years vs. chimpanzee female sexual maturity at ~10 years, and first birth >14 years for some populations of wild Chimpanzees: Walker et al., 2006; Kramer et al., 2009 vs. Emery Thompson and Sabbi, this volume) because primaparous human mothers receive high levels of help (advice, caretaking, etc.) and provisioning. Age at first reproduction in humans is determined by when a female can reproduce *with extensive help from others*, something not possible for other primates.

9) Much higher early adult fertility by human females (Kaplan et al., 2000; Emery Thompson and Ellison, 2018) that is highly subsidized through provisioning of both reproductive aged females and their offspring. Mean inter-birth intervals in hunter-gatherers are around 3.3 years, vs. 5–6 years for wild chimpanzees (Marlowe, 2005; Emery Thompson and Sabbi, this volume).

10) Declining fecundability of human females by the mid-twenties, despite that fact that, at that age, females have an additional life expectancy of nearly forty years (fertility begins to decline when only ⅕–⅛ of the mean adult lifespan is over). Chimpanzee

females maintain high fertility into their forties if they are healthy (Emery Thompson et al., 2007).

11) Cessation of ovulatory cycles in human females (menopause) by their mid-forties, and a post reproductive period constituting a large fraction of adult life (Levitis et al., 2013).

12) Dichotomy of human male reproductive trajectories with the end of reproduction in the early fifties for most males who remain monogamously pair-bonded to post-menopausal females (e.g., Hill and Hurtado, 1996, figure 9.9; Kaplan et al., 2010), but significant fertility for a smaller fraction of males from their fifties to their seventies. While few chimpanzee males produce offspring after their mid-thirties and no chimpanzee male has been observed to father an offspring after age forty-three (Emery Thompson and Sabbi, this volume) human males in traditional societies often reproduce after age fifty because of late adult income peak (Kaplan et al., 2000; Koster et al., 2018), and the ability to accumulate resources and political alliances over the lifespan. Gurven (personal communication) has found a wide range of variation in the fraction of expected male fertility achieved after age fifty in small scale societies, ranging from only 1.8 % in the Piro to 3.6% in the !Kung and Tsimane, to 14.3% in Forest Ache, 14.8% in Yanamamo, and then 31.4% in Gambia. Clearly, there are socioecological conditions that allow human male reproduction far later than is ever achieved in apes (even when lifespan is considered).

13) Onset of significant physical and cognitive senescence ten to fifteen years after reproductive cessation for most human males and all females (Salthouse, 2009; Chan et al., 2014).

14) Steep physical deterioration and senescence in humans in conjunction with dependence on kin provisioning and care by the early seventies.

15) Rapid mental and physical senescence and high yearly probability of death (>20% per year) after age seventy (e.g., Hill and Hurtado, 1996).

The derived human life history emerged over the past 5 million years although there are indications that the Hominini tribe (chimpanzee-bonobo-human) may already have some traits in common that are more humanlike than the other Hominids (orangutan, gorilla) — smaller size, less sexual dimorphism, greater relative brain size (Emery Thompson and Sabbi, this volume). In general brain growth and early tooth eruption patterns of early hominins suggest that the derived human life history began to change significantly well after origins of genus Homo (Schwartz, 2012; Rosas et al., 2017), but hominin tooth eruption patterns are complicated to interpret because of early weaning and cultural food processing for human infants (Dean & Cole, 2013).

This derived human life history evolved due to a series of constraints and conditions that sometimes resulted in contradicting adaptive challenges, and ended with a spectacular cooperative breeding ape. Key among these was:

16) increased early hominin mortality due to a terrestrial activity and sleeping niche with greater predator exposure;

17) lowered later hominin mortality due to weapons, fire and frequent provisioning of sick and injured individuals;

18) increased later hominin mortality due to conspecific violence including high rates of infanticide/juvenile homicide because of provisioner conflicts with juvenile recipients, and higher levels of adult homicide from quick-kill weaponry;

19) numerous bursts of rapid hominin population growth due to worldwide expansion and colonization of "empty niches" with initial high food abundance;

20) lowered seasonal variance in food supply in hominins because of opportunistic ominvory, food transfers to buffer shortfalls and unique inter-group visiting allowing resource access to distant regions;

21) extensive juvenile food dependence among later hominins due to the skill- (and learning-) intensive extractive/predatory feeding niche;

22) an overall greater energy consumption/expenditure budget per gram of body weight than apes, presumably to pay for large brains and greater somatic maintenance and repair to facilitate longer lifespans (Pontzer, 2016);

23) multiple overlapping dependent juveniles in hominins and obligate alloparental provisioning (Hill and Hurtado, 2009), as dependent juveniles accumulate.

Finally, it is important to note that the human life history has extensively modified human social structure from that of other great apes, and has been uniquely influenced by cultural norms and social learning. Life history traits related to pair bonding and paternal investment along with extensive juvenile dependency resulted in a social structure that includes bisexual dispersal and/or philopatry, bilocal coresidence, extensive peaceful visiting across social groups in order to provide or receive kin assistance and unique cooperative relations with affinal kin not seen in any other species of life on earth (Chapais, 2009, 2011; Hill et al., 2011). As hominins became more extensively dependent on cultural adaptations, and generalized their social learning proclivities from food procurement and tool-making techniques to other aspects of life, they began to socially acquire mortality-reducing (or increasing) behaviours and fertility-modifying patterns, including social norms regulating traits like age at weaning, age of first mating and pair bonding, typical parental investment patterns and control over offspring's reproductive behaviour. The population trends in some LH traits in Homo sapiens is based partially on imitation and social norms rather than independent individual "decisions" about optimal life history. Fitness-maximizing life histories are constrained by cultural norms, and non-adaptive life histories can become prevalent via social learning (e.g. the demographic transition in worldwide fertility). This means that human life histories are exceptionally influenced by cultural transmission and social learning.

Three Major Areas of Research

Life history research in humans is particularly useful and scientifically significant (with broad implications) when it integrates age-specific mortality, fertility and developmental patterns with other key phenotypic adaptations and behaviours that make humans an exceptionally successful mammal. The summary of the derived human life history given above logically leads to a recognition of three major research topics. The first of these is about adaptive origins: how can we explain the evolution of the derived human life history and its special features that ultimately make humans a spectacularly successful life form on earth? The second of these is about population differences across time and space: how can we explain observed variation in

life history traits across different socio-ecological conditions, and to what extent is the variation due to local genetic evolution, adaptive phenotypic plasticity or a mismatch between evolved LH mechanisms and current environments experienced by modern humans? We particularly want to know the underlying physiological mechanisms and ontogeny of the key life history traits (e.g. Flatt and Heyland, 2011; Ellison, 2016), as well as the range of variation regularly produced via evolved reaction norms and phenotypic plasticity that evolved during hominin history. This will require a far more sophisticated understanding of the physiological mechanisms responsible for the expression of life history traits, and the ability to conditionally adjust life history over a single lifespan. The study of mechanisms should be fully integrated with theoretical models of optimal life history in order to provide a complete understanding of evolution. Finally, we need to understand the evolutionary process of genetic and phenotypic frequency change. How are optimal life histories selected over time when current reproductive value determines optimal future mortality and fertility and vice versa? Some researchers have suggested that this optimality problem requires dynamic programming (Mangel and Satterthwaite, 2016).

Ten Interesting Issues in Evolutionary Demography

Below I present a list of ten interesting, important and unsolved issues in anthropological demography. This list is not meant to be exhaustive; it simply represents my own interests and observations during the past twenty years of life history research (two of these were also discussed as "human life history puzzles" in Mace, 2000). My identification of the questions below is meant to stimulate future research, and occasionally provide hypotheses, not to provide definitive answers to any of the questions listed.

1) The Hunter-Gatherer Demographic Paradox

Howell's (1979) monograph *Demography of the Dobe !Kung* was a highly influential early publication in anthropological demography. Since then, almost a dozen detailed demographic studies of hunter-gatherer populations have provided quantitative estimates of fertility and mortality rates under socio-ecological conditions that are presumed similar to those in which our human ancestors existed for hundreds of thousands of years (see Hewlett, 1991; Pennington, 2001; Marlowe, 2005; Gurven & Kaplan, 2007; Ramirez Rozzi, 2018). Almost all these studies, however, show substantial positive population growth rates, leading us to wonder whether we really know what the ancestral human life history looked like. Put bluntly, until we can discover empirically a real ethnographic life history that results in zero population growth and consists of reasonable (not pathological) fertility and mortality levels adapted to commonly experienced ecological constraints, we may not fully understand how the human life history diverged from other apes.

Modern hunter-gatherer demographic parameters that result in significantly positive population growth cannot directly reflect the human life history trait values through most of ancestral history. Even with progressive worldwide colonization, our species must have shown very close to zero population growth for almost all of the past three-hundred-thousand years. Malthus would be shocked at the measured population growth rates for most modern hunter-gatherers. Life tables from nearly a dozen hunter-gatherer populations, and median life history parameters from many more, all imply population growth of more than one half percent per year (ibid., see Table 1). Median hunter-gatherer values (Marlowe, 2005) of 55% juvenile survival to adulthood, Completed Family Size of 7.1 and presumed 1.5% adult mortality rate lead to population growth

rates of > 2% per year. Modest tweaking of measured demographic parameters within the range observed in careful ethnographic studies still does not achieve zero growth. There are only a few ethnographic exceptions. For example, the Onge of the Andaman Islands were reported to have high mortality, high rates of childlessness and a female age at first birth of twenty-eight years, far below zero growth, but no supporting data are provided to back that claim (Cipriani, 1961).

A population growth rate of only one tenth of one percent per year means that a founding population of ten individuals will grow to over 700 billion people in only 25,000 years! Clearly, the life tables we have observed in modern hunter-gatherer groups cannot represent most of the history of Homo sapiens. This problem was first overtly discussed by Hill and Hurtado (1996: Chapter 14), but more completely explored by Boone (2002), Gurven and Kaplan (2007), and recently by Blurton-Jones (2016: Chapter 11). All authors carried out subsequent simulations to see how low the fertility and survival rates would have to be in order to achieve zero population growth. Assuming that adult mortality rates, prior to senescence, in ancestral human populations were usually about 1.5% per year (74% of women who reach sexual maturity survive to the end of a twenty-year reproductive career) and a 105 sex ratio, we can examine what Completed Family Size (CFS) and juvenile survivorship rate is required to get zero growth. Probing low fertility options, the simple answer is that a CFS of about ~4.5 live births with 50% juvenile mortality will lead to zero growth. But a CFS of 4.5 with a female reproductive span of twenty years (twenty to forty years old from first to last birth) implies a 5.7 year inter-birth interval. This is much longer than the IBI ever measured in any traditional human population and would require physiological birth-spacing mechanisms that probably do not exist in humans (lactational anovulation combined with nutritional stress leading to a birth interval almost twice as long as that typically observed in extant hunter-gatherers (Marlowe, 2005)).

Table 1. Median forager from Marlowe, 2005. Data on each group from Hewlett, 1991; Pennington, 2001; Gurven & Kaplan, 2007; Ramirez Rozzi, 2018.

Group	Juv surv	sex ratio	age fbirth	age lbirth	CFS	Adult mort	females per gen	gen time	increase per year
Med. Forager	0.55	0.49	19.25	39	7.1	0.015	1.90	39	2.32%
Efe	0.78	0.49	19	39	2.7	0.015	1.03	39	0.07%
Hiwi	0.51	0.49	20.5	37.8	5.1	0.015	1.28	37.8	0.73%
Kung	0.6	0.49	19	37	4.7	0.015	1.38	37	1.02%
Agta	0.42	0.49	19.5	41	7.6	0.02	1.56	41	1.36%
Hadza	0.56	0.49	19	39	6.1	0.015	1.67	39	1.71%
Ache	0.61	0.49	19.5	42.1	8.2	0.01	2.43	42.1	3.39%
Baka	0.66	0.49	18	39	7.3	0.015	2.35	39	3.46%

Hunter-gatherer children are typically weaned by age 2.5, and even with later weaning natural suckling rarely results in anovulation after about age 2.5 (because human children naturally begin to eat pre-processed adult foods by that age). Normally, nourished forager women will conceive within half a dozen cycles of ovulatory resumption (Bentley, 1985). Hence a mean IBI of >5 years is probably not possible in human societies unless they are undergoing catastrophic starvation or stress. Of course, we could allow the population mean CFS to be low due to high levels of primary and secondary sterility, but only populations with extremely high STD

infection rates ever show such a pattern in modern ethnographic studies. Alternatively, we can assume more realistic fertility (mean reproductive span nineteen to forty-one years old, mean IBI = 4.5 years, mean TFR = 6.1) and estimate levels of juvenile mortality required to produce zero growth. This modification is more in line with Charnov's (1986) observation that juvenile mortality is the life history variable that shows the greatest change when ecological conditions become good or poor. Based on a CFS of 6.1, we estimate that only 34% of children born could survive to the age of first reproduction if the population is stationary. This is again doubtful, because even the highest-mortality hunter-gatherer populations documented show much higher juvenile survival (Marlowe, 2005) than our simulation requires. Also, this mortality level would imply that human juvenile survival is worse than that of wild chimpanzees (unlikely, given observed levels of alloparental care in humans).

Our conclusion is that only a combination of both the lowest natural fertility rates and the highest juvenile mortality rates ever ethnographically observed can come close to producing zero population growth. Such a life history probably implies resource limitations (food intake is related to both fertility and juvenile mortality in all mammals) much more severe than ever observed in any modern group of hunter-gatherers (so much for the "original affluent society" label). Because of this, both Hill and Hurtado, and Blurton-Jones explored other solutions to the hunter-gatherer paradox based on frequent population crashes (a few generations of growth followed by a serious crash repeatedly), or higher adult mortality due to warfare, or very high infanticide rates. Hill's student Keckler (1997) did simulations showing that frequent and severe population crashes (including exterminations caused by warfare, climate variations or disease epidemics) could result in long-term zero population growth, but this would require us to revise our basic understanding of human history. Blurton-Jones' conclusions were similar. The most important lesson at this point is that the hunter-gatherer demographic paradox reminds us that we still cannot state *with confidence* that we know the typical life history parameters that characterized much of human ancestral history. Modern hunter-gatherer studies do not yet give us the answer.

2) Body Size Variation Around the World

The standard mammalian LH model developed by Charnov (1991) presumes that energetic throughput is reflected by the empirically derived allometric growth law such that change in mass is a decelerating function of achieved body weight (dw/dt = Aw^.75). Charnov simplified female mammalian life history to assume that some energy harvested goes into growth during the juvenile period, and that energy is converted to reproductive effort at adulthood. This means that energy for reproduction is a direct function of body size, and, for a given species of mammal with a species-typical offspring size at birth, fertility increases monotonically with female body size. However, the proportional gains in fertility with each extra gram of body growth are lower as mammals get larger. Eventually, growing one more time unit before sexual maturity will lead to a greater proportional loss in probability of reaching reproductive age than will be the proportional gain in fertility from growing during that additional time unit. This defines the optimal age at which to stop growing and start reproducing. According to this model, then, mortality rates are the main determinant of the optimal female adult body size for any particular feeding niche and the corresponding growth rate around the age of sexual maturity. Higher mortality should lead to earlier cessation of growth and smaller adult body size. This model was adopted by Hill and Hurtado (1996: Chapter 11) in an attempt to "explain" typical adult female body size and

age at first reproduction for female Ache hunter-gatherers of Paraguay. Hill and Hurtado then employed the same model to "predict" both smaller body size and later age at reproduction for !Kung Bushmen using the published growth and mortality parameters for that group. A decade later the same model was explored by Walker et al (2006) to account for body-size variation (due to variation in both growth and mortality rates) in a sample of twenty-two small-scale traditional societies. Walker et al concluded that some populations were small because of nutrition and slow childhood growth, and others because high mortality favored early sexual maturity. Finally, the model was adopted by Migliano and colleagues (2007), using data from South East Asian Negritos and African Pygmies to suggest that short stature in general ("pygmy phenotype") was mainly due to high-mortality environments and early cessation of growth. However, the Migliano et al model was questioned by demographers working with African pygmies who found no evidence of high African pygmy mortality (Becker et al., 2010), and recently the Migliano et al model was shown to be incorrect for Baka pygmies, who have high juvenile and adult survival but are small because of genetically determined slow growth during early childhood (Ramirez Rozzi 2018). Miglano and Guillon (2012) extended their original argument and provided important cross-cultural analyses suggesting that differential mortality rates are indeed associated with much of the variance in height across a sample of small-scale populations around the world. However, their analyses are confounded by the fact that mortality and height are both strongly affected by nutrition, disease and economic well-being in all human societies (see Steckel, 2009 for review) such that a positive relationship between survival and height is expected, even when mortality rate has no direct causal impact on adult height. In their paper, Migliano and Gullion present only one result that cannot be parsimoniously explained by the association between better nutrition, better survival and higher childhood growth. That result is an apparent positive relationship between adult survival and age at menarche (ibid: table 3). However, that result seems extremely improbable and should be examined carefully. It is well known from observation and food intervention studies that greater food intake increases survival and decreases age at first reproduction (see Hill and Hurtado, 1996; tables 1.1 and 1.2 for review). The Migliano and Guillon result, if true, would contradict hundreds of studies in human and mammalian nutrition and biology that show that poorly nourished mammals show higher mortality and *later* ages of maturity. While Migliano and Guillon interpret their result to be consistent with a life history prediction that lower mortality should lead to later age at maturity, that prediction is only valid when nutritional intake is approximately constant (Charnov, 1991). In the real world, with tremendous differences in food intake across and within societies, there is no reason to expect that those who reach menarche at a later age will also have better survivorship — quite the opposite.

It is unclear whether the traditional mortality rates among South East Asian Negritos have been high enough over evolutionary time to produce the small body size of those groups. There is no year-by-year survival curve in the original Agta study, and various calculations have placed the forest-period survival rates to age of reproduction anywhere between 42% to 50% — not particularly low for a H-G population (Hewlett, 1991; Gurven & Kaplan, 2007; Migliano et al., 2007; Ramirez Rozzi, 2018; Early & Headland, 1998, Headland, personal communication). Very similar survival rate (51%) is reported for the Batak (another Asian Negrito group, see Migliano et al., 2007). One obvious alternative possibility is that South East Asian Negrito body size is an example of "insular dwarfism" acting on humans much like small body size has evolved in many other mammals living on islands. Importantly, early island populations of hominins in

this region already show very small body size, as do recent non-descendant native populations (see Mijares et al., 2010 for ancient Philippines; and Brown et al., 2004; Bromham and Cardillo, 2007; Tucci et al., 2018 for discussion of H. floresienses and primate island dwarfism). Ironically, however, models of mammalian insular dwarfism generally assume that mortality on islands is *low* due to lack of predators, and that small body size is mainly an adaptation to both feeding competition and lack of need for large body size to escape predation (Lomolino, 1985). In any case, no popular theory of insular dwarfism assumes higher extrinsic mortality as the cause (Lomolino, 2005; Meiri and Raia, 2007). This leads us to wonder about human body size variation in general across time and space. To what extent are large and small body size due to advantages of longer or shorter growth periods driven by mortality rates, and to what extent is body size an adaptation to other ecological constraints (such as climate, feeding niche, frequency of violent contests)? It is also important to note that Charnov (2001) himself stated that the power-function growth pattern across phylogenic groups was not relevant to within-species growth, and that other life history models to explain body size, assuming sigmoid growth models (growth stops when the daily energy harvested in that niche is equaled by the increasing metabolic costs of the growing body) were more appropriate. The theoretical basis for the mortality-rate-driven body size variation within species should be carefully examined anew. In this light it is important to consider adaptive explanations for small body size that are not derived from LHT at all, but may be due to factors like mobility constraints in tropical forests (Venkataraman et al., 2018). Casual inspection does suggest that the largest hunter-gatherers in the ethnographic record live in open country and the smallest often inhabit tropical forests. How much is body size variation simply due to better or worse nutrition, and how much is genetically determined adaptation to other long term ecological constraints? Why does isolation on islands lead to both notably small (e.g. South East Asian Negritos) and notably large (e.g. Maori, Somoan: Swinburn et al., 1999) mean body size? Finally, how do population differences in body size correlate with life history variables in human populations around the world?

3) The Demographic Transition

Probably the most investigated and written about topic in recent human demography is the transition to lower mortality and lower fertility that swept through many human populations beginning around the end of the eighteenth century, and which is still in progress in much of the developing world (Caldwell, 1976; Coale, 1989; Lee, 2003; Goodman et al., 2012; Sear et al., 2016; Colleran, 2016). Good evolutionary analyses have clearly demonstrated that lowered fertility, greater survival and greater investment in offspring could hypothetically maximize fitness under the right conditions, but, empirically, recent widespread fertility reduction does not maximize fitness in human populations where relevant parameters have been measured (e.g. Borgerhoff Mulder, 1998; Kaplan et al., 1995, Kaplan, 1996; Kaplan and Lancaster, 2000; Goodman et al., 2012; Bolund and Lumaa, 2017). Nevertheless, the question of whether the fertility transition is adaptive is complicated, because: (1) the fertility transition has proceeded through phases that might have been adaptive in some times and places (Hruschka and Burger, 2015); and (2) the fertility transition may not be permanent (Burger and DeLong, 2016). However, it seems clear that the demographic transition is not simply an adaptive reaction norm that maximizes fitness in modern times through low fertility. This realization forces us to examine the proximate mechanisms of fertility outcomes that might have been adaptive under past conditions but

would lead to less than maximum fitness under recent conditions (mismatch). Whatever those mechanisms, they may be under strong negative selection currently. While a clear understanding of the demographic transition will require considerably more work on the evolved phenotypic plasticity of fertility decision mechanisms (adaptive reaction norms that produce fertility variation in the range of ancestral ecological conditions), some important considerations can be identified.

Firstly, the trend to not convert increased resource access into increased fertility (or offspring survival) has existed in humans for a long time before its manifestation in the demographic transition. In other mammals, resource availability directly determines fertility or survivorship and subsequent equilibrium population density (e.g. Robinson and Redford, 1986; Boutin, 1990). In other words, if we double the resource availability, we find a short period of population growth followed by an approximate doubling of the population density on the landscape. This has not been true in humans for a long time. Instead, in historic times, when the resource base was doubled, we find only a slight increase in human population density and instead an increase in *standard of living* (and per capita income) of the population. People use extra resource availability to improve shelter quality (housing), clothing, adornment, quality and quantity of utility goods and status display to other people. These forms of "extra-somatic" investment do not generally lead to linear increases in the reproductive value of individuals and hence probably do not maximize inclusive fitness. In short, the human tendency to engage in extra-somatic investment, storage and display (wealth accumulation) rather than fitness maximization had already begun thousands of years before the demographic transition in recent times. How did such psychological mechanisms, producing wealth accumulation, status display and an increase in standards of living at the expense of fitness maximization, evolve in our ancestral past?

Secondly, early theoretical models of the demographic transition focused on examining the conditions leading to increasing investment per offspring as the cause of associated low fertility. Some models do show that very low fertility and high parental investment could be an adaptive response (e.g. Kaplan, 1994, 1996), but the conditions required for fitness maximization with very low fertility are never seen empirically. That theory shows that parents should decrease fertility when the proportional increases in offspring reproductive value from greater PI are greater than the proportional fertility loss due to that investment. While most parental investment models assume diminishing returns to PI, there are hypothetical conditions where greater investment might give accelerating returns across feasible levels of investment. For example, if, by investing a little bit more than other parents, the offspring with the highest PI get all the best jobs (winner-take-all payoff structure), or all the mates, etc., then maximum investment in a single offspring (or very few offspring) might maximize summed offspring reproductive value across all offspring (fitness). In other words, if returns to PI are positively accelerated across the entire range of feasible options, then lifetime production of just one or very few offspring might maximize fitness. But, in reality, nobody has ever shown such a payoff structure for PI in any real ecological conditions experienced by humans. Therefore, how could humans evolve such a reaction norm if the conditions have never existed?

Thirdly, the conceptualization of the demographic transition as a problem of quantity vs. quality of offspring does not jibe with observed parental behavior. Instead of parents investing more and more per offspring when they have fewer offspring, parents also invest more and more in themselves as they decrease family size. Additionally, the signalling benefits of some public "parental investment" are unclear. Opting for a higher standard of living, rather than

fitness maximization, appears an old human pattern. What really happens during the modern demographic transition? Parents have fewer children, invest more in each of them (especially education), but also buy nicer houses, clothes, fancy cars, go on expensive vacations and purchase a myriad of status enhancing and display items. Why does the human psychology prioritize such things, and under what ancestral conditions might that human psychology have arisen?

Finally, the role of cultural norms and social learning must be integrated into the biological and mechanistic view of fertility. There is overwhelming evidence that copying low fertility patterns from higher status groups is the single strongest proximate determinant of the demographic transition (Colleran, 2016). Demographers must confront cultural evolution head on if we are going to fully understand the trend to lower fertility.

4) Menopause and Cooperative Breeding

Adult human females cease ovulation at an age long before they are expected to die. While most mammals show some reproductive senescence and a short post-fertile life span, the complete termination of fertility function among females long before the typical age of adult death, and the apparent significance of post-reproductive helpers, makes human menopause a rather important life history problem. Indeed, even in relatively high-mortality hunter-gatherer societies, more than 40% of adult female years lived are experienced as post-reproductive (Levitus et al., 2013). Since fitness in mammals is strongly related to offspring production, how can the early termination of reproduction maximize fitness? This leads to two possible answers. Firstly, perhaps menopause does not maximize fitness relative to the alternative of continued reproduction. If so, then we need to determine why continued reproduction late into the lifespan is not observed in human females. Are there constraints that make this impossible even though it is typical of most mammals? Secondly, if menopause does maximize inclusive fitness, it must do so via the positive impact of post-reproductive women on their close kin.

The most popularly considered and discussed evolutionary explanation of menopause has become known as the grandmother hypothesis (Hawkes et al., 1998). However, there are half a dozen different versions of the grandmother hypothesis now which all borrow that label (and more variants are likely to be proposed). An evaluation, therefore, requires that we specify which "grandmother hypothesis" we have in mind. Evolutionary demographers working on the puzzle of menopause in the 1980s first proposed that menopause might be favoured because the probability of an older adult woman dying before her offspring were raised to "independence" was high in ancestral societies. Selection would favour women who stopped reproducing in middle age and instead invested in helping daughters and grand-offspring (cf. Williams, 1957). However, empirical mortality data quickly showed that idea was wrong. Survival is high for middle-aged hunter-gatherer women and menopause takes place when women still have a good chance of surviving another fifteen years, to the age of "independence" of their offspring. The second alternative considered was that the inclusive fitness effects of grandmother support in the human extractive foraging economy might be so high that women could maximize fitness by ceasing reproduction and focusing on helping their grandchildren once they had enough grandchildren available to help (Hawkes et al., 1989, 1998).

There are at least three problems associated with this "grandmother-helper" hypothesis. Firstly, if the reproductive value of a woman's offspring can be greatly improved by helping, why not evolve a life history of older sibling helpers, rather than grandmother helpers? After

all, siblings are twice as related to their younger siblings as is a grandmother, and should be more willing to forgo reproduction for a while to help younger siblings who share as many of their genes as their own offspring would. Kaplan et al. (2010) have proposed that the answer to this problem lies in the unique increases of energy production over adulthood found in the human economic niche. Grandmothers (and grandfathers) are far more capable of provisioning grand-offspring than are older adolescent siblings. The second problem is that the adaptive grandmother-helper hypothesis should predict that menopause is facultative: that women with many grandchildren experience menopause, and perhaps earlier, but women with few or no living grandchildren should keep reproducing directly. No such pattern is seen around the world; instead, age at menopause is surprisingly invariant across human populations. The third problem is that empirical analyses have always appeared to show that higher fitness could be achieved via direct reproduction (if it were to continue at the level that younger women achieve), than could be achieved via the helping effects of grandmothers. Grandmother help is indeed significant, but appears not to be sufficient to justify adaptive cessation of direct reproduction (Hill and Hurtado, 1991, 1996; Rogers, 1993; Sear and Mace, 2008). This suggests that menopause might be partially due to a physiological constraint of declining fertility as women age.

After the two general adaptive hypotheses for menopause were proposed, a myriad of interesting specific adaptive models have been put forward. Firstly, some researchers have suggested that the exponential nature and rate of atresia and follicular exhaustion in mammalian ovaries is a phylogenetic constraint that makes it difficult for mammals to extend fertility much beyond age forty-five (e.g., Ellison and Ottinger 2014; Jones et al., 2007; but see Cloutier et al., 2015). Since few mammals ever live to age forty-five, the steep follicular exhaustion of mammalian females is not a problem for most species. This might mean that the evolutionary puzzle for humans is not so much cessation of ovulation in middle age, but, instead, how humans evolved a long post-reproductive lifespan after follicular depletion. However, Cloutier et al. (2015) show follicular exhaustion accelerates with age in humans but not chimpanzees, suggesting that perhaps early high fertility in humans is traded off against the extension of follicular viability, and that the timing of menopause could represent an adaptation despite the constraint of slow follicular atresia. If post-reproductive lifespan, rather than timing of menopause itself, is the adaptive puzzle, one simple answer might be that the long lifespan is found in both sexes because it is selected for in males due to late life fertility (see the section on derived human life history above, and Tuljapukar et al., 2007). However, most researchers seem more inclined to point to evidence of grandmaternal assistance to descendant kin as the most likely explanation for the long female post-reproductive period (e.g., Hawkes et al., 1989). Since virtually all ethnographers report that post-menopausal women are helpful to younger kin, a simple proposition is that the inclusive fitness effects of grandmother helping (daughters, sons, grand-offspring) might be sufficient to explain the delay in senescence (and long lifespan) after reproductive function has ceased.

In fact, the grandmother-helper hypothesis can also be combined with the "fertility decline" hypothesis to incorporate cooperative breeding into the menopause model explicitly. In the Hill and Hurtado (1991, 1996: Chapter 13) menopause model, we assumed that older women could achieve about half the fertility of women at their peak fertility years, because that is approximately the decline with age seen in other primates that do not have menopause. We then asked if higher fitness would be possible via grandmother helping or by continued direct reproduction at that rate. The answer was clear: direct reproduction would still contribute more genes than helping. However,

what if we, instead, assume that natural fertility declines more steeply over time due to physiological senescence. Perhaps if fertility typically declines to 1/3 the maximum rate via follicular atresia by the mid forties, women might then achieve higher inclusive fitness by ceasing ovulation altogether and investing in grandmother helping. Indeed, Hill and Hurtado (1996: Chapter 13) develop a model showing that when Ache fertility drops to 1/6 of peak female fertility, women would indeed gain higher fitness by helping kin rather than using resources to continue direct reproduction.

An even more attractive variant of the grandmother-helper hypothesis might be termed the "inclusive fitness helper hypothesis". This idea considers that human women receive high levels of kin support for their own reproduction. What if kin helpers were generally to stop helping older women reproduce over time because of follicular atresia and decreasing fecundability with age? Investing kin members should assess how best to use their own resources and which one of their available kin to help reproduce. If younger women have high natural fecundability and older women are inefficient (due to fertility senescence) in their conversion of resources into gene copies, then most relatives should stop subsidizing the reproduction of older female relatives and instead help younger related females. This might force older females to evolve menopause as their best option, given that they would not obtain much outside kin provisioning any longer. Just such a pattern appears to have evolved in some ant reproductive queens whose declining fertility with age can be "smelled" in the hydrocarbons on the exoskeleton, and as fecundability declines they are ultimately no longer fed by workers; instead, they reabsorb their ovaries and become workers themselves (Hill & Hurtado, 2012).

The realization that the high fertility of younger women in human societies is already due to help they are receiving from their mothers and others has also led to modifications of the "reproduce vs. help" calculations. Instead of asking what fitness a woman could achieve if she continued to reproduce (at observed population fertility levels) or helped other kin, we need to think in terms of kin-group selection. Can lineages with grandmother helpers achieve higher fitness than lineages without post-reproductive helpers? Once again, we need to estimate the survival and fertility effects of grandmother-helping achieved by lineages of females without post-reproductive helpers compared to those with helpers. Which type of kin group leaves more gene copies: one with low fertility and offspring survival due to absence of helpers, but in which all females reproduce through the whole lifespan; or one with higher early fertility and offspring survival (and higher fertility of sons as well?), but in which older women all cease reproduction and become helpers at some age?

Evolutionary modelling of menopause brings focus on one of the most important issues in evolutionary demography. Can we measure the true impact of kin help on demographic parameters and document the nature of human cooperative breeding? Indeed, more data would be helpful in deciding how the human socio-reproductive system should be described. I have sometimes used the term "assisted breeding" in order to avoid confusion with strict biological definitions of "cooperative breeding" that require reproductive suppression by the dominant female (see Clutton Brock, 2002). Likewise, Kramer and Ellison (2009) have referred to the helping socio-reproductive system as a "pooled energy budget" for small-scale societies. In the past twenty years, however, dozens of evolutionary demographers have described the human reproductive system as "cooperative breeding" (Hrdy, 2000, 2005, 2017; Burkhardt et al., 2009; Mace and Sear, 2005; Kramer, 2005, 2010; Hill and Hurtado, 2009; Van Schaik and Burkhardt, 2010; Burkhardt and Van Schaik, 2016; Sear and Coali, 2011; Smaldino et al, 2013, Meehan et al., 2013). While reproductive suppression among females is not typical in humans, provisioning and helping (by kin and non-kin) of offspring and

their mothers is extensive and probably necessary for long-term population growth. How can we measure the fitness impacts of helping by kin and non-kin in cooperatively breeding social units? To examine evolutionary origins of helping, we probably want to measure the impact of kin help alone, but later human societies, including all foraging societies studied, show extensive non-kin helping as well. We need to measure the fitness impacts of non-kin cooperation (e.g. dyadic reciprocity arrangements) vs. the fitness that would be experienced without that cooperation. In the measurement of kin-helper effects, we (Hill & Hurtado, 1996, 2009) and others (e.g., Blurton-Jones, 2016; Sear and Mace, 2008) have pointed out that substitution by other kin seriously complicates the measurements. In theory, one could measure the impact of a grandmother's help on her grand-offspring's survival simply by comparing the survival of children who do or do not have a living grandmother. However, in reality, because of marginal inclusive fitness benefits, we expect that other kin will opportunistically provide substitute help when grandmother's help is missing. So, by comparing the survival of children with and without a grandmother, we may grossly underestimate the impact of a/the grandmother's help on average. This is a problem to be faced in all life history research on cooperative breeding and alloparental helping in humans.

5) Age at Sexual Maturity and First Reproduction

In most mammals, age of female sexual maturity and first reproduction are tightly coupled. As mentioned earlier, the standard mammalian LH model is that juvenile females grow until the proportional gain in reproductive value is matched by the proportional loss due to mortality. At that age females become sexually mature. Hence, fast-growing mammals and those living in high-mortality landscapes are expected to reach sexual maturity at young ages: very soon after those females conceive offspring and give birth. In humans, this model may be inappropriate for two reasons. Firstly, adult females do not reproduce using only their own energy capture and allocation, so the effects of growth and body size on future energy capture are less clear. Instead, female reproduction is highly subsidized, and marginal increases in female reproductive value are probably derived from social networks built to gain kin help rather than from growth and increase in body size. Secondly, sexual maturity in humans often takes place long before first reproduction, because of cultural patterns and social norms that regulate marriage and hence copulation frequency for young adult females. This is a prime example of how social learning and enforced social norms can interact with other ecological constraints to produce life history variation.

Age at first marriage for females (and hence regular copulation) is not highly variable in hunter-gatherer populations (always near the age of menarche — Marlowe, 2005), but varies considerably among other types of societies (e.g., Dixon, 1971; Blanc and Rutenberg, 1990; Jones, 2010). Among males, culturally determined variation in allowable age at first marriage is much greater than among females, even in hunter-gatherer societies. Hence, age at first reproduction becomes a research problem in cultural evolution and social norms, rather than simply a fitness optimization problem in biology.

6) Variation in Age of Peak Fertility

In most female mammals, peak fertility and fecundability is observed soon after sexual maturity. Male fertility is more complex, because acquiring mates often requires both strength, achieved dominance and social alliances, and hence takes place at a later age closer to peak strength. Humans do not seem to fit the pattern for either mammalian sex. First, peak female fertility

in hunter-gatherers and small-scale societies varies surprisingly. Combining yearly data into five-year intervals, we find peak female fertility ranging all the way from the first adult five-year interval (15–19 years), to middle age (30–34 years, Ramirez Rozzi, 2018: figure 3). How can we explain a fifteen-year age difference in peak female fertility across small-scale natural fertility societies where almost all women are married by their mid-teens? These differences are probably not artifacts of small samples, since there are more than two hundred women years at risk in both the samples for the societies with youngest (Aka) and oldest (Ache) peak fertility. What cultural or environmental variables might underlie such variation? Thus far there is little theory or even speculation to address this. Secondly, human males often show high fertility in middle age and well beyond the age of peak body strength (Tuljapurkar et al., 2007; Walker et al., 2002). Indeed, in some polygynous societies, male fertility peaks in their fifties even though male strength peaks uniformly in the early twenties. This is probably due to wealth accumulation and political power patterns, and a male age-specific resource production profile that generally peaks in middle age, across economies as disparate as hunter-gatherers and modern America (Koster et al., 2018; US Bureau of Labor Statistics 2017). How and why late male fertility is achieved in some societies but not others, and why male fertility does not peak at later ages for the most wealth-stratified societies in human history (Ross et al., 2018) is an interesting problem in evolutionary demography.

7) Sex Differences in Reproductive Skew (Monogamy vs. Polygyny)

In most mammals, male fertility variance is much higher than female fertility variance. This is due to the investment asymmetry in mammals whereby females obligatorily invest substantially more resources (due to internal gestation and lactation) in each offspring produced than do males, who instead compete fiercely to gain access to fertile females. Larger, stronger, healthier males with better territories or more resources to offer female mates are chosen more often by females, and win more often in direct physical competition for access to fertile females. Because the energy content of an ejaculation is minimal and no paternal investment is obligate in mammals, male fertility is limited only by access to females. In contrast, female mammals show limited differences in fertility, mainly due to body condition, energy balance and size effects on fecundity. The probability density distribution of achieved lifetime fertility is right-skewed for both males and females, but with some very successful individual males, and many more males who completely fail to reproduce. Hence, the skew in this LRS distribution is much greater in males than in females.

How does the sex difference in reproductive skew look in humans compared to other mammals, and how much does it vary across societies? And why is it that extremely high variance in male income is empirically associated with lower polygyny rather than higher polygyny rates as for most mammals (Ross et al., 2018)? Reproductive skew measures provide a good indication of potential for sexual selection. Humans are believed to be generally monogamous with exceptionally high levels of paternal investment in offspring. Is this view congruent with the measured sex differences in reproductive variance? Differences in that skew are usually a good metric of the level of polygyny typical of the species. In a perfectly monogamous species with lifelong pair bonding, reproductive skew in both sexes should be identical. In a highly polygynous species, reproductive skew is much greater in males. By calculating the ratio of male to female reproductive skew, evolutionary demographers are uniquely positioned to examine actual mating patterns, rather than reported cultural ideals in marriage practices. For example, a recent research project examining sex differences in reproductive skew in ninety-seven human societies and seventy-six mammalian

species discovered some interesting patterns (Ross et al., 2023). Firstly, humans do indeed show higher reproductive skew in males than in females. Secondly, humans have much lower sex differences in reproductive skew than most other mammals, but they fall within the mammalian range. Thirdly, polygynous human societies show larger sex differences in reproductive skew than those that practice monogamous social norms. Fourthly, polygynous human societies show lower sex differences in reproductive skew than do most polygynous mammal species. This appears to imply that paternal investment is still quite important even in polygynous human societies.

Finally, the low sex difference in reproductive skew found in humans is partially due to higher female reproductive variance than is usually true in mammals. This surprising result might be explained by the large reproductive subsidies that are required by human women during their reproductive career. Just as in other cooperative breeding species, some females can obtain a lot of help and others little or no help (maybe even mild reproductive suppression due to social stress). Hence, variation between females is not due only to health and their own resources, but also to differences in the resources and labour offered by helpers and provisioners. This is a bit like ants, where the reproductive variance between queens can be greater than the reproductive variance between drones when queens mate polyandrously and there are significant differences in colony size (and number of workers). The high female variance result seems to confirm that human societies do practice a mild form of "cooperative breeding". Further work should examine conditions under which both males and females experience high reproductive variance across human societies.

8) Extreme Longevity (Kin Assistance or Kin Parasitism)

In almost all human societies examined, a small percentage of older adults survive for several years after their productive net energy production (daily harvest minus daily consumption) rates drop below zero. For example, using data from three foraging societies, Kaplan et al. (2000) show that net energy productivity in males drops below zero (they produce less energy than they consume) by age sixty and in females by age sixty-nine. Also, individual senescence and a steep upturn in age-specific mortality rate is generally obvious by the mid-sixties to early seventies. Yet demographic data from the same groups (Ache, Hiwi, Hadza) show that 35% (Ache), 27% (Hiwi), 43% (Hadza) who survive to age fifteen will also survive to age sixty-five. And some individuals, even in traditional small-scale societies, will survive to age eighty, a full fifteen to twenty years after their net resource productivity drops below zero. Clearly this is only possible because of social provisioning of the elderly.

The widespread provisioning of elderly individuals leads to some interesting speculation about "functional" old age in human societies compared to other species, where a post-productive survival period does not exist (Rose, 1994). Do unproductive older individuals "pay back" those who feed them by providing important services (e.g. tool making, wisdom and experience in decisions, useful social and political alliances, etc.). Or are old people essentially subsidized by kin (and others) even though their inclusive fitness contributions are negative? Presumably, if this is the case, emotional bonding mechanisms are responsible for this maladaptive pattern. In simple fitness currency, their kin, and the elderly individuals themselves should both be motivated to terminate investment in their survival when the resources being used to keep them alive could be used to generate greater impact on the reproductive value of younger kin.

Geronticide is common in human history, and the highest suicide rates for both sexes are for people in their sixties and seventies (Bertolote and Fleischmann, 2002). Variation across societies, however, is substantial. In places where aged individuals are revered (often lineage-based

societies with ancestor worship), the signal value of an actual living apical lineage ancestor, who has memories of even older ancestors, might be symbolically important if it helped to legitimize property claims and rights *vis à vis* other competing lineage groups. Hence post-productive provisioning and care for older kin might make adaptive sense if we knew all the details of what exactly elderly kin members provide. In other places, however, it is hard to see that the non-productive elderly serve an fitness-enhancing function to those who care for them. Their knowledge base might be limited, or even out of date (with generational change in relevant environments), and they may undergo cognitive senescence to the point that information and advice they provide is of limited value. Nonetheless such people are sometimes kept alive for years. I, myself, for example, have lived in a house in one traditional society where I observed an elderly woman (aged eighty-six) who was unable to walk, feed herself, or even get out of bed for more than four years, was incontinent for two of those years, and yet is still bathed, fed and cared for like an infant (she is still alive at the time of writing). In such cases, extreme old age looks like a stage that diminishes the fitness of close kin, and yet, care of the aged is quite common. More data on the frequency of this life stage should lead us to examine the proximate emotional bonding mechanisms between humans that lead to cooperation, caretaking and provisioning and may result in a human life history strikingly different from other mammals.

9) Sex Differences in Lifespan

In perhaps all human societies, and among most mammal species, females have longer lifespans than do males. This difference is one of the strongest and most consistent sex differences in nature. For example, in 2018 there were only seven males among the oldest 100 people living on earth (Wikipedia 2018). The 2009 Guinness Book of World Records listed seventy-six people in the world known to be over 110 years of age. Seventy-two of those listed were female, as was the oldest chimpanzee ever known in captivity, and the ten oldest people to have lived documented since birth records have been available. Women live longer than men on average. LHT should provide guidance on this pattern. For example, it is generally accepted that higher reproductive effort per unit time is the main cause of higher mortality in males. This is expected if the payoff structure for male reproductive effort is highly skewed with ever increasing payoffs for the most successful males (see reproductive skew above). Winning in male mating competition requires very high reproductive effort per year (fighting, territorial display, signaling phenotypic quality, mate-guarding females, etc.). That high male reproductive effort must come at the expense of investing in somatic maintenance, repair and immune function, for example; furthermore, some forms of mating competition lead directly to higher mortality (such as male-male combat for females or territories). Likewise, cooperatively breeding females often live especially long lives because of the outside assistance they receive during adulthood. In cooperative breeding birds, adult workloads are lessened, and load lightening appears to improve survival and fertility (Meade et al., 2010) and may slow rates of physiological ageing (Guindre-Parker and Rubenstein, 2017).

However, early male death in humans also seems unexpected from an evolutionary perspective because a significant proportion of human males, but not females, continue to reproduce into old age (see "the derived human life history", and Tuljapurkar et al., 2007). Human males accumulate resources and political power over the lifespan, such that older males can attain considerable reproductive success. This means that there should possibly be stronger selection on male survival than on female survival after the age of menopause. These two contradictory

predictions from LHT invite researchers to provide a clear and consistent picture of the sex difference in the human lifespan. Why do males die at younger ages, despite the fact that in traditional societies it is not uncommon for men to expect more than 10% of their offspring to be conceived after they are aged fifty? How much variation is there in human societies in the sex difference in lifespan, and do the cross-cultural differences in sex differential survival to old age co-vary with the fraction of male reproduction that takes place after female menopause? Can physiological differences in disease resistance be overridden by cultural practices? For example, in most societies, female children also show slightly higher survival than male children, probably because of subtle differences in immune function. However, that small difference is easily overridden to produce excess female juvenile mortality in many societies where female infanticide and neglect are adopted in order to bias lineage sex ratios to contain more males.

10) Sex Ratio Manipulation and Non-Reproductive Adults

Humans in large modern societies often show a slightly male-biased sex ratio at birth (around 106 males/females) and that ratio usually decreases over the lifespan because males die at higher rates than females at all ages (Pongou, 2013). This pattern is not universal, however, particularly when we examine small-scale traditional societies. Male-biased sex ratios at birth and at later ages are commonly found in hunter-gatherer demographic studies. For example, 7/7 hunter-gatherer societies with measured sex ratio at birth showed male-biased ratios in one review, and 17/29 still showed a male-biased ratio in adulthood (Hewlett, 1991). In modern states such as India and China, male-biased sex ratios are extreme and related to cultural preferences for males (Hesketh and Wei Xing, 2006) who will carry on a lineage name, inherit patrilineally transmitted property and social rights, and live patrilocally with the parents who are supported in older age by co-resident children. However, the male-biased sex ratio is also common in groups and subpopulations that may not be patrilineal, have little property to transmit (e.g. Jacobson et al., 1999) and especially among societies that experience high rates of warfare (Divale and Harris, 1976). This leads us to wonder whether a coherent evolutionary model can explain an adaptive preference for sons among a majority of human societies in world history.

Fisher (1930) and others (e.g., Charnov, 1982) have outlined a basic theory of parental investment (PI) in offspring by sex. Fisher pointed out that, since all offspring have one mother and one father, populations should tend to evolve 50:50 sex ratios because when one sex was in short supply, parents producing that sex could expect higher fitness per offspring. These theoreticians noted that parental investment in male and female offspring should be equal when the population sex ratio is 50:50. If offspring of one sex is cheaper to raise to sexual maturity, then parents should simply produce more of that sex since total investment in each sex offspring should be equal. This logic led early human evolutionary demographers to assume that the 106 male to 100 female sex ratio bias at birth might simply be due to the higher juvenile male mortality rates around the world. If boys die at slightly higher rates, then average required PI per male born will be slightly lower and therefore according to this theory parents would produce slightly more sons.

The fact that so many human societies not only show male-biased sex ratio at birth, but also show forms of neglect of female children, and often male-biased adult sex ratios, suggests that we might explore another possible explanation. Perhaps humans, as cooperative breeders, have evolved male-biased sex ratios, via lineage selection, because excess males are intended statistically to function as helpers for close kin (much like the female-sex-ratio bias of workers

among ants and wasps, or the male-biased sex ratio of termite colony workers). In other words, human lineages may have evolved non-reproductive castes. This is precisely the pattern found in other cooperatively breeding mammals (McNutt and Silk, 2008; Silk and Brown, 2008). Non-reproductive males are more common than non-reproductive females in most human societies. This might be due to the many activities in which males have a slight comparative advantage, such as work effort (males have higher work capacity than females because of greater lean muscle mass), food provisioning (males produce more food than females in general), or support during conflict (males are active participants in coalitionary violence). This theory of male-biased sex ratio may link the common prevalence of a male non-reproductive "caste" to sex-ratio manipulation in traditional human societies. Children born male that later adopt female-gender socio-economic roles (childcare, weaving and manufacture of clothing and female implements, plant collection rather than hunting, etc.) are reported at significant frequencies in a large number of small-scale ethnographic studies (e.g. Jacobs, 1968; Lang, 1998). In modern societies, between 5–10% of the adult male population is exclusively homosexual (Sell et al., 1995). The fact that this phenotype has a strong genetic component (Ganna el al 2019) and must have arisen via natural selection on specific alleles, suggests that this might possibly represent an adaptive non-reproductive caste.

The term "homosexual" is not a good fit for the cooperative breeding hypothesis, which is more about socio-economic gender roles than sexuality. Such males adopt female body language, adornments, styles of speech and engage in female-specific sex roles overtly, but their actual sexual behavior is not always well-known or studied. Terms like "berdache" or "wo-spirit" (in North American ethnographic studies) and similar native terms for males who adopt a female socio-economic role exist in almost every small-scale, traditional society that is well described (in my own tribal fieldwork such males were described using local terms like "panegi" (Ache), "omegit" (Kuna), "bayot" (Binisaya), etc.). Some aspects of gender orientation in modern societies may be related to this ancestral history, but also appear to show some significant differences not discussed here.

The frequency of male-biased sex ratio and the prevalence of individuals born male who adopt female socioeconomic roles (MTF) in adulthood (e.g. Jacobs, 1968; Lang, 1998) suggests that perhaps humans as cooperative breeders have evolved to overproduce male helpers (Vasey et al 2007; Vasey and Vanderlan 2010; Vanderlaan et al., 2013). One might hypothesize that lineages producing more male offspring outcompete lineages with an unbiased sex ratio under many socioecological contexts typical of small-scale human societies. Several other observations seem to provide some support for this possibility. First, female children who later adopt male socioeconomic roles (FTM) are much more rarely described in traditional societies, and in modern societies their prevalence is still less than the prevalence of male children who adopt female identities in adulthood (MTF), although the difference in frequency has diminished considerably (eg. Sell et al., 1995; Leinung and Joseph 2020). Second, both the male-biased sex ratio and the prevalence of MTF individuals seems absent in closely related great apes (Emery Thompson, personal communication). Third, MTF individuals are more likely in large families (that could use more helpers), they are usually later in birth order, and they are often small in body size (and hence less likely to be competitive male breeders) (see Vanderlaan et al., 2011). Fourth, a few studies have found that in traditional societies, females with MTF siblings have higher fertility (e.g., Iemmola and Ciani, 2009; Vanderlaan et al., 2011), or receive other fitness-enhancing benefits (Vasey et al., 2007). Theoretical modelling suggests that such an effect could maintain the genes required for a non-reproductive caste of MTF over time (e.g., Chaladze, 2016). However, because of their apparent increasing frequency in modern

societies (Leinung and Joseph 2020), important future life history research should also examine the role and possibly adaptive nature of FTM individuals in human cooperative breeding kin groups.

Conclusion

Nancy Howell, an early pioneer and highly influential anthropological demographer, once characterized the field as "devilishly difficult but uncommonly interesting" (1986). Howell continued to lead as she made the transition from the "demography" label to the "life history" label, incorporating evolutionary biology to describe the timing and development of !Kung bushman mortality, fertility and development (Howell, 2010). I agree with Howell's assessment: human demography and life history theory are far more complex than we had originally and naively presumed back in the 1980s when I and others began to explicitly incorporate LHT into the field. This is because biology and life itself are more complicated than many of us once realized. The relationship between genotypes and phenotypes remains a complex black box for the most part, and the constraints on what is possible (rather than optimal) among imaginary phenotypic alternatives depend on a complex biochemical and developmental recipe that is only understood in the most general terms. Furthermore, the relationship between individual phenotypes and the filtered selective transmission of information (both DNA, and epigenic, including culture) renders selection to be a statistical set of patterns that are sometimes obscure with respect to the actual phenotypes of interest. I make these observations not to draw pessimistic conclusions, but simply to remind demographers of the future, that empirical studies of real populations and their actual life histories often constitute pathbreaking contributions as we struggle to determine what aspects of LHT really provide major insights in the construction of life cycles, which are dead ends, and which seemingly logical propositions later turn out to have rested on flawed assumptions or faulty measurements. Theoretical guidance is crucial, but ultimately the lessons of how human life cycles are constructed will have to be validated empirically.

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1 Note this chapter has been posted on the Open Science Framework website since 18/10/2019, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date

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SECTION 2: EVOLUTIONARY ECOLOGY AND DEMOGRAPHY

In his chapter on 'Evolution in the History of Population Thought', Kreager contrasts the "top-down" vs. "bottom-up" approaches as they differ in demography and evolutionary biology, an important distinction that we make in the Introduction as well. A "bottom-up" approach focuses on immediate social details and mechanisms, whereas a "top-down" approach is macro in scale, focusing on ultimate explanations based on evolutionary processes and broad economic or ecological conditions. The distinction can also be framed by defining "bottom-up" as a process of moving from the statistical description of data (upward) to its interpretation, and a "top-down" approach as being driven by theory and concept, which guides the approach to data.

The practical utility of a "top-down" approach is evident in the following three complementary chapters, which each draw heavily on the tradition of evolutionary ecology (see also chapters by Hill, Mace and Borgerhoff Mulder). Evolutionary ecology is a subfield in biology that has been at the forefront of using demographic methods in ecology and biology, for the simple reason that variation in evolutionary fitness results from variation in mortality and fertility. Evolutionary ecology has classically tended to study observable behaviours, and many in this field refer to themselves as behavioural ecologists. Examples of such behaviours might include the ways animals find mates, or why the amount of investment seen by parents varies from species to species. Ecology can shape the benefits of one strategy compared to another, given circumstances like the frequencies of competing strategies, the quality and distribution of resources or population density. The field grew out of the general study of animal behaviour (ethology), so a good mental image of the field is a team of biologists observing nesting birds with binoculars. Evolutionary ecologists generally tend to emphasize the outwardly observable behaviour of interest and the social and ecological conditions around it, with less emphasis on specific genetic mechanisms or variation (for genetics see the section on 'Genetic Evolutionary Demography'). Human evolutionary (or behavioural) ecologists, as part of their interest in how both demographic and behavioural outcomes vary between environments, have studied several topics of wide interest in demography, including the demographic transition, variation in age at first birth or variation in total fertility across societies.

Having these chapters early in the volume is essential, because each covers a range of factors that influence demographic patterns and each comes from a similar theoretical grounding, highlighting the reliance on ethnographic methods and quantitative analysis, a crucial confluence in evolutionary ecology's history. Note that none of them focus on an overview of a singular vital rate (fertility or mortality), but rather encompass the ways demographic patterns and surrounding ecological circumstances interact. This partly reflects the influence of life

history theory, which often studies how demographic behaviours or patterns are connected, rather than specializing in each in isolation.

Blurton-Jones is a key figure in the development of human behavioural ecology (HBE). Here, he presents us with a thorough overview of major topics in HBE, and in evolutionary anthropology generally, through the lens of his decades of work in Tanzania with a hunter-gatherer population known as the Hadza. Along with the Ache of Paraguay (see chapter by Hill), the Hadza are among the most well-known populations for long-term behavioural and demographic studies of a small-scale society (another overview of long-term demographic studies of a small-scale society can be found in the chapter by Gurven et al., who work with the Tsimane of Bolivia).

Cully and Shenk are two of the new generation of human evolutionary demographers who have worked on many topics of wide interest. In their chapter, they begin with a reference to one of our authors from section one, Bobbi Low, whom they credit as originating ecological evolutionary demography. This subfield interprets contemporary variation in demographic patterns through an evolutionary lens. They orient this subfield as focusing on two of the four Tinbergen levels, which we encounter in the next section (see the chapters on function by Mace, and on mechanism by Vitzthum). Cully and Shenk demonstrate how this approach brings a lot of flexibility and nuance to the study of social behaviour through their excellent overviews of concepts like methodological individualism and ecological selectionism (focusing on the individual and ecology, respectively).

In her chapter on contextual effects, Uggala does an excellent job of bridging the gap between traditional and evolutionary approaches to demography by focusing, in part, on differences in terminology for similar concepts in the analysis of neighbourhood effects on fertility and mortality. Many of the variables studied by evolutionary ecologists, in the "ecology" part of their endeavours, are the same as those that demographers address as neighbourhood, household or contextual effects. The chapter also gives a useful synopsis of how to handle selection effects, which is relevant for researchers of any field. Note that here we mean "selection" in the statistical sense, in that some study designs can inadvertently "select" a non-random subset of a population. In evolutionary studies, "natural selection" refers to something quite different: the non-random cross-generational "selection" of traits shared by individuals who have greater odds of survival and reproduction (see the section on The Measurement and Interpretation of Selection and Fitness for an overview of the "natural selection" type of selection).

5. Controversies and Unfinished Business in Hadza Demography and Evolutionary Ecology

Nicholas Blurton Jones

Demographic study of eastern Hadza hunter-gatherers between 1985 and 2000 showed a stable population with relatively normal parameters and quite a high rate of increase. Aspects discussed here are: population models and small remote populations; environmental keys to evolutionary demography of sub-Saharan savanna hunter-gatherers; grandmothers and longevity; men as helpers; whether economic and reproductive interests influence which norms invade, spread and endure.

Introduction

Hadza are a population of about 1000 people living in the Eyasi basin in northern Tanzania. They have successfully maintained their identity and their way of life as hunters and gatherers into the twenty-first century. Fieldwork among Hadza in the twenty-first century may tell the story of changes and continuities in the character and contexts of Hadza life (Marlowe 2010, Apicella et al 2012, Marlowe & Berbesque 2012, Berbesque et al 2016, Crittenden 2013, Wood & Marlowe 2013). Researchers may be able to use changes in Hadza circumstances as 'natural experiments' that improve our understanding of adaptation to the savanna environment in which much of human evolution took place.

My field research was confined to the twentieth century, in a collaboration with Kristen Hawkes and James F. O'Connell of the University of Utah. Pilot visits were made in 1982 and 1984, and I made a series of repeated censuses between 1985 and 2000. In 1986 and in 1989 I observed Hadza children's foraging (1989, 1997), supplementing the extensive field work by Hawkes & O'Connell in 1985–86, and in 1988 on adult foraging efficiency and time use (O'Connell et al 1988a, 1990, Hawkes et al 1989, 1997). Throughout this period, encroachment by neighbours with other economies was evident; tourists began to visit the Hadza from about 1995 and the related effects became apparent. All of this research stemmed from previous unpublished fieldwork by Lars Smith in 1974–77, and before that by James Woodburn beginning in 1959. Woodburn was the first anthropologist to write about the Hadza in English, introducing them to academic anthropology, especially in Woodburn (1968a, b), still the best introduction to the Hadza hunting and gathering life. Much of what has followed has simply added more quantification to his ethnographic summaries.

In 2016 I published a lengthy account of the fifteen years of intermittent fieldwork on Hadza demography between 1985 and 2000 (Blurton Jones 2016 'BJ2016'). After a chapter on the

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geography and resources of the Eyasi basin, and an effort to unearth the history of "outside" influences upon the area, detailed discussion of methods of age estimation and levels of in- or out-migration, the account included details of the demographic analyses. In the second part of the book, I used individual variation in the demographic measures to test for demographic effects of helpers, looking at fertility and mortality in relation to the availability of fathers, mothers and grandmothers, and siblings. These analyses were linked to our previous work on ecology and behavior, and to Marlowe's fieldwork which began while he was my PhD student in 1995 and continued until ill health forced his early retirement in 2014. In my analyses I gave close attention to the possibility that family differences in vigour or access to resources might generate false associations between helpers and helped. I was also especially mindful of Hill & Hurtado's (1996) suggestion that helpers might distribute their help in ways that obscure their effect. My 2005 and 2006 papers imply that older Hadza women, living wherever their help would maximize their own fitness, may do exactly as Hill & Hurtado suggested.

Here I summarize some of the new data and issues that might reward further attention in the Hadza or other populations, or provoke exploration in related fields. These include 1) use of population models in studies of small, remote populations; 2) the richness of the environment, and under-appreciated aspects of savanna foods; 3) issues in the study of grand-mothering and reproductive competition; 4) male reproductive strategies and the difficulty of finding father effects in the Hadza data; 5) differences between expert hunters and others, and 6) a note on behavioural ecologists' continued invasion of the social sciences.

Population Models and Small Remote Populations

In her ground-breaking study of !Kung hunter-gatherer demography, Howell (1979) made extensive use of stable population models. If fertility and mortality remain constant for a few decades they result in a stable age structure, one in which the proportion of people in any age group remains the same, even if the population is increasing or decreasing. "Stable" refers to this constancy of age structure and is distinguished from a "stationary" population, one that neither increases nor decreases. The stable population assumption has been used to derive, from large numbers of carefully studied populations, tables of age structure under different regimes of fertility and mortality. These tables, such as Coale & Demeny 1983, allow one to relatively quickly match observations to a population regime and then extrapolate many measures that can be read off the tables and compared to field observations. These can be invaluable for a quick check on the plausibility of fragmentary data and historical accounts. However, stable population models have limitations. As the world industrializes, and changes come to the most remote locations, we can expect, and many observe, quite sudden changes in fertility, mortality and migration which disrupt the use of stable population theory. Demographers actively seek remedies (Stott 2016).

Like Hill & Hurtado (1996), I aimed to make my demographic analysis independent of the values in any set of stable population tables and thus allow the Hadza to be as unusual as the data warranted. Two kinds of dependence have been discussed. While Gage (1988) implies that hunter-gatherer (HG) or other small remote populations could fail to fit any known model population, Hill &Hurtado were particularly concerned to keep their age estimates independent of model assumptions. I made no use of published models to make age estimates, nor to extrapolate unmeasured demographic parameters for the Hadza. Thus I allowed the Hadza

population to be different from, for instance, the 326 populations used to derive the Coale & Demeny (1983) models. My raw data were census and anthropometry lists; reproductive history interviews of 227 women (91 % of women aged 15–50) which provided essential data on births, relative ages and deaths of infants and small children and close relatives; "where are they now" interviews with small groups of adults in many locations (valuable for determining deaths of adults); and history interviews with a few older adults.

Like people in many isolated rural societies, Hadza do not keep records nor (until sometime early in the twenty-first century) know the year. Thus, during our observations, the great majority had no accurate knowledge of their ages. Estimating age was thus a major task of both fieldwork and data processing, as it has been among !Kung and Ache and other such populations. Diekmann et al (2017) describe a method that gives measures of error in estimates of individual ages derived from matching age ranks to known ages. My method is described at length in Chapter 4 of my book and in its supplementary information (www.cambridge.org/Hadza, click on Resources, Supplementary Materials). I used a combination of previous data, interviews about historical landmark events, such as the departure of six men to join the Kings African Rifles at the beginning of World War Two, and a major earthquake in May 1964, age rankings by the subjects, or by parents concerning their children. During our visits data were gathered on new births and deaths, both by observations and interview. Together with the ages of the youngest babies recorded during pilot visits and Lars Smith's 1977 census, and three children recognized in partial genealogies in Dorothea Bleek's notebooks from her 1930 field visit to Hadza (Cape Town University Library, Bleek archive) we compiled a list of people of "known" ages. The age estimations involved more information than the simple regression of age rank on known age in Blurton Jones et al (1992). The results survived a variety of checks described in BJ2016. The ages of old people are the most contested; some researchers still are not acquainted with Howell (1979) and Hill & Hurtado's (1996) evidence that so-called "primitive life" is not especially short. While all directly observed hunter-gatherers, like all directly observed human populations, show quite high life expectancy at age 45 (>20 more years, Blurton Jones et al. 2002, Gurven & Kaplan 2007), it is still commonly asserted that, during pre-history, few if any people lived past age 45. Indications to the contrary, such as Walker et al 1988, Mollesen et al 1993 and others are discussed in BJ2016 SM 8.7.

Dyson (1977) estimated Hadza fertility and mortality from data gathered during the 1966–67 International Biological Program visits led by Nigel Barnicot and James Woodburn. My data showed that fertility and mortality had changed very little between 1967 and my study period. Thus I was able to use the stable population assumptions to derive predictions from a simple population simulation. The simulations allowed input levels of fertility, mortality and migration to be as observed, or to be experimentally manipulated. The simulation can generate predicted age structure and other measures that were shown to stabilize rapidly and thus could be compared with the observations. The most valuable predictions are those tested by data gathered independently from the original fertility and mortality. Examples were rate of increase from the population register, age structure from anthropometry sessions and age at death (a measure independent of the risk group that has a crucial influence on age-specific mortality).

Hadza emerged as fairly typical of a high-mortality rural population. With TFR 6.1, life expectancy at birth 32.7 (and 39.2 at age 20, and 21.3 at age 45) for sexes combined, much like other hunter-gatherers (see Blurton Jones et al 2002, Gurven et al 2007), their demographic

parameters differed little from those of Europe before the late 1700s. Dyson's and my analyses imply quite rapidly increasing populations, measured as the intrinsic rate of natural increase *r*, during the second half of the twentieth century at between 0.0139 and 0.0162. Hadza mortality was intermediate between !Kung (e_0 = c.30) and Ache (e_0 = 37.8 male, 37.1 female). Confidence intervals acquired from resampling gave a 95% confidence range for Hadza fertility. Ache fertility lies above the high end of this range, and !Kung below the lower end.

Comparing my results with results from Coale & Demeny (1983) tables (BJ 2016: 183) showed nothing unusual, except for a larger difference between male and female mortality than shown in their best matching model. This suggests to me that reference to existing tables could be a safe and economical way to make use of brief field visits or fragmentary and uncertain historical data. An analogous approach could be valuable for demographic study of higher primates (Gage 1998). As Howell has pointed out, if the researcher suspects that a population shows some unusual feature, this should be visible as a departure from model tables which can be used as a null hypothesis. Given the varied census effort, the extreme mobility of Hadza, and their habit of not infrequent name changes, I did not explore the use of two-census methods used successfully on other populations by Gage et al (1988). However, more detailed analyses of fertility, mortality and reproductive strategies require much more detail than can be obtained in a quick census. Especially important are data on individuals, including even partial longitudinal data. Most of the successful recent demographic and life history studies of small remote populations have employed repeat visits, reproductive history interviews, anthropometry, and more (examples include Howell 1979, 2010, Hill &Hurtado 1996, Early & Headland 1998, Borgerghoff-Mulder 1992, 2009, Winking & Gurven 2011).

Rapid increase, and the potential for rapid increase among hunter-gatherers was discussed by Hill & Hurtado 1996, Keckler 1997, Boone 2002, and before them by Birdsell 1968, Hassan 1978 and Carr-Saunders 1922. Keckler proposed that rapid increase followed by crashes may have been representative of many hunter-gatherer populations. In my Chapter11I explore this in relation to Hadza increase, the fragmentary indications of Hadza fertility response to drought or abundance and the lack of evidence of significant density dependent regulation. In relation to seasonal movements, and secular changes in rainfall, we get a picture of a more populous, interactive and turbulent past than the traditional picture of hunter-gatherers as small isolated bands wandering alone under perpetual threat of extinction.

Environmental Keys to Evolutionary Demography of Warm-Climate Hunter-Gatherers

The sub-Saharan savanna figures prominently in discussions of human origins and in the archaeological record. In Africa, tropical forests receded and grasslands (which include bushland and wooded grassland — open grass plains are exceptional, Belsky 1990) expanded during crucial periods in human evolution. Many of us think the nature of savanna resources ultimately determined key features of our species.

One of the benefits of toiling along behind high-endurance !Kung and Hadza may be an improved appreciation of this habitat from a forager's viewpoint. Foremost, after the critical issue of water, is the rarity and unpredictability of catches of the spectacular large mammals of today's savanna, which include some (or their taxonomic kin) that dominate the archaeological record. The relationship of ungulate biomass to rainfall allows calibration of the likelihood of

bygone times with both fewer and sometimes more frequent catches in Hadza country (Coe et al 1976, O'Connell et al 1988, Blurton Jones 2016 Fig 2.3). Given year-to-year variation in rainfall (a range not unlike longer-term variation in annual averages) Hadza have experienced times with 30% less, and 30% more rainfall with approximately matched variation in large ungulate biomass between 1700 and 4800 kg/sq km.

The field observer quickly learns about the nature of the plant foods consumed daily. Because of the way savanna plants adapted to the extreme seasonal variation in rainfall, there are abundant calories available in the sub-Saharan savanna if you are able to recognize, acquire and process them (Hawkes et al 1997, Kaplan 1997). Notable "staple" food plants are many different storage organs (tubers) and durable fruits and nuts such as Baobab (Adansonia), Mongongo (Ricinodendron), Marula (Birrea) available for long segments of the dry season and into the wet season. Widely distributed are the inconspicuous fruits of the several Grewia species, which Lee showed cover a lengthy season in the Kalahari. In Hadzaland Baobab trees grow in extensive "patches" in locations as evident in Obst's 1915 map as they are today.

The abundance can be truly astonishing. Lee (1979) documents the abundance of Mongongo nuts in a grove and data on food value and processing, Wiessner (2014, fig. 2) shows the wide distribution of groves. Eastern Hadza country harbors abundant tubers (Vincent 1985), extremely abundant (we have air counts) Baobab trees. Baobab pith and seeds yields 3.81 kcal/g thus an easily gathered 10 kg totals 38,100 kcal to be processed at leisure in camp. There are no mongongos but there are Marula nuts west of Lake Eyasi where Berbesque recorded them being used by Hadza children (personal communication 1916). Peters (1987) maps the wide distribution of Mongongo, Marula and Baobab in the sub-Saharan savanna. Day-to-day variation in the amounts an individual acquires can be quite large. In this and other features the savanna plant foods resemble on a smaller scale the rare catch of a hunter, encouraging transfers from those who got more that day to those who got less.

The seasons can starkly illustrate a difference between plant food use in the forest by our nearest living non-human primate relatives, and in the savanna by hunter-gatherers. Early-wet-season fruit sets a context for foraging not unlike that of a forest-living frugivore. As two individuals stand side by side at a fruit tree there is little incentive for anything other than continued picking of what is right there in front of you, and little either can do to enhance the other's picking rate. The dry season (and much of the wet season when short fruiting seasons are over or yet to begin) presents a different picture. The abundant and nutritionally rewarding foods in the savanna come in large packages. Some are difficult to acquire but with luck can result in a large pile that can quickly be made ready to eat (e.g. roasted tubers). Others are easier to acquire but require longer processing (e.g. Baobab pods and seeds, or Mongongo nuts). At the same time, the haul may make a splendid target for a free-loader. This may contribute to the tendency for humans to regard other humans as resources (Draper 1989), together with the apparently greater ease of joining human groups than that observed among many other primates. The potential for sharing or free-loading on savanna plant foods needs to receive more detailed attention.

The most rewarding resources (deep tubers, mongongos, Baobab) are difficult for children to acquire or process, perhaps sometimes due to their lack of strength — body weight is as good a predictor of return rate as age (Blurton Jones et al 1997), and the skills appear rapidly learned (Blurton Jones & Marlowe 2002). Even Hadza children obtain only about half their daily calorie

requirements before about age 15 (Blurton-Jones et al. 1989,1997, Crittenden et al. 2013); !Kung children are even more restricted (Blurton Jones et al 1994).This difficulty of childhood access to savanna foods provides a simple ecological reason for the lengthy dependence of pre-adult humans, and the overlapping "stacked" family of dependents that results (Gurven & Walker 2006, with consequences for the mother illustrated in Blurton Jones & Sibly 1978 fig 2). Occupying savanna offered massive resources but at the cost of economically dependent juveniles. This provides an opportunity of fitness reward to adult helpers who can transfer some of their haul to related children.

Grandmothers and Longevity

In this section I summarize the new data on Hadza grandmothers. I have neither the space nor the competence to review all the current models of the evolution of grandmothering.

All known contemporary and historical (i.e. with written records) human populations show a lengthy post-fertile life and we may treat this as a product of evolution and seek explanations from natural selection. Post-fertile life is best measured as "PrR" (Levitis et al 2013). This is the proportion of years lived from first reproduction, that are lived post-reproductively (in life table notation, Lx from the age when 95% of fertility is completed divided by Lx from the age when the first 5% of fertility is achieved). Although requiring data on age-specific fertility and mortality, this avoids the debatable use of maximum observed age. Ellis et al (2017) show that on this measure post-reproductive life-spans are rare among mammals. Levitis et al Table 1 shows that human PrR substantially exceed those of other primates. The PrR measure is also high among several Cetaceans (Croft et al 2016, Ellis et al 2018, Foster et al. 2012).

Field experience among Hadza impelled Hawkes to attend to grandmothers as providers and to think about the evolutionary challenge of their existence (Hawkes et al 1989, 1997, 1998, 2018). The importance of grandmothers as helpers, or potential helpers in societies of many types (Sear & Mace 2008) should not be overlooked. Grandmothers may be under-appreciated by those who work in public health in the developing world. We should note that, while twenty-first-century gerontologists work in a world of 80-, 90- and 100-year-olds, grandmothers throughout most of the world are active women in their late 40s, 50s, 60s and early 70s.

Hawkes' "Grandmother Hypothesis" ("GMH") is a well-developed attempt to account for the origin of grandmothering by natural selection. We can begin with the contrast between the ecology of weanling great apes, able to feed themselves upon weaning (e.g. Bray et al 2018), and the ecology of dry-season savanna foods — they are abundant, and adults can acquire large quantities but children cannot. An older woman (OF) less burdened by a suckling and a series of still-young dependent weanlings can produce a surplus. Giving some of it to her adult daughter (YF) may enhance the daughter's reproductive success, thus contributing to OF's fitness. Selection might favor an OF who lives a little longer and remains a little more vigorous. In early formulations, Hawkes left the ancestral age-specific fertility as a conserved character. In recent computer modeling, Hawkes and colleagues (Chan et al 2016, Kim et al 2018) have shown that under the realistic conditions of the model, where age at last birth is allowed to evolve, it remains at the ancestral level while lifespan increases.

In Hawkes et al 1998 (see Hawkes et al 2018 for a summary) more consequences are drawn from Charnov's (1993) model of mammalian life history evolution. Increased longevity implies lower adult mortality. This is expected to lead to a later age at first reproduction and longer

growing period, just as observed (and without reference to the belief that it takes twenty years to learn to become a hunter-gatherer). Again, following Charnov's framework we should expect the prolonged period of growth to lead to a larger body size. Humans are indeed larger than their closest relatives. In O'Connell et al (1999) we included an increase in size in our argument that grandmothering might be linked to the origin of Homo and the larger body size of H. erectus. Adhering closely to the view of life history evolution presented by Charnov (1993), and in an exegesis by Hawkes (2006), Hawkes et al (1998) proposed that the combined productivity of OF and YF should enable a greater rate of production of offspring. Human fertility rates are indeed much higher than great ape fertility (Walker et al 2008), and inter-birth intervals correspondingly shorter.

Hawkes continues to expand the human traits that may have followed, such as the adaptations of infants and children in the "stacked family" to intense competition for helpers and increased payoffs for attending to and predicting the behaviour of other individuals, and an influence upon adult sex ratios and the competitive situation of males (Coxworth et al 2015). Thus a relatively simple coherent consequence of selection could be held responsible for a number of features of human uniqueness. None of the unique features of our species have lacked their particular alternative explanations. Few of the alternatives appear as coherent or comprehensive as GMH, though some share the same ecological argument for the dependency of weanlings and pre-adults. All could benefit from more empirical support.

Hawkes et al (1997) showed a positive effect of Hadza grandmother foraging time on the growth of weaned children, and Blurton Jones et al. 2005, 2006 reported that older Hadza women lived where one would predict if their help enhanced their own fitness. But we have had to wait a long time to assemble the evidence of their demographic effect (BJ2016 Chapter 18). Hill & Hurtado (1996) warned us that helpers can obscure their own effects by, for instance, directing more help to those most in need (which often may offer the greatest benefit to the helper's fitness). Since dead grandmothers cannot help anyone, and, among Hadza, the paternal grandmother seemed as likely as the maternal grandmother to live with a child, in BJ2016 Chapter18.4 I compared children who had a living grandmother with those who had neither grandmother alive. Children under 5 are more likely to survive if they have a living grandmother than if they do not. Either grandmother seems to provide a benefit. The effect remains significant if the first year of life is excluded. The effect is strongest on children of the youngest mothers. The effect is large, beta = 0.5017, p = 0.010, with Odds Ratio 1.65 (95% range: 1.13–2.41). The odds of survival with a grandmother are more than one and a half times greater than the odds of survival with no grandmother. This effect gives $l_{15} = 0.63$ with a grandmother alive and 0.45 with neither alive, a striking demographic difference.

Several alternative explanations for the apparent effect of grandmothers on child survival were examined in BJ2016 Chapter 18.5. 1) Associations between a living grandmother and child survival could simply reflect familial differences in vigour/frailty. Multilevel logistic regressions that controlled for grandmother identity did not remove the impact of grandmother presence. 2) Epidemics are known to carry off the very old and very young. This could create associations between a living grandmother and child survival, but would generate some synchrony between the deaths of grandmothers and children. The data did not support this. 3) Adult Hadza women who had lost their mother during childhood tended to be shorter and lighter, and, in other populations, smaller women have been reported to have less child rearing success than larger

(e.g. Monden & Smits 2009). Controlling for mother weight or height did not remove the "grandmother effect".

The Hadza grandmother effect seemed to be robust, resisting several alternative explanations. We assume the effect is primarily due to the large amount of food that Hadza grandmothers acquire (Hawkes et al 1989, 1997) and can be seen daily to share with younger kin. Although we have noticed the occasional strikingly warm relationship between grandfather and grandchild, grandfathers (not all are still married to the grandmother) seemed to show little effect on child survival.

Hawkes' "Grandmother Hypothesis" ("GMH") includes the proposition that the help given by grandmothers allowed a shortening of inter-birth intervals (IBI) below the lengthy IBI of higher primates. Hadza IBI, at 2.8 years, are close to the hunter-gatherer mean of 3.0 years (Walker et al 2008, Marlowe 2010). I could see no significant difference in closed, non-replacement IBI between Hadza women who had a living mother or mother-in-law ("senior helper") and those who had neither. But there was a striking and statistically significant difference in the length of successful IBI, intervals that added a surviving child to the family (BJ 2016 fig. 18.6). The median successful interval for women with a senior helper was 3.0 years, for those without, 3.67 years (the means were 3.2 and 4.8). These data support the view that a helper can allow shorter IBI to pay off in increased reproductive success (RS). The l_{15} without grandmother roughly corresponds to rate of increase in *r* of about 0.004 (doubling in 173 years) or an r of about 0.15 with grandmother (doubling in 47 years). Were grandmothers responsible for explosive human population growth rates evolving from the teetering population dynamics of our closest primate kin?

In BJ2016 Chapter 19 I tried to use the Hadza data to address Cant & Johnson's (2008), and Johnstone & Cant's (2010) (C&J) important suggestion that conflict between the generations may have played a significant part in the evolution of menopause and post-reproductive life. One essential feature of both Hawkes's GMH and C&J's suggestion is that the resources acquired by the grandmother are limited and, if used for the grandmother's continued reproduction, cannot be used for her daughter or daughter-in-law's reproduction. In the case of food collected by the grandmother, the resource is clearly divisible and depreciable in Clutton-Brock's (1991) terminology. Other forms of care, baby-sitting, or remembering where to find water in a rare extreme drought, may not share this feature and thus entail no allocation between mother and daughter's reproduction. They are non-depreciable; I think of them as "umbrella care": you could shelter five Hadza-sized children under a large umbrella as effectively as you could shelter one, and for the same cost. For instance, the effects of some aspects of 'baby-sitting', often envisaged as the main task for grandmothers by those unfamiliar with the vigour of Hadza grandmothers and their !Kung counterparts, do not diminish as the recipients of help become more numerous. Vigilance may be valuable, but in a bush camp the effort of watching over one toddler (with no door to disappear through) may not detract from the ability to watch over another, including your own. Likewise, the "library function" of old individuals provides a non-depreciable good; it benefits all who follow and costs the grandmother the same regardless of the number of beneficiaries, nor need it interfere with her own reproduction.

C&J examine conflict between the reproduction of an older female OF (mother or mother-in-law) and younger female YF (daughter or daughter-in-law). They describe different resolutions of contests over the grandmother's resources that can arise from different dispersal patterns,

which affect the relatedness between co-resident OF and YF and the fitness interests of each. Two settings contrast with the usual mammalian pattern in which young give way to old. When a group is enduring, with a mating system in which males mate during excursions outside their home group, the older should give way to the younger in reproductive competition. They suggest that this accounts for the post-reproductive life observed in Orca females (Olesiuk et al 2005, Croft et al 2016). The second setting that promotes the old giving way to the young is female dispersal. Since older literature (and some contended modern literature) claims that female dispersal characterizes hunter-gatherer and higher-primate populations, C&J suggest that this may be a key to the evolution of human menopause and post-fertile life. Female dispersal produces an asymmetry in potential competition over resources gathered by OF. Under female dispersal, while the offspring of YF are related to OF through OF's sons, the future offspring of OF are unrelated to YF and bring YF no fitness benefit. YF should contest more vigorously and prevail. OF is expected to give way, doing better to direct her resources to her son's wife's children and forgo further child-bearing.

The logic is enticing and, together with the special features of human forager resources, would account for the scarcity of post-reproductive life among mammals, where male dispersal is widely observed. There are data from patrilocal farming societies that show negative relationships between the reproduction of OF and YF, especially daughters-in-law (Strassman & Garrard 2011) and they can be used to support the importance of competition. In the Hadza data I was unable to find consistent support for negative relationships between reproduction of any category of OF and YF, either in fertility or child survival. In contrast, positive associations between OF and daughter's reproduction were both striking and significant, perhaps a challenge to our claims about allocation of resources between mother and daughter.

In discussing C&J I pointed first to the frequency with which the composition of Hadza camps changes. Moving away has been described as a solution to conflicts among hunter-gatherers. I also suggested that the breadth of Hadza food sharing, characteristic of most known hunter-gatherers (arguably a consequence of the character of savanna foods) may have overshadowed or even reversed any underlying competition between mother and adult daughter. Reduction in day-to-day variance of food intake may be a significant feature of female cooperation among foragers. Hadza serial monogamy, resulting in many families of half-sisters, may offer a potential addition to the competition models of C&J (see also Moya & Sear 2014).

The contest approach sometimes appears to differ from Hawkes' GMH by taking human lifespan as a given, and then to aim to account for a reduced age at last birth. This view would arise if one measures life span and life events not in years but as percentages of an unvarying given life span, which equates the human 70 years with a chimpanzee's 45 years (BJ2016 SI 19.2). Taking life span as a given leaves us seeking no adaptive reason for the excess of human life spans over those of other great apes.

Ancestral human dispersal patterns may be difficult to determine (Vigilant & Langergraber 2011, Koenig & Borries 2012). The original view in anthropology was heavily dependent on interviews with men about kinship terms and marriage rules (nowadays interesting to human behavioral ecologists as efforts to promote men's interests in controlling sexual access to females (Rodseth 2012), a little-discussed instance of male cooperation). The relationship of these rules to actual distribution and movement of people was rarely reported. If we take recent hunter-gatherers, and observational statistical studies as representative, we see a variety of residence

and dispersal patterns, centering on bilocal (Alvarez 2004, Hill et al 2011, Marlowe 2004a). One comment: if chimpanzees are taken to represent an ancestral female dispersal, then the brevity of post-fertile life among chimpanzees emphasizes that we should not lose sight of the importance of the transfer of divisible, depreciable resources among hunter-gatherers. The services that post-reproductive Cetaceans provide for their younger kin are as yet not totally clear. Effects on survival, strongest upon sons, are shown, as well as some evidence about the support of sons in conflicts (Foster et al 2012), leadership (Brent et al 2015) and food sharing (Wright et al 2016).

Finally, we should acknowledge that positive grandmother effects have been demonstrated in only one other hunter-gatherer population: the Ache, among whom Hill & Hurtado (1991) showed effects but reported that they did not outweigh a high hypothesized rate of continued child-bearing. Howell (2010) found negative associations of co-resident !Kung grandmother and paternal grandfather with early childhood body-mass index, perhaps not the best measure to use with 1 to 5 year olds — but her descriptions suggest the value of parents to a young couple.

Another competing view of grandparenting and the evolution of human life histories is presented in the chapter by Kaplan. Usually referred to as the "Embodied capital" theory (Kaplan et al 2000) it emphasizes the economic contributions of older men as well as older women, and the accumulation of skills during the pre-adult period. A number of writers promote an idea that links the value of learning to prolonged childhood, and that of a longer adult life to recoup the benefits, often linking this to evolutionary changes in brain size. Many variants of this view can be found, its latest re-incarnation is Pretelli et al (2022), but I think they greatly underestimate the speed with which hunter-gatherer children can learn subsistence skills.

Men as Helpers: Looking for Father Effects

Investigators of human evolution and behaviour have long believed that humans live in pairs because men contribute to the household income. Supporting evidence from hunter-gatherers is surprisingly thin. In their extensive and careful review of the literature on natural-fertility societies, Sear & Mace (2008) found little evidence that fathers' presence increased the survival or growth rates of their children. Obst (1912) regarded Hadza men as exemplary fathers, to be contrasted with their neighbours. In the field I, and doubtless others, have seen warm and attentive interactions between Hadza men and their children. Yet in my Chapter 15 on marriage and Chapter 21 on men as helpers I found only sparse support for significant effects of paternal provisioning. Although the samples are large, as large as for grandmothers, the analysis is far from simple, and may merely illustrate the difficulty of non-experimental research.

Direct observation could measure men's transfer of resources to their wives. Marlowe (2010: table 8.5) approximated this with the amounts of food brought back to camp per day by married Hadza men. He found that men with infants brought home more than men with older children or no children, or step-children. He suggested this was an indication of the importance of men's provisioning. But if we want to look at actual consequences, we must collect a larger sample than is possible in direct observation studies. Then we need some proxy measures that reflect the transfer of resources from husband to wife. Neither number of marriages, nor number of divorces seemed important given the serial monogamy that is common among Hadza.

I used two proxy measures. Firstly, in Chapter 21 on fathers as helpers, I used father absence. For each year of a woman or a child's life, I checked whether the father was recorded as still married to her or not. The probability of a divorced father being in the same camp as his children was very low (BJ2016 Fig 21.1). If the father was still married to mother, then the probability of child death was lower, but not significantly so, with p =0.330 and the 95% confidence interval for the odds ratio ranging from 0.48 to 1.28 (i.e. roughly from half the chance of a death when the father is present to substantially more deaths when he is present (OR = 1 when there is exactly no effect)). With 191 child deaths out of 695 children and 3869 child-years of data, and the father absent in one third of the child-years, this was not strong support for a helper effect of the father on the survival of children. But there were small, positive, statistically significant effects of a father's presence on the growth of children aged 5–12.

A second proxy measure (in Chapter 15 on marriage) for the evidence of an effect of men's provisioning (BJ 2016 fig 15.8a & b, and table 15.3) was the percent of adult life (20 to 40 for women, 25 to 50 for men) in a marriage ("pctmarr"). This measure was used on the assumption that transfer of resources, to be effective, would be a continuous series of events. The husband was assumed to bring food home and make some available daily, weekly and preferentially to his wife. Then the more time that a woman had a husband during her child-bearing years, the more resources she would have received. Correlations with fertility would be expected either if intercourse was more frequent within marriage or if fertility was enhanced by the supply of resources from the husband. So the positive correlations of a women's time in a marriage with their number of living children (in table 15.3 panel A, not significant in panel B) and with her fertility (number of births, in both panels of table 15.3) are not unequivocal support for an advantage from resource transfers. Success at keeping children alive ("sssurv") should provide clearer evidence of the fitness-enhancing effect of resource transfers. But there is not a significant relationship between pctmarr and sssurv among Hadza women (b =0.6025, p =0.236, adjusted R-squared = 0.4%, N = 116 women). While the beta is large, the p value was far from significant and R-squared is trivially small, "pctmarr" accounted for virtually none of the variance in "sssurv". The women seemed to gain no child survival from the presence of a husband.

Borgerhoff Mulder (2017) raised two important points about this analysis; firstly, a potential confounding factor. The women who were seldom alone would tend to have shorter average IBI than those who spent a smaller percent of their adult life in a marriage ("pctmarr"). I had shown (BJ2016 ch. 17) that among Hadza, as among many others, short IBI were associated with lower offspring survival. The confound was not difficult to test. Adding each woman's mean IBI to the regression models did not change the picture. While mean IBI was positively associated with child survival (b =0.4302, p = 0.000), controlling for its effect did not reveal a hitherto hidden benefit of pctmarr on child survival. Pctmarr remained non-significant (b = 0.1337, p = 0.659).

Given the frequency of divorce and remarriage among Hadza, we could ask whether the result was distorted by step-fathers, who Marlowe 2010 observed to bring home less food (Marlowe 2010:215). Regression of "sssurv" on the number of divorces weakly suggests the potential confound may be realistic (b = –0.1473, p = 0.056, with adjusted R-sqd 2.0%). But adding the number of divorces to the original regression model failed to generate a significant positive effect of time in a marriage upon the proportion of the woman's children that she kept

alive (ssurv = No of divorces b = –0.1456 (p = 0.063) + pctmarr b = 0.0493 (p = 0.878)). The adjusted R-sqd at 1.3%. remained trivially small.

In other circumstances, different measures of marriage may be more meaningful. Among the Pimbwe, the number of sequential marriages is a predictor of women's RS but not men's (Borgerhoff Mulder 2009). The best proxy for male transfer of resources may be different in different populations or contexts. If we look at a wider range of populations, recalling Richard Lee's (1968) demonstration of the latitudinal variation in proportions of meat or plant foods in foragers' diets, we will include many in which there is clearly greater opportunity for men to control resources and for women to have less independent access to resources than among !Kung or Hadza. In such contexts, a woman's best way to gain access to resources may be to associate with one or a few men who gain sexual access for differential allocation of resources. This is surely today a common feature of marriage but perhaps it was not the original form.

Unlike post-reproductive women, men of any age, no matter how much they could help their wife and children, have competing routes to increased fitness. Age-specific fertility of Hadza men continues well above zero into their 60s (BJ 2016 fig 7.7). In some populations, especially those with strongly maintained monogamous marriages, men's reproduction closely tracks their marital status and the age of their wives (Tuljapurkar et al 2007, Vinicius et al 2014). Such populations also tend to have lower variance in male RS (Schacht et al. 2014, Betzig 2012). But in other populations, Hadza included, men sometimes desert their wives, embark on a new marriage to a younger woman and raise a second family of children, almost doubling their RS. That only a few succeed, and many fail, does not show weaker selective gains to the successful.

Winking & Gurven (2011) added a fifth population, Tsimane, to Blurton Jones et al (2000) and Hurtado & Hill (1992) and carefully extended the comparison to cover a man's entire reproductive career. They found that "the fertility costs due to greater offspring mortality are overcome by only minor differences in the ages of first and second spouses." While they discuss some influences on the opportunity for men to remarry a younger spouse, they give little emphasis to Hurtado & Hill's (1992) "fertilities per male" (mean TFR x N of adult women / N of adult men). Perhaps we should attend to this measure as an indicator of the level of male-male competition and the pay-off for mate guarding, Schacht & Bell (2016), and as a reflection of the competitive situation in which males exist. Mate guarding is one alternative idea about the origin of marriage (Sear & Mace 2008, Hawkes et al 1995, Coxworth et al 2015, Loo et al 2017, Lukas & Clutton-Brock 2013, Opie et al 2013, Chapais 2013). We might wonder how some men succeed in the competition.

Hunting Big Game

During the 1980s fieldwork we were struck by the preponderance of large animals taken by Hadza, and the rapidity with which most of the meat disappeared to other men's huts. Experimental follows of men paid to pursue only small game showed, we argued in Hawkes et al (1991), that exclusive pursuit of small game would be the evolutionarily stable strategy (ESS) for men who foraged only for the benefit of their own children's weekly supply of meat. We have argued with Wood & Marlowe (2013, 2014) and others about this. There are several strands to the argument; most are covered at one point or another in Hawkes' papers, and I will not address them here.

Finding little indication of direct reciprocation of meat shares (Hawkes et al. 2001a), Hawkes (1990 and subsequently) proposed that some form of social consequence maintained the pursuit of big game. Hawkes labeled the process "Show-off", as conspicuously misleading a term as my "Tolerated theft" (1987). Both catchy labels have led readers astray. Tolerated theft is just a statement of the simplest economics of potential conflicts over food acquired in large packages with high temporal variance. Hadza hunters do not show off; they maintain a modest demeanor as described among !Kung by Lee (1969) and Wiessner (2014), and among Hadza and others by Woodburn 1979), nor do they fit the image of ceaseless "womanizers" given by one science writer.

Smith & Bliege Bird (2000) and Hawkes & Bliege Bird (2002) later proposed that Hadza men's pursuit of large game, and other risky pursuits, are examples of Zahavi's handicap principle (elaborated and its theoretical basis tested by Grafen 1990, Biernaskie 2014). Because of the exertion and risk of hunting large animals, in competition with lions and hyenas and perhaps at some cost to one's wife and children, and because of the availability of the meat being impossible to hide from camp members (given the easy visibility of smoke and vultures), hunters ensure that their qualities are known to many. More recently, Bliege Bird et al. (2012) have discussed "the hierarchy of virtue": competition for a reputation as the most altruistic (a large experimental literature covers such reputations); and Hawkes has emphasized the information that other men may take from a man's success at big-game hunting that may lead to concessions over access to mates. Perhaps we should become more explicit in our hypotheses about the audience's interests in the signal, and the consequences of their interest for the signaler. We might also look more closely at how the show-off proposals compare and contrast with ideas about social selection (West-Eberhard 1983, Nesse 2007, Barclay 2013).

To pursue the "show-off" idea, Marlowe and I independently asked women to nominate men who they regard as good hunters ("who often hit large animals") ("GH"). In separate small samples we find the abundance of nominations correlate positively with observed hunting success. We also find that GH have greater RS than other men (which is not uncommon, von Rueden & Jaeggi 2016). My analyses leave some mysteries. I did not find the wives of GH more fertile once their age is taken into account, and I found their children were significantly more likely to die (95% range of the odds ratio was 1.06–1.35, and BJ2016 fig. 21.2), even after one accounts for the wife's age as a continuous variable, or a wife's membership of the least successful age group (under-19-year-olds) (BJ2016Table 21.2). In my data, GH gain most of their excess RS by spending less time unmarried and by remarrying younger women (as Marlowe 2010 also reports) and raising a second family (a history not unknown in industrial societies).

The GH are a minority and, if we remove them from the sample, among "ordinary Joe's" (who all profess to hunt almost daily, with very little success, and who nonetheless collect shares from any large kill) we see a marginally significant positive effect of the father's presence in the household on their children's survival, although it is not possible to totally exclude a confound with the tendency for marriages to break up after the death of a child.

Thus Hadza men can follow alternative strategies. In addition to GH and Ordinary Joe there were seven men who worked for money from outside sources, "wage earners", who had two wives with several children surviving and growing well, and six intriguing "Cads" who, despite having children by a large number of women, had no greater RS than average. The variety of strategies illustrates that a simple dichotomy into "parenting effort" versus "mating effort"

is only a beginning. Each can take many forms. "Mating effort" especially includes a range of possibilities such as mate guarding and other strategies of competition, including "show-off" or pursuit of status. If there are alternative strategies open to men then we might expect mechanisms to have evolved that enable them to select an appropriate strategy. The process should take account of their own abilities and experience, the strategies adopted by their competitors and the availability of opportunities.

Do Economic and Reproductive Interests Influence which Norms Invade, Spread and Endure?

While GH achieve superior RS, their wives, it seems, do not. If we had found that wives of GH had higher RS we might have been tempted to stop the investigation, because the result seemed to fit so well the received wisdom that men hunt to feed their wives and children. We could avoid that temptation by, for instance, wondering whether GH get higher quality wives (Hawkes et al 2001b). In the sample of 323 marriages, controlled for the woman's age, wives of GH are slightly more likely to have been nominated as hard workers by other women ($b = 1.999$, $p = 0.019$). But another temptation, long ago discarded, is reappearing: the temptation to announce a "norm" and turn away. Let me discuss a hypothetical example.

Marlowe (2004b, 2010 fig 7.4) reports that 55% of Hadza women said they preferred to marry a good hunter. Why? To eat more meat? But note that we have shown no benefit of the meat bonanzas to the wife or young children of a good hunter. Instead, the wives are more likely to have to put up with his affairs, eventually more likely to be deserted in favour of a younger woman, and their small children will suffer a lower chance of survival. Perhaps it is just a "norm" among Hadza that women should marry a good hunter. In the uneasy relationship between simple "old-school" behavioural ecology and cultural transmission theory, and isolation from the broader history of the social sciences, some nowadays apparently find the use of the "norm" as an explanation tempting. But there may be gains from stubbornly pursuing the behavioural ecology paradigm. Do individual economic and reproductive interests influence which norms invade, spread and endure?

The idea of a norm is not useless; for instance, it brings to mind the possibility that people keep telling young women that they should marry a good hunter. These people could be self-interestedly increasing their chance of a share in the bonanzas. If the mother of two daughters persuaded both to marry a GH, she might eat meat more often. If she persuaded her sister to persuade her daughter, and her son, overhearing the exhortations, noted the likely benefits of becoming a good hunter, the older women would have a chance of eating meat at least weekly, perhaps almost daily. By her redistributions, the older woman might benefit a wide array of young kin with a greater meat intake, and this might, barely conceivably, outweigh the losses to each daughter.

So there may be reasons for some people to promote the "norm". But why should the young women fall for the propaganda? We should have asked them, although some of the youngest seemed not to know who the good hunters were (Blurton Jones 2016:272). I did ask a few women whether they would have a problem acquiring food if their husband went away for a while "on safari". Fifteen out of the eighteen said they would have no problem.

Like the older women, if the young woman could persuade her sister or cousin to marry a good hunter, she may share the supposed benefit of a more even supply of meat. If all

these women were successful in their efforts at "farming men" then we might expect to see aggregations of GH. But my Hadza field assistant said that if you were the only good hunter in camp, people came to expect too much of you. On the other hand, if there were many, you were no longer anyone special. It was best to be one of a few. Wood (2006) interviewed thirty-four Hadza men on their preference for joining a camp with skilled hunters or with poor hunters. Twenty-six preferred to join a camp of skilled hunters; only eight preferred a camp with less meat. The camp composition data may give some answers, and show whether the wife of a GH gains a different social position from others. Although at present we cannot see her gains in RS, she may more often have older related helpers in her camp than comparable women married to less successful hunters, or other social advantages which (as in other species) may translate in the longer term to fitness advantages. Perhaps the father's status affects her older children (Scelza 2010), even after the GH has moved on to another marriage. If sons of GH become GH there could be a runaway effect of female preference for GH.

So in challenging the tempting "excuse" of a "norm", neither ignoring it nor taking it as an explanation, we were led to potentially answerable questions. They centre around the shared or conflicting interests of individuals or classes of individuals. Why would anyone make propaganda, promote a norm, if the targets want to perform the desired behaviour anyway? It might be a useful rule of thumb to believe that when we are tempted to postulate a "norm" and close the investigation, we should instead look for conflicts of interest. This research strategy is not as new as the temptation. It has obvious links to research on adaptation in animal communication (Davies et al. 2012), an illustrious precedent in the work of Alexander (1979) and his students on kinship systems, and some vigorous history in the social sciences (Harris 1968, Bicchieri 2006).

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1 Note this chapter has been posted on the Open Science Framework website since 29/06/2019, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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6. Ecological Evolutionary Demography: Understanding Variation in Demographic Behaviour

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Ecological evolutionary demography is the branch of evolutionary demography that focuses on the potential adaptive value of demographic behaviour at the level of the individual. First defined by Low and colleagues some twenty-five years ago, ecological evolutionary demography has gained important ground in developing our understanding of the ultimate evolutionary ecological drivers of fertility and mortality, often in combination with more proximate determinants of these demographic outcomes. In doing so, the field has provided solutions for apparent paradoxes associated with human fertility — how humans sustain high fertility despite highly dependent young and slow development of offspring, as well as the demographic transition — and has led to an improved understanding of the basic pattern of human mortality. A third core area in mainstream demography — migration — has received less attention from an ecological evolutionary perspective, but work on dispersal generates insights into how various "push" and "pull" factors affect the costs and benefits of leaving the natal community, and how such strategies vary across individuals, households and societies. Given the broad framework underlying ecological evolutionary demography investigations of demographic behaviour, the field has outstanding potential for integration across demography and the evolutionary social sciences. We offer several potential pathways for immediate pursuit and anticipate that this will invigorate further the impact of the field on understanding human demographic behaviour.

Introduction

> Demography lies at the heart of every statement about selection.
>
> — Jones (2010, p. 74)

Biological, anthropological and formal demographers have long pursued a set of overlapping interests in parallel and with limited interchange. This is despite clear overlap in goals and methods: demography's core concepts of fertility and mortality are central to the definition of biological fitness that serves as the foundation of the evolutionary sciences (Jones, 2010) and evolution has provided much-needed theory for the primarily descriptive discipline of population demography (Kaplan and Gurven, 2008; Sear, 2016).

"Ecological evolutionary demography" (EED) (*sensu* Low, Clarke, and Lockridge, 1992) represents a marriage of these interests. It is the study of contemporary human demographic

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behaviour from an evolutionary and ecological perspective. With its origins in the fields of evolutionary and behavioural ecology, ecological evolutionary demography focuses equally (1) on how individual demographic behaviours adjust to particular socio-ecological contexts both historically and cross-culturally, and (2) how individual-level constraints affect decision-making *within* a given socio-ecological context (Smith and Winterhalder, 1992a). In particular, ecological evolutionary demography anticipates that individuals will adjust their (demographic) behaviour in the pursuit of maximizing lifetime reproductive success[1] (LRS) such that, consciously or unconsciously, an individual makes decisions that attempt to maximize fitness.[2] EED is distinct from the broader discipline of human behavioural/evolutionary ecology in its explicit interest in demographic outcomes: fertility, mortality and migration. EED overlaps with other areas of evolutionary demography, but is distinct from mainstream evolutionary demography due to its strong empirical focus on using data from (relatively) contemporary populations to: (1) understand the evolution of species-typical traits (e.g. the human mortality profile); (2) test evolutionary hypotheses about demographic traits; and (3) to understand variation in contemporary demographic patterns.

The evolutionary ecological view of human demography thus has been largely divorced not only from mainstream, medical and anthropological demography, but also from much research in the field of evolutionary demography as practised by evolutionary biologists whose work generally focuses on non-human species. For example, although both sets of scholars refer to themselves as "evolutionary demographers", ecological evolutionary demographers studying human demographic behaviour — generally from interview, survey or historical data — make up a small fraction of the evolutionary demography society,[3] the primary academic society supporting scholarship in evolutionary demography. Instead, many evolutionary demographers focus more heavily on the evolutionary biology of life history trade-offs, with a particularly strong emphasis on understanding the limits to lifespan (e.g. Carey, 2003; Zuo and others, 2018; Colchero and others, 2016; Dong and others, 2016), and how longevity trades off with fertility (e.g. Kirkwood and Rose, 1991; Gagnon and others, 2009; Bolund and others, 2016), generally with a stronger focus on animal and plant models and experimental — as opposed to observational — methods.

Chapter Outline and Objectives

In the remainder of this chapter, we hope to clarify both the particular contributions made by ecological evolutionary demographers to the broader field of evolutionary demography, and the scope for increasing integration of the EED perspective within core areas of evolutionary demography. This chapter is modelled on Low et al. (1992), and aims to provide a broad, if not comprehensive, overview of EED as it has informed understanding of core demographic concerns: fertility, mortality and migration. In each area, we synthesize recent and seminal theories and case studies and show how these provide new and important insights into the

1 More recently, and in light of worldwide demographic transitions, ecological demographers have begun to explore proxies — especially status — for LRS as ultimate motivators for demographic behaviour (see e.g. Kaplan, 1996; Mattison and Sear, 2016).

2 By fitness, we do not mean fertility. While fertility is sometimes used as a proxy for fitness, as discussed, pursuing maximum fertility often does not maximize fitness.

3 https://evodemovi.weebly.com

ultimate drivers of demographic behaviour. These sections are flanked by an expanded discussion of the theoretical and methodological toolkits used by EED and a conclusion that notes how EED is poised to contribute to our understanding of complex demographic behaviour within and across societies. While obviously relevant, ecological drivers of demographic behaviour, per se, are not central to this chapter; these are reviewed usefully by Uggla (this volume).

Core Frameworks, Methods and Datasets

Ecological evolutionary demography uses core principles from the field of human evolutionary and behavioural ecology, notably life history theory, to understand the ultimate causes of demographic behaviour. The goal of life history theory is to explain the evolution and development of strategies that optimize the usage of resources across the life course and across varying ecological conditions (Stearns 1992). Life history strategies exist at the species level as responses to past ecological conditions and at the individual level as responses to variable ecological and developmental conditions (Ellis and others, 2009). According to this framework, demographic behaviour is the outcome of allocation decisions whereby an individual chooses how to invest energy and resources across a number of competing biological demands, including somatic effort (growth, maintenance of the body, immune function) and reproductive effort (mating and parenting). Variation in life history traits such as age at sexual maturity, age at first birth, birth spacing, age at last birth, and number of offspring born, results from trade-offs in the distribution of resources or energy to these competing life functions (Stearns, 1992; Charnov, 1993; Roff, 1993). The "principle of allocation" contends that greater investment in one domain — growth, maintenance, mating, gestation, parenting — occurs at the expense of others. The costs and benefits of different strategies and trade-offs vary as a function of individual characteristics (e.g. age, sex, health status) and local circumstances (e.g. resource distribution, level of competition for mates or resources), meaning that strategies that are optimal for an individual in one environment are not optimal for a different individual in a different environment (Ellis et al., 2009; Bogin, 2009; Chisholm, 1999; Hill, this volume).

There are several aspects of the ecological evolutionary approach that complement mainstream demographic approaches. First, whereas mainstream demography is primarily "bottom-up" — building theory from observed associations — ecological evolutionary demography is primarily "top-down" — testing well-developed theories with demographic data (Kaplan and Gurven, 2008). In essence, ecological evolutionary demography tends to pursue what Ernst Mayr and then Niko Tinbergen (Mayr, 1961; Tinbergen, 1963) referred to as "ultimate" questions, surrounding the fitness value of traits in contemporary environments, whereas mainstream demographers are typically more interested in "proximate" questions, examining the correlates and predictors of patterns of demographic behaviour, often without asking why or how the behaviours benefit or disadvantage the individuals who perform them (Low and others, 1992: 5). Importantly, proximate responses to environmental factors that affect demographic behaviour will not be maintained if they are not favoured by selection. Thus, in our view, a complete understanding of demographic behaviour requires an evolutionary perspective, as this perspective is the most likely to provide information about the stability of observed associations over time and across contexts.

Second, ecological evolutionary demography focuses strongly on individual decision-making within specific contexts — employing "methodological individualism"[4] (Weber, 1978; Smith and Winterhalder, 1992a) to make inferences about how an individual's characteristics lead to optimal behaviour that is specific to that individual. Mainstream demography has historically made greater use of data aggregated at larger levels (e.g. cities, countries or other populations) to make inferences about how social and economic variables affect demographic behaviour at regional scales. A focus on the individual level is well represented in recent work in demography (a tradition known in the field as "microdemography"), but has tended to emphasize quantification of, proximate causes for, and/or policy-relevant aspects of demographic events. This difference in approach affects how the costs and benefits of demographic behaviour are understood (Low and others, 1992: 11). In particular, benefits at the societal level may be directly contradicted by individual-level benefits. For example, encouraging fertility reduction (e.g. see Bulatao, 1985) is very unlikely to be successful if such behaviour is promoted to "benefit society" and more likely to be successful if it is accompanied by tangible benefits to parents of fewer children. Daughter-neglect is similarly resistant to "public good" incentives; a variety of examples suggest that the valuation of daughters arises in relation to the perceived usefulness of those daughters to individual families (e.g. Das Gupta and others, 2003; Fraser Schoen, 2014). Indeed, EED is explicitly interested in how *variation* in demographic behaviour arises and is sceptical of inferences drawn from pooled data that compare *central tendencies* due to the problem of overextending inferences caused by the ecological fallacy[5] (Pollet and others, 2014), and the potential to obscure underlying causes of demographic behaviour that are driven by individual-, not population-level considerations (e.g. Alvergne and Lummaa, 2014; Low, 2000).

Third, ecological evolutionary demography is especially concerned with the ways in which the specific socio-ecological contexts in which individuals are embedded modify individual demographic behaviour (see also Uggla, this volume). EED employs "ecological selectionism"[6] — under the assumption that different ecologies are likely to produce different behavioural optima. For example, different types of subsistence systems correlate with different demographic behaviours in terms of age of marriage, number of marriage partners and level of fertility both across and often within societies. Across populations, horticultural societies, which are limited in terms of the labour needed to work the land ("labour-limited"), are commonly polygynous with relatively early ages at first marriage (Goody, 1976; Harrell, 1997), while intensive agricultural societies, where resources are limited by the amount of land available ("land-limited"), are more likely to be monogamous and focus investment on a smaller number of offspring. Within-society variation is leveraged by Daniel Nettle to explore how environmental harshness in contemporary England maps onto reproductive behaviour. He finds that individuals residing in deprived neighbourhoods have faster life histories,

4 Methodological individualism "holds that properties of groups [...] are a result of the actions of its individual members". (Smith and Winterhalder, 1992b). We use it here to emphasize that EED is primarily interested in variation in demographic behaviour at the individual, rather than group, level.

5 The ecological fallacy refers to incorrect inferences made by assuming relationships observed at the aggregate level represent individual-level processes (Pollet and others, 2014).

6 "Ecological selectionism" asks "What are the ecological forces that select for behavior X?" (Smith, 2000) and thus anticipates behaviours being shaped differently by different environments or socio-ecological contexts.

reproduce earlier, more often and with lesser apparent investment in each child (Nettle, 2010). There is also increasing attention being paid to understanding the effects of ecological context at multiple levels within and across communities: Mattison et al. (2022), for example, show that indicators of market integration differ across individual, household and community levels, each with different influences on reproductive and health outcomes.

Finally, while a key strength of ecological evolutionary demography is the focus on empirical work in contemporary or recent historical populations, researchers have also borrowed formal models from economics (e.g. Kaplan, 1996), population genetics (e.g. Coulson and others, 2010), and formal demography (e.g. Jones and Bliege Bird, 2014; Rogers, 1990) to draw conclusions about demographic behaviour. An exciting recent development has been the increasing incorporation of models from cultural evolutionary theory (e.g. Mattison et al., 2018; Kolodny, Feldman, and Creanza, 2018), such that demographic behaviour is predicted not solely on the basis of what behaviours are predicted to be optimal, but also on the basis of how behaviours are socially transmitted. Although the attempt to integrate these disciplines is in its early stages (Creanza and others, 2017), demographic behaviour (as opposed to demographic intent) is readily observed and may provide one of the more straightforward routes forward for refined synthesis. This line of thinking should also address with much more clarity the extent to which cultural processes may be ultimately responsible for demographic behaviour, as commonly assumed by demographic models (Low and others, 1992: 8), versus the extent to which "materialist" incentives drive demographic behaviour (Sheehan and others, 2018; Shenk and others, 2013) in line with much thinking in human behavioural ecology, versus how these two "forces" interact to drive demographic behaviour (Henrich, 2004).

Methods & Data

Congruent with its focus on the individual, ecological evolutionary demography relies primarily on datasets that include details of individuals' demographic behaviour as the behaviour manifests within particular contexts (i.e. the household and the local community). The earliest examples derive from first-hand data collection in small-scale communities, whose demographic behaviour, by "ethnographic analogy",[7] could provide unique insights into presumed behaviour of prehistoric human ancestors. James Woodburn was an early pioneer of such work with the Hadza (Woodburn, 1968; Konner, 2017); his work on the Hadza subsequently inspired numerous demographic inquiries from a "neo-Darwinian" perspective focusing on small-scale societies, including Lee and DeVore's seminal work *Man the Hunter* (1968). Nancy Howell's *Demography of the Dobe !Kung* (1979) "set the standard for hunter-gatherer demography" (Konner, 2017). This tradition has continued in more recent examples, including Frank Marlowe's *The Hadza* (2010), and Nicholas Blurton-Jones' *Demography and Evolutionary Ecology of Hadza Hunter-Gatherers* (2016). Other important works in the EED tradition include Pennington and Harpending's *Structure of an African Pastoralist Community* (1993) and Hill and Hurtado's *Ache Life History* (1996). In each case, the authors

7 "Ethnographic analogy" is used to project the behavior of such small-scale communities into the distant past, because such populations are thought to be similar to Pleistocene ancestors given the continuity of selective environments (e.g. Marlowe 2005).

have painstakingly gathered data on the demographic statuses and events experienced by individuals, including births, deaths, marriages and divorces, as well as genealogies that allow these individuals to be linked together in families and lineages. Unlike much of the data gathered in mainstream demographic work, ecological evolutionary demographers often spend years residing within their study communities, so that the data provided is of exceptionally high quality. Indeed, many contemporary methods to circumvent problems of estimating demographic events that arise in non-literate populations were pioneered by ecological evolutionary demographers (e.g. see Konigsberg and Frankenberg, 1992; Quinlan and Hagen, 2008).

A variety of secondary data sets have also propelled ecological evolutionary demography into arguably more complex social realms. These data sets are collected by individuals and groups for different purposes (Smith and others, 2011), but contain data that may be used to reconstruct individual life histories and demographic behaviour. Increasingly used by human behavioural ecologists and evolutionary demographers (Nettle and others, 2013), such data sets provide a number of specific challenges and opportunities that both expand and constrain their use in tests of evolutionarily informed hypotheses.

Common sources for secondary datasets analysed by ecological evolutionary demographers include parish records, household registers and research-driven demographic and public health data sets. Firstly, historical demographic records have been employed successfully by several ecological evolutionary demographers. Such records are invaluable for linking families across multiple generations, within specific, known historical, demographic and ecological contexts, and for detailing the variation in demographic decision-making as it relates to individual constraints and opportunities. Indeed, many of the topics of interest to human behavioural ecologists, such as choice of marriage partner, fertility and mortality schedules, evidence of parental investment and reproductive success (Smith, 2000; Smith and Winterhalder, 1992a), can be examined using data contained in parish registers, allowing for sophisticated evolutionary analysis of pre-existing data in well-described historical contexts (Boone, 1986, 1988; Voland, 2000; Lummaa, 2004; Clarke and Low, 2001).

Secondly, large, statistically robust data sets, including high-quality data on many variables of interest to evolutionary demographers, and derived from large-scale populations, are readily available and often financially cost-free to analyse. These data sets have a number of advantages compared to small primary data sets historically of interest to ecological evolutionary demographers, including large sample sizes, rich data and often longitudinal designs (Mattison and Sear, 2016) that facilitate in-depth analysis of individual life histories. They also point to significant variability within so-called WEIRD (Western Educated Industrialized Rich and Democratic) societies (Henrich and others, 2010) — variability that may be usefully mined to explore the context-specific nature of demographic behaviour in contemporary, industrialized settings (see Stulp and others, 2016).

Yet secondary data sets are subject to a number of important methodological challenges. Firstly, demographic events are often recorded long after they occurred and are subject to errors, including those due to systematic biases in recall (e.g. a propensity to forget deaths of certain classes of individuals, such as the unbaptized). There are techniques to estimate the level of under-registration that is produced by such problems (e.g. Eriksson and others, 2018)

and to infer missing data (e.g. Langkamp and others, 2010). Nonetheless, care must be taken to ensure that data are reliable for analysis (see e.g. Wrigley, 1997). Secondly, the population that is able to be registered (i.e. that is "under observation") may differ systematically from the populations about which the dataset serves to generalize. The characteristics and behaviours of migrants may differ systematically from those of individuals who remain in the study area, for example. This can make it difficult to characterize the constraints affecting different classes of individuals and, in turn, how these affect reproductive and demographic decision-making (e.g. Strassman and Clarke, 1998) and the timing of demographic events (Voland, 2000). Thirdly, because secondary data sets are compiled for a variety of different reasons, the ability to use them to examine the complex causal factors affecting individual decision-making can be limited. Reliable socio-economic information is often lacking from parish and household registers, for example, confounding attempts to describe resource-based differences that are often thought to play key roles in demographic behaviour. Large-scale secondary datasets will only have the variables deemed of interest by previous researchers, regardless of whether these are the most relevant variables for any particular analysis (e.g. Shenk and others, 2013). Fourthly, large-scale secondary data sets leave researchers with many "degrees of freedom" (Stulp and others, 2016) that affect how they operationalize variables and conduct analyses and hence draw their conclusions (e.g. Silberzahn and Uhlmann, 2015). Pre-registering protocols may decrease unintentional researcher biases (Munafò and others, 2017), but caution must be exercised assiduously to maintain objectivity. Finally, cross-cultural comparative data analysis has produced exciting results that underscore both the general and context-specific nature of demographic behaviour (e.g. Borgerhoff Mulder and others, 2009; Hill and others, 2011), but poses special difficulties due to the differences in how data were collected or studies deployed across populations. None of these difficulties applies only in relation to work in EED, and all, in our view, are outweighed by the usefulness of inferences that can generally be drawn from appropriate analyses of rich datasets (see also Stulp and others, 2016).

Fertility

Fertility, survivorship and population growth rates together define an individual's fitness. Thus, it is not surprising that fertility has been a key focus of ecological evolutionary demography at least since the 1980s (Sear and others, 2016). In that time, ecological evolutionary demographers have shed light on two key paradoxes: how humans sustain high fertility despite the high costs of childbearing, and why fertility has dropped in industrialized settings in association with the so-called "demographic transition". In addition, the field has contributed theoretical and empirical advances for every component of fertility, from understanding the variation in age at first reproduction, to understanding the predictors of fertility, to predicting interbirth intervals and parity progression, to characterizing variation in age at last birth and explaining menopause. We focus here on some of the key contributions of ecological evolutionary demographers to illustrate the breadth and promise of the field.

The Paradox of "High" Fertility

Evolutionary scholars consider the species-typical fertility of humans to be paradoxically high.[8] Despite the high costs of fertility to human females given extreme altriciality of human infants, human women have faster rates of reproduction than predicted based on non-human primate models (including those of the great apes), and the duration of breastfeeding for our highly dependent offspring is correspondingly short (e.g. Kramer, 2005; Sellen, 2001) (Figure 1). The highest population fertility on record belongs to the Hutterites, a North American Anabaptist sect, whose population reached a total fertility rate of eleven children (Eaton and Mayer, 1953) while the record fertility for an individual woman was set in the eighteenth century by a woman who reportedly gave birth to sixty-nine children (Glenday, 1988). Such figures are remarkable given how dependent human infants are — with brains three times larger than that of a chimpanzee (Navarrete and others, 2011), the energetic demands of human infants are superlative (Walker and others, 2008; Foley and Lee, 1991; Kuzawa and others, 2014). Early in the infant's life, the vast majority of calories provided to feed these demands derives from breastmilk (Sellen, 2007), seriously constraining a woman's ability to meet her own energetic demands alongside those of her young infant, not to mention other dependent offspring at older ages (Gurven and Walker, 2006).

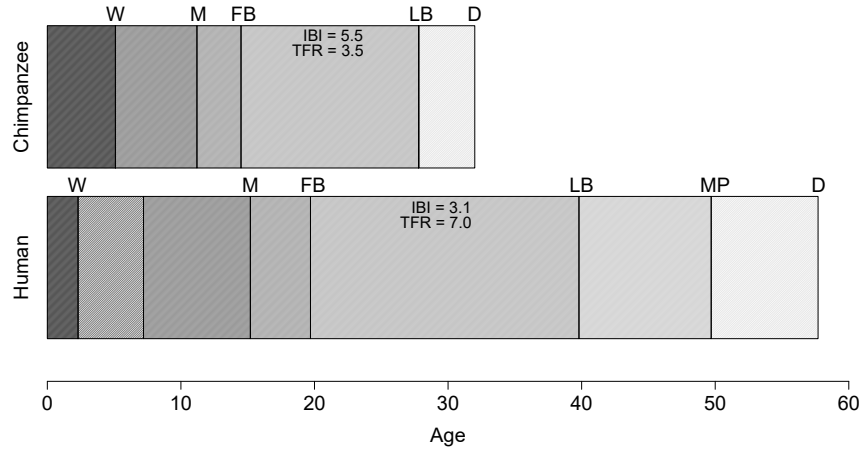

Figure 1. Life histories in chimpanzees (top) and humans (bottom). Human life histories are longer for virtually every distinct phase therein. However, human weaning occurs earlier than expected based on non-human primate models; inter-birth intervals are correspondingly short. W=weaning; M=1st menses; FB=1st birth; LB=last birth; D=death; IBI=inter-birth interval; TFR=total fertility rate. Adapted from Kramer, 2005.

The solution to this, of course, is that human mothers receive significant assistance from others ("allocaregivers"), who subsidize the high costs of child-rearing. Indeed, humans are often

8 This is in contrast to mainstream biodemography, which anticipates even higher rates of fertility, given the apparent physiological capacity to reproduce more (e.g., Bongaarts, 1975). We take this up again in describing new questions around the age at last birth.

considered "cooperative breeders" (e.g. Kramer, 2005, 2010; Mace and Sear, 2005; Sear and Coall, 2011; Hrdy, 2005), which implies that assistance in child-rearing is a key feature of the human life history. *Who* provides the most such assistance is debated. The longest-held view is that men form pair bonds with women and, in exchange for female fidelity, take up a provisioning role for their mutual children (e.g. Lancaster and Lancaster, 1987; Kaplan and others, 2000; see Mattison, 2016). This view has been challenged by proponents of the "grandmother hypothesis" (Hawkes, 2004), which posits a larger role taken by maternal grandmothers in caring for dependent offspring, together with evidence suggesting that a variety of other caretakers, including siblings (Kramer, 2005; Turke,1988; Mattison and Neill, 2013), step in at different times and places (Sear and Mace, 2008). We follow Sear (2016a) in emphasizing that while the solution to this paradox involves a *universal* tendency to assist mothers, *flexibility* in humans allows specific caretakers to assist in different contexts.

The Paradox of 'Low' Fertility

Evolutionarily high fertility in humans gives rise to the second paradox addressed usefully by ecological evolutionary demography: the demographic transition, a global phenomenon in which high fertility and mortality rates declined to low levels beginning in late-eighteenth-century Europe followed eventually by much of the remaining world. While most scholars link demographic transitions to economic, social and technological changes associated with industrialization and economic development, the specific causal mechanisms most important in transitions remain the subject of debate. Vining (1986) famously argued that "the" demographic transition contradicted evolutionary explanations for fertility given that (a) individuals voluntarily limited their fertility significantly despite increasing access to resources, and (b) wealthy and high-status people often lowered their fertility to a greater degree than people with fewer resources. Since Vining's paper was published, numerous human evolutionary demographers taking an ecological approach have tackled the question of why the demographic transition has occurred — especially why fertility has declined — and how fertility decline is consistent with evolutionary explanations.

In the broadest sense, evolutionary models of the demographic transition fall into three categories (Borgerhoff Mulder 1998): (i) some argue that the transition is optimal with respect to fitness; (ii) some that lower fertility is the consequence of Darwinian but non-genetic means of inheritance (e.g. cultural evolution); and finally, (iii) some argue that such behaviour is maladaptive.

Causal explanations in ecological evolutionary demography (i.e. (i) above) fall into several categories, which are not mutually exclusive (despite occasional claims to the contrary) — many of which align closely with approaches taken by non-evolutionary demographers (see Shenk and others, 2013 for review). Many researchers taking a life history approach have argued that reductions in rates of risk and mortality — particularly infant and child mortality — change levels of optimal fertility, motivating parents to have fewer children and invest more in each given the greater likelihood that their children will survive and reproduce (e.g. Chisholm and others, 1993; Leslie and Winterhalder, 2002; Quinlan, 2006). Other researchers examine the costs and benefits of investing in self and children. Specifically, when the costs of children are low (for instance when children's agricultural labour helps to subsidize the costs of their

upbringing), fertility should be higher than when the costs of children are high (for example, where land saturation tightly limits inheritance or in modern market economies where children are not economically productive but are costly to raise) (e.g. Kramer, 2005; Mace, 1998; Sear and Coall, 2011; Luttbeg and others, 2000). Kaplan (1996), following Becker (1993), has argued that fertility declines with increasing payoffs to investment in human capital (primarily education) in modern labour markets; these effects may be complemented by increases in adult life span and child survival rates, which also result in greater payoffs to investments in self and in children given the length of time over which benefits accrue (Cervellati and Sunde, 2005; Galor, 2012). The opportunity costs of raising children also increase in modern labour markets, especially for women (Low and others, 1992; Turke, 1989), who may reduce fertility to pursue career opportunities or otherwise delay reproduction to an age when infertility becomes more likely (Kaplan and others, 2000).

Cultural evolutionary theory (ii, above) focuses on the social processes that lead to fertility decline, arguing that humans have evolved learning biases that may lead to (or at least intensify) low fertility through emulation of high-status individuals with few children (e.g. Boyd and Richerson, 1995; Richerson and Boyd, 2005). Related models suggest that in modern societies, the decreasing density of pronatalist kin leads to increasing transmission of low-fertility norms (Newson and others, 2005). Cultural transmission models can be seen either as mechanisms of how fertility decline spreads or as causal models that posit why individuals adopt low fertility — in the former sense they are not "ecological" approaches, but in the latter sense they are.

Increased wealth does not imply a quality-quantity trade-off: Although a quality-quantity trade-off is one way to explain the demographic transition, wealth does not automatically give rise to such a trade-off. In other words, greater wealth (or maternal quality; see Emery Thompson and others, 2016; Ellison, 2003) should not, on its own, produce a fitness advantage through a reduction in childbearing. As encapsulated by Kaplan's (1996) embodied capital theory, wealth, *per se*, is not what drives investments into child quality over quantity. Rather, socio-ecological contexts that provide sufficient benefits to skills acquisition or other investments in child quality are what set the stage for steeper quantity-quality trade-offs. Because the wealthy tend to inhabit contexts that reward investments in child quality (i.e. wealth and perceived returns to parental investment often covary (Mace, 2008; Lawson and Mace, 2011)), it often appears as though humans violate the more general expectation that wealth *alleviates* the quantity-quality trade-off (Low and others, 2002; Hopcroft, 2006). If so, looking at the relationship between wealth and fertility *within* groups experiencing the same strength of fertility trade-offs should unmask a positive association between wealth and fertility that is not apparent when one does not control for the socio-ecological context producing this trade-off (Mace, 2008) (Figure 2). Few studies have attempted such a multi-level approach, but Alvergne and Lummaa (2014) found evidence both for and against an ecological fallacy applied to wealth and fertility in Mongolia — on the one hand, once context (here, urban versus rural) was accounted for, wealth showed a positive relationship with lifetime reproductive success; on the other, women's education traded off steeply with childbearing, suggesting that status acquisition could drive fertility to below-optimal levels (see also Shenk and others, 2016). Future work assessing fertility trade-offs must therefore

be attentive to how the context establishes returns on investments in child quality and how individual attempts to secure status trade off with investments in posterity.

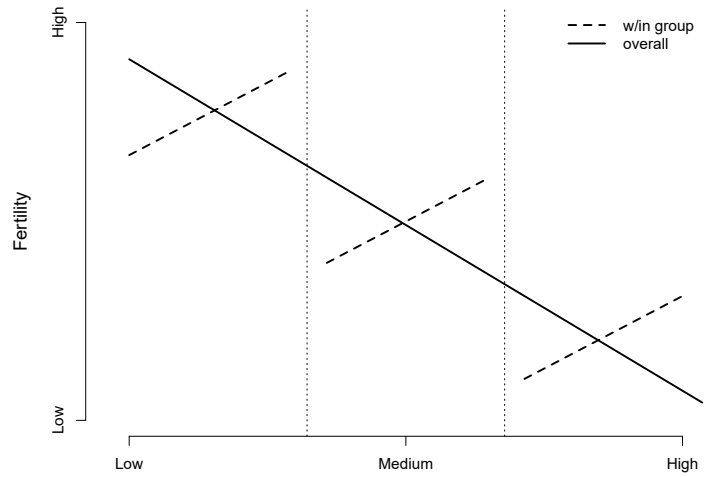

Figure 2. The ecological fallacy and the demographic transition. Data aggregated across contexts may obscure or reverse positive relationships between wealth and fertility that arise within wealth strata.

Timing of Fertility

The historical focus on overall fertility is complemented by a more recent focus on the timing of fertility, including the timing of age at first birth, interbirth intervals and, even more recently, the age at last birth. Timing of reproduction is increasingly recognized as an important contribution to fitness, particularly because fertility poorly predicts fitness in non-stationary populations (Jones and Bliege Bird, 2014). All else being equal, in growing or stationary populations, earlier reproduction is favoured (Voland, 1998): earlier-born offspring represent a greater marginal benefit to parental reproductive success than later-born offspring; and earlier reproduction shortens generation times, increasing fitness over many generations (Lewontin, 1965; Jones, 2011). At the same time, earlier reproduction reflects a key transition in a woman's life history from investments in growth to investments in reproduction (Stearns, 1992; Allal and others, 2004). The timing of this shift is important to future reproductive success, as a woman draws from the reserves built up during the pre-reproductive period to support reproduction after growth has halted (e.g. Hill and Hurtado, 1996). Accordingly, reproduction that occurs too early is associated with poor consequences for mothers and children, including low birthweight (Koniak-Griffin and Turner-Pluta, 2001), whereas greater investments into growth are associated with better outcomes, such as reduced stillbirths and infant mortality (e.g. Sear and others, 2004). In general, organisms should benefit from earlier reproduction if there are no associated costs (see Brown and Sibly, 2006), but should delay reproduction when this improves future reproductive prospects.

Trade-offs in the timing of reproduction arise across the reproductive lifespan, affecting each bout of reproduction (see Sheppard and Coall, this volume). Thus, in addition to age at first birth, trade-offs have been invoked to explain the spacing of births (Blurton-Jones, 1986) and,

more recently, the timing of age at last birth (Mattison et al., 2018; Towner, Nenko, and Walton, 2016). In general, longer interbirth intervals are interpreted to reflect increased parental investment in children (e.g, Blurton-Jones, 1986; Bereczkei and others, 2000) and as a means of protecting mothers from the physiological and energetic costs of overly rapid reproduction (e.g. Panter-Brick, 1991). Birth spacing is also a useful nexus for investigating parent-offspring conflict (Trivers, 1974) as children are wont to demand more investment from their parents than is optimal vis-à-vis parental fertility (McDade, 2001; Kushnick, 2009). As with timing of age at first birth, costs and benefits of early versus late reproduction vary according to individual circumstances, and increased availability of resources (e.g. energetic, temporal, financial) are anticipated to alleviate the costs of reproduction and to sustain faster rates of reproduction, all else being equal (Gurven and others, 2016). Unlike age at *first* birth, subsequent births may be less likely to reflect trade-offs in investments in self versus children, as major investments in self are theorized to occur prior to first reproduction (Stearns, 1992), and are more likely to reflect motivations to switch investment from one child to another. Similarly, earlier ages of *last* birth can be theorized to reflect shifts toward investments in child quality, as age at last birth is a primary means of reducing overall fertility, freeing parents to allocate resources to existing children (Towner, Nenko, and Walton, 2016; for a summary of theories on age at last birth, see Mattison et al., 2018).

In sum, fertility and the timing thereof are key drivers of fitness, affecting population growth and dynamics. Ecological evolutionary demography has provided theories addressing both why fertility is potentially so high in humans (due to our system of cooperative breeding), and why it may display, on aggregate, a negative relationship with wealth and economic development. In each case, the costs and benefits of reproduction must be weighed against competing costs and benefits of growth, maintenance, resource acquisition, and status maintenance and the likely effects of each on both current and future offspring. All else being equal, anything that acts to alleviate the costs of reproduction (e.g. presence of allocarers, wealth) can be expected to increase fertility, while anything that contributes to the costs of reproduction (e.g. physiological and energetic costs, high opportunity costs of children) can be expected to decrease it. More fundamentally, EED does not expect fertility to behave the same way in every context, but anticipates that "fertility schedules should respond to ecological conditions." Indeed, although it is possible to describe a human pattern of fertility in relation to other species, it is probably more accurate to describe human fertility as exceptionally flexible, even under "natural fertility" contexts. Thus, a major impulse in evolutionary ecological demography has been to understand the ecological and individual predictors not only of number of children, but also the timing and cessation of childbearing, including strategies surrounding the timing of reproductive maturity, the timing of childbearing, and how these trade off with investments in oneself and in parenting other children.

Mortality

Mortality is relatively little studied by EED compared to other areas of evolutionary demography. This is despite evolutionary demography — and especially biodemography — making key early contributions to theories and descriptions of human mortality (see Wachter, 2008; Sear et al., 2016 for reviews of this literature). Perhaps because mortality is less readily observed (and more difficult to ask about) than fertility, EED with its emphasis on primary data collection

in small-scale societies has engaged somewhat less with this core area of demography. Yet, mortality is central to understanding the evolution of human longevity (Hawkes, 2004; Kaplan and others, 2000) and more general patterns of life history (e.g. Charnov, 1991; Charnov and Berrigan, 1993; Ellis and others, 2009); thus, increasing research efforts in the evolutionary ecology of mortality would help to shed light on both general and site-specific causes and consequences of mortality (Burger and others, 2012). Here, we describe how EED has contributed to (1) understanding the basic pattern of human mortality, both in terms of contemporary variation and as it likely evolved over the last 200,000 years, (2) understanding how mortality reflects parental investment in children, and (3) describing how mortality can act as a predictor of variation in human life histories.

The Human Mortality Pattern

An early debate surrounding the human lifespan involved establishing a baseline, ancestral pattern of mortality. The Hobbesian view of a nasty, brutish and short human life had several proponents, including paleo-anthropologist Henri Vallois, who claimed that, among humans, "few individuals passed forty years, and it was only quite exceptionally that any passed fifty" (Vallois, 1961: 433; see also Weiss, 1981; Gurven and Kaplan, 2007). Indeed, evolutionary demographers previously believed that Paleolithic humans experienced life expectancies of only fifteen to twenty years (Cutler, 1975; Weiss, 1981). Such inferences were supported by prehistoric life tables built using osteological evidence recovered at sites such as the Libben site in Ohio (Lovejoy and others, 1977) and Indian Knoll in Kentucky (Herrmann and Konigsberg, 2002) where recovered remains revealed low infant mortality and high adult mortality. These mortality profiles were attributed to "immunological competence" acquired in childhood in small populations subjected to durable pathogenic environments (Lovejoy and others, 1977). Average life expectancies are also relatively short in chimpanzees under diverse ecological conditions (Hill and others, 2001; see also Muller and Wrangham, 2014; Emery Thompson and others, 2007; Wood and others, 2017), although it is reasonably common for individual chimpanzees to live beyond their reproductive years (Emery Thompson and others, 2007). Evidence from Neanderthals, the only other hominin to live contemporaneously with modern humans, lived for rather short durations on average (see Trinkaus, 1995), providing additional support for the idea that ancestral lifespans were significantly shorter in the human evolutionary past.

Yet life tables reconstructed based on data collected, sometimes prospectively, in diverse contemporary hunter-gatherer populations forced a revision of the foregoing view. Such data suggest that the mortality pattern that is characteristic of our species is well described by a Siler distribution (Gurven and Kaplan, 2007; Siler William, 1979; Gage, 1989; Wood and others, 2002) (Figure 3), in which mortality decreases sharply from infancy through childhood, remains more or less constant into middle age, and then rises steadily into old age in "Gompertz fashion" (Gurven and Kaplan, 2007: 322). Based on analysis of demographic data from foraging and foraging-horticulturalist communities, Gurven and Kaplan (2007) conclude that despite high mortality and significant variation across populations, a considerable fraction of humans would have lived to middle age and into post-reproductive periods even under the most stressed conditions. "For groups living without access to modern health care, public sanitation, immunizations, or adequate and predictable food supply, it seems that still at least one-fourth

of the population is likely to live as grandparents for 15–20 years." (p. 331) Indeed, Gurven and Kaplan (2007) helped to establish the slow rate of senescence in humans as a distinctive feature of human mortality profiles. Taken together, this evidence contributes to the EED view of longevity as a crucial evolved feature of the human life history (Gurven and Kaplan, 2007; Konigsberg and others, 2006), in which large-scale cooperation among individuals results in decreased mortality and frequent non-reproductive contributions to fitness (cf. Hamilton, 1966) that are focused instead on intra-familial transfers of resources and care (Lee, 2003; Kramer, 2010; Hawkes, 2004; Peccei, 2001; Kaplan and others, 2000: 200).

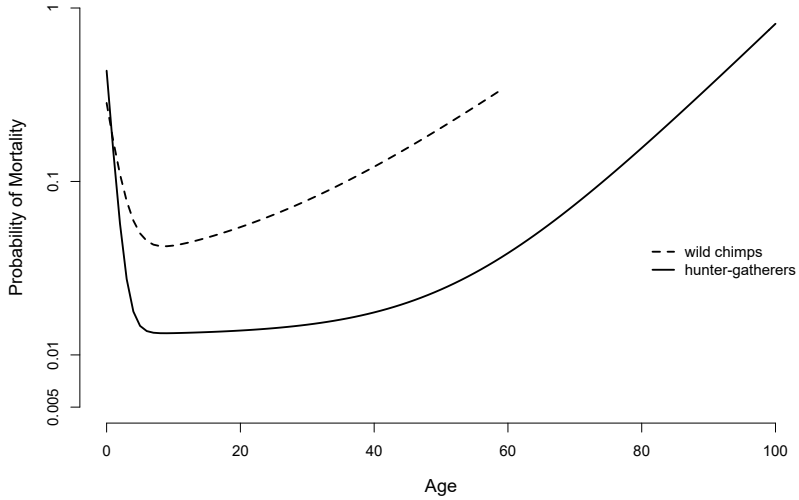

Figure 3. Characteristic mortality in humans and chimpanzees is described well by a Siler distribution and is similar in profile across these taxa, but humans have considerably lower mortality overall, and live for correspondingly longer.

Mortality Is a Proxy of Parental Investment

An obvious implication of the EED view of extended longevity in the context of cooperative breeding is that mortality is a reasonable proxy of inputs into child growth and development. Indeed, cooperative breeding is an enduring focus of work in human behavioural ecology, and probably the most common use of mortality data in EED is for testing hypotheses related to parental (and alloparental) investments in children. Parental investment theory (Trivers, 1972) stipulates that parents will invest in offspring to maximize parental reproductive success, and that such investments will be biased according to their children's ability to convert a unit of parental investment into reproductive success. Thus, son-biased parental investment is thought to pay off when sons are better able to translate investments into reproduction (e.g. due to polygyny; see Sieff, 1990) and investments in a single heir to pay off when a more even distribution of resources leads to lineage failure (Hrdy and Judge, 1993; see also Johowa and others [n.d.]). Variation in mortality can serve as a marker of non-parental investment in children as well. Mattison and colleagues (Mattison et al., 2015; Mattison et al., 2018) explored differences in mortality in adopted versus biological children in colonial-era Taiwan as a test of kin-selection theory to see whether adopted daughters were neglected compared to biological daughters and therefore subjected to higher age-specific mortality (they weren't). General tests

of cooperative breeding hypotheses frequently use vital status to infer levels of allocare from different sources (Sear and Mace, 2008). Survivorship has further been used to evaluate quality-quantity trade-offs in human populations (see Lawson and others, 2012), again reflecting the assumption that increased investment into fewer children leads to higher rates of survivorship.

This also serves as a reminder that natural selection may often favour *neglect* of children. Such neglect can range from the extreme (e.g. infanticide, abandonment) to the subtle forms of neglect that most people with siblings will claim to have been subjected to during their childhoods (Hrdy, 1992, 2009). Indeed, while rarely beneficial to a given child, parental neglect may often be optimal for parents, especially in cases where children are insensitive to parental inputs (Caro and others, 2016).

Mortality Predicts Life History Variation

Mortality provides insights as both an outcome variable and as a predictor of demographic behaviour. Major sources of mortality for humans in the course of our evolutionary history and small-scale societies include malnutrition, infectious and parasitic diseases, and conflict with other humans (Gurven and Kaplan, 2007; Gurven and others, 2007; Hill and others, 2007). Yet, as described above, ecological evolutionary demographers have influentially argued that humans have achieved major reductions in mortality compared to apes via increased cooperation through food sharing and alloparenting/cooperative breeding (Kaplan and others, 2000; Kramer, 2010). This has implications for the timing of life history stages across the lifespan.

One influential hypothesis links variation in extrinsic mortality — sources of mortality that are relatively insensitive to adaptive decisions of organisms (Stearns, 1992:182) — to differences in the progression of life histories within and across populations. According to this argument, organisms in high-mortality environments discount the future and prioritize immediacy (Pepper and Nettle, 2013) to capitalize on fitness opportunities earlier in life because a high probability of death means that reproduction is likely to be curtailed (Daly and Wilson, 2005; Ellis et al., 2009; Charlesworth, 1994; Promislow and Harvey, 1990). Similarly, harsh environments generally favour offspring quantity over quality as a bet-hedging strategy to increase the probability that at least some will survive long enough to reproduce (see Einum and Fleming, 2004; Ellis and others, 2009 for a discussion of conservative versus diversified bet-hedging). By contrast, slower life histories are favoured in environments that are predictable, not harsh, but competitive (e.g., Kaplan, 1996), because the rewards of investing in growth and the accumulation of skills and resources are likely to pay off as the future appears more secure and as competition for resources and mates among conspecifics intensifies (Ellis and others, 2009). Cross-cultural evidence supports these general expectations — age at menarche and age at first birth occur approximately one year earlier for every 10% decline in child survivorship to age 15 (Walker and others, 2006; see also Wilson and Daly, 1997; Low and others, 2008) and small body size and early fertility peaks are observed in contexts with high mortality rates (Migliano and others, 2007).

An interesting corollary of hypotheses focused on extrinsic mortality are a group of "socialization" hypotheses that link the quality of parental investment and childhood environments to rates of development. The idea here is that the quality of parental investment serves as a mechanism by which children receive information about the levels of stress and

support in local environments, including extrinsic mortality and morbidity (Belsky and others, 1991; Bereczkei and others, 2000; Chisholm and others, 1993; Ellis, 2004; Pepper and Nettle, 2017; reviewed in Ellis and others, 2009). Children reared in environments with low levels of parental investment are thought to cue in on these indicators during childhood to predict future environments and will adjust their life history strategies to accommodate harsh and/or unpredictable environments. Individuals reared in environments with cues of harshness and/or unpredictability — e.g. low socio-economic status, frequent residence or parental transitions — experience faster life histories, including earlier sexual debut, more sexual partners, and earlier age at first birth (see Ellis and others, 2009, for review, and Baldini, 2015, for a critique; see Pepper and Nettle, 2017, for a more recent review and theoretical treatment). This theory has major implications for understanding reproductive behaviour that is otherwise deemed "pathological" according to a public health perspective, and for the interventions employed to decrease the frequency of the early onset of reproduction (Draper and Harpending, 1988; Belsky and others,1991). For demography, it goes beyond standard demographic transition theory to link mortality to reproductive behaviour and attendant psychological mechanisms.

Future EED Work on Mortality

Several interesting questions remain to be addressed by an ecological evolutionary demographic perspective on mortality. Firstly, a question that continues to inspire significant interest in mainstream evolutionary demography involves whether there are limits to extensions of the lifespan (e.g. Oeppen and Vaupel, 2002; Tuljapurkar and others, 2000; Dong and others, 2016). Much of the answer to this question depends on the extent to which existing causes of mortality decline can be applied in forecasting future mortality decline. Burger et al. (2012) note that much of the exceptional decline in human mortality has arisen within only the last four generations and that the difference between contemporary mortality in industrialized populations and that of hunter-gatherers is much greater than the difference between hunter-gatherers and chimpanzees. This, in conjunction with significant contemporary variability in human mortality profiles between populations, may suggest that different factors are at work now compared to the factors operating to lower mortality in our evolutionary past. Indeed, whereas widespread sharing may have reduced deaths associated with famine and malnutrition, most deaths in contemporary hunter-gatherer populations are apparently due to infectious disease, especially post-contact, with additional mortality due to degenerative diseases and, in some groups, homicide. The contributions of modern healthcare and sanitation to declining mortality may extend the human lifespan much further than sharing (Burger and others, 2012); if such extensions facilitate ongoing downward inter-generational transfers, the implications for fitness are very different than if transfers to support longevity move in the other direction (Lee, 2013; Cyrus and Lee, 2013).

Secondly, an interesting question surrounds the extent to which fertility trades off with mortality and the types of evidence that may be used to evaluate such trade-offs. Studies exploring this issue are generally equivocal due to the difficulties associated with assessing the costs of reproduction (Gurven and others, 2016). An intriguing recent study provides evidence supporting such a trade-off in Utah where women's lifespans were more strongly lengthened following demographic transition than were men's, whose costs of reproduction were less affected (Bolund and others, 2016). More generally, if fertility is a determinant of mortality,

then its effects must be controlled in analyses of mortality. If the costs of reproduction are easily borne by contemporary women (e.g. because their nutritional inputs are sufficient to sustain high fertility or because fertility is low in most modern contexts), then mortality may be relatively immune to the effects of fertility. More empirical data testing this association are needed. Finally, and much more generally, although evolutionary demography was best known early on for its work on mortality (Wachter, 2008; Sear and others, 2016), this area of scholarship has not kept pace with work on the evolutionary ecologies of fertility, which have dominated work in modern EED. Evolutionary ecological demography stands to contribute much to this core area of demographic study.

Migration

Migration is a fundamental driver of evolutionary and demographic change, and a key component of the balancing equation in demography. While the topic is extensively studied by mainstream demographers, it has more rarely been the focus of evolutionary analysis. Yet there has been important work in this area in ecological evolutionary demography. As discussed below, much work has modelled the decisions of adults to disperse from the natal community in terms of costs versus benefits of staying versus leaving. Other work focuses more closely on "post-marital residence", i.e. the decisions made by couples over where to reside after establishing a reproductive union (Stone, 2014). Post-marital residence is highly variable in human societies (Mattison, 2019), from couples remaining in their natal communities (i.e. natalocality), to moving in with or close to the husband's kin (virilocality), to moving in with or near to the wife's kin (uxorilocality). Whether an individual disperses to a new area or stays in their natal community is relevant to key evolutionary questions of mating effort and parental investment, including access to and competition over mates and resources. Drawing from the perspectives of life history theory and the evolutionary study of territoriality, much research has examined the costs and benefits of remaining versus dispersing in different contexts with the goals of understanding when the balance is tipped in one direction or the other and how such decisions affect downstream health and demographic behaviour.

Ecological evolutionary demography provides models of dispersal decisions that unify many disparate costs and benefits (Emlen, 1995; Koenig and others, 1992). Fitness costs of dispersal range from energy, time and risk (of injury, disease, hunger, hostile people or dangerous animals in novel territories) to loss of access to nearby kin (Wood and Marlowe, 2011; Hill and others, 2011). Benefits to dispersal include the fitness benefits associated with control of new territories and associated resources (Hamilton and May, 1977) and mating opportunities (Clarke, 1993), and the reduction of inbreeding risk (Moore, 1993). Finally, scholars have recognized distinct benefits of remaining in the natal territory, including benefits derived from knowledge of local resources and risks as well as increased potential for kin investment and transmission of social information from known community members. As described below, the relative costs and benefits of staying versus leaving are predicted to differ systematically for males versus females, by age and by birth order. In humans, institutions that ratify inheritance can further constrain dispersal decisions (e.g. Clarke, 1993; Clarke and Low, 1992; Koenig, 1989; Strassman and Clarke, 1998; Towner, 2001, 2002). Access to resources has played a correspondingly large role in shaping human dispersal patterns.

Sex-Biased Dispersal

Ecological evolutionary demography has provided key insights into the role that subsistence plays in driving patterns of sex-biased dispersal. Whereas much of mainstream demography views sex-biased dispersal patterns as products of cultural institutions regulating marriage, EED pushes the causal arrow back to viewing these institutions as products of natural selection (Sear, Mattison, and Shenk 2024). For example, different ecologies are predicted to favour male versus female (or relatively egalitarian) control of resources, which, in turn, drives male versus female kin to reside together (e.g. Jordan and Mace, 2007; c.f. Alesina and others, 2013). In general, ecologies with economically defensible resources favour territoriality and group defence (Dyson-Hudson and Smith, 1978; Cashdan and others, 1983; Mattison and others, 2016b). When the resource base becomes productive enough that male reproductive success is more significantly enhanced by resources than is female reproductive success, kinship systems become more male-oriented and virilocality can ensue. Thus, many human subsistence systems, especially those emphasizing the inheritance of land in intensive agricultural systems (e.g. Goody, 1976; Shenk and others, 2010), are characterized by resource defence (Alvard, 2003) and show patterns of either female dispersal (i.e. virilocality) or (typically male) unigeniture (e.g. Boone, 1986; Goody, 1976; Hrdy and Judge, 1993; Murdock, 1967). On the other hand, subsistence systems characterized by horticulture, expansive recourse bases or male absence (e.g. due to fishing) are often uxori- or nata-local, with female kin organizing subsistence efforts (e.g. Mattison, 2011; BenYishay and others, 2017; Alesina and others, 2013; Holden and Mace, 2003). Finally, hunting and gathering are often associated with flexibility in dispersal, with spouses moving between locations strategically over the life course in ways that maximize cooperation among kin as opposed to resource defence (e.g. Wood and Marlowe, 2011; Kramer and Greaves, 2011).

Ecological Constraints on Dispersal

Human dispersal decisions are contingent not just on the subsistence system and related inheritance practices, but also on individual resource-related conditions such as the wealth and status of both self and parents (Goody, 1976; Low and Clarke, 1991; Mace, 1996; Voland and Dunbar, 1995). Such considerations are formalized by the ecological constraints model of delayed dispersal (Emlen, 1995; Koenig and others, 1992; Strassman and Clarke, 1998), which suggests that when offspring have access to cooperative breeding opportunities or improved territories at home, they may delay dispersal either because (a) they will achieve greater fitness benefits (at least temporarily) from serving as helpers at the nest (i.e. by helping to improve parental fitness (Turke, 1988)) in a good breeding territory or agricultural estate, and/or (b) with the hope of inheriting the breeding territory or agricultural estate from their parents. Emlen (1995) has argued that many aspects of the organization of the family across species rest on the principles of inclusive fitness theory, ecological constraints theory and reproductive skew theory acting in concert, with the benefits of cooperation with relatives trading off with competition for resources and reproduction, in explaining the composition and longevity of family groups as well as the age and sex characteristics of dispersers. More specifically, ecological constraints on the resources needed for reproduction (e.g. Koenig and Mumme, 1987), in combination with the benefits of staying in the natal territory under such conditions (e.g., Stacey and Ligon,

1991), have been argued to lead both to reproductive delay and, as a consequence of such delay, to the formation of extended family units (Emlen, 1994, 1995).

Fundamental to the ecological constraints model is the lack of superior alternatives away from the natal household. Interestingly, if the opportunity costs of leaving are low, then many of the factors that are associated with delayed dispersal in the context of ecological constraints lead to dispersal in the absence of such constraints. For example, non-heirs — especially those born at higher birth orders, in larger families or in areas with harsher ecological conditions — are more likely to benefit from dispersal than children with lower birth orders, who reside within smaller families or who are formally appointed as heirs (Boone, 1986; Clarke and Low, 1992; Hrdy and Judge, 1993; Voland and Dunbar, 1995). The likelihood of dispersal among humans peaks in the late teens and twenties (Clarke and Low, 1992; Castro and Rogers, 1984) across cultures. Childless and unmarried people — the same categories of individuals predicted to have the lowest opportunity costs of caring for siblings (e.g., Kramer, 2005) — are more likely to disperse than married individuals or individuals who have children, because these categories of individual will benefit more from additional opportunities to secure mates or the resources necessary to start a family (Glover and Towner, 2009; Strassman and Clarke, 1998; Towner, 2002, 1999).

Push and Pull Factors

The ecological evolutionary perspective focusing on the costs and benefits of dispersal (and as a corollary the costs and benefits of natal philopatry) parallels the discussion of push and pull factors in the study of migration among demographers. Push factors are the reasons that motivate people to leave one community (e.g. poor job prospects, land saturation, high mortality rates) and pull factors are the reasons that motivate people to move to a new community (e.g. good job prospects, access to land and better health care) (e.g. Schoorl and others, 2000; Massey and others, 1994; Jedwab and others, 2015). Most of the work on dispersal in EED focuses on these motivations, providing a link to the literature in mainstream demography. Yet there has been less attention among human evolutionary demographers to recent and ongoing patterns of rural to urban migration and international migration from the developing world — a central focus of migration scholarship in mainstream demography (see Mace, 2008). This will inevitably affect demographic studies of small-scale populations (Neill, 2007; Mattison and Sear, 2016), however, and theoretical links between urbanization, risk, fertility and parental investment (Hrdy, 1992; Mace, 2008) suggest a productive nexus for theoretical and empirical work in ecological evolutionary demography. Gillian Bentley and colleagues, for example, have examined the impact of growing up in Bangladesh vs. in the UK on reproductive function among women of Bangladeshi origin through the lens of life history theory, arguing for a critical period of environmental sensitivity during childhood. They found that growing up in the more stressful environment of Bangladesh (in terms of nutritional stress and exposure to infectious disease) was associated with lower allocations to reproductive effort in terms of progesterone levels (Mora and others, 2007) and ovarian reserves (Begum and others, 2016), but not in terms of levels of estradiol (Núñez-De La Mora and others, 2008) or age at menopause (Murphy and others, 2013). Such insights are important when considering how to extend the demography of small-scale societies within the contexts of migration, where characteristics

of sending populations may suggest different interventions into health and well-being for migrants than those employed as standard in receiving populations.

Additional topics that are of central importance to understanding ecologies of migration, if somewhat peripheral to ecological evolutionary demography, include the genetic signatures of migration, which have been used to map the journey out of Africa onto humans' contemporary global distribution. This work has taken many dimensions, including tracing the timing and route of the migrations through archaeological and genetic markers (e.g. the many articles in Crawford & Campbell, 2012 and Cavalli-Sforza et al., 1994), with some arguing that there has been selection among humans for alleles that favour expansion or migratory behaviour under conditions of resource surplus (e.g. Harpending and Cochran, 2002). While many mainstream demographers do not share the interest of anthropologists in the ancient human past, understanding these patterns creates a baseline for understanding policy-relevant types of human migration in the contemporary world and recent past. Another exciting perspective that will add to our broader understanding of human migration derives from cultural evolutionary theory, which has been particularly interested in the effects of migration on social learning processes. For example, unlike its effects on population structure, migration need not erode between-group cultural variation as acculturation to local norms and customs can serve to maintain barriers (Mesoudi, 2017). Similarly, social assortment prevents acculturation and has interesting implications for the maintenance versus erosion of cooperation within groups (Mesoudi, 2017; Boyd and Richerson, 2009) and the likelihood of large-scale demographic events such as warfare (Divale, 1974; Macfarlan and others, 2018; Mathew and Boyd, 2011; Richerson and Boyd, 1998). This work has direct relevance to mainstream demographers interested in diffusion models of behaviour, and also to the patterns and pace of the demographic and social assimilation of immigrants into host populations.

In sum, ecological evolutionary demography has much to contribute to understanding migration decisions. Although historically focused on specific decisions surrounding marriage and family-building, the ultimate rationale provided by evolutionary theory is poised to provide a unifying model of the push and pull factors that have elsewhere been described to affect migration decisions in other contexts (e.g. labour migration). Because migration affects access to resources and social support, it has important consequences for the key drivers of human decision-making, affecting all realms of interest for human behavioural ecologists (Winterhalder and Smith, 2000).

Concluding Thoughts: Key Insights, Limitations, and New Directions

In the quarter century since Low, Clarke & Lockridge (1992) published their article defining the field of ecological evolutionary demography, we have learned much about how individual-level constraints and differences in socio-ecologies affect fertility, mortality and migration. Key topics addressed by this work include resolving both why humans have, as a species, higher fertility than expected based on our long life histories, and why fertility has dropped in association with the demographic transition. The field has also described the basic pattern of human mortality and the reasons our mortality is so low, as well as its interlinkages with other core topics within demography (e.g. fertility and the lifespan). Ecological evolutionary demography has engaged somewhat less with migration studies, but ecological constraints theory and optimization approaches are poised to unify the disparate factors known to affect

the decisions of whether, when and where to migrate. In our review of this material, we have touched on several limitations or fringe topics that we believe will be important to revisit as the field continues to grow and strengthen. We draw attention to those here and offer additional suggestions that aim to further integrate ecological evolutionary demography with other core areas of the wider field of evolutionary demography.

A fruitful pathway for integrating the various subfields of demography, including evolutionary demography, is to begin to bridge more systematically the proximate-ultimate division that characterizes much of the current scholarship. An emerging area with good potential to do this involves the study of psychological mechanisms underlying fertility decisions (McAllister and others, 2016; Pepper and others, 2016; McAllister and others [n.d.]). For example, desired family size, including both what happens when you surpass your desired fertility (Mcallister and others, 2012), as well as the "unmet need" or unfulfilled desire for children in post-demographic transition contexts (Testa, 2007), when mothers don't have as many children as their stated fertility desires (Kaplan and others, 2003), are usefully studied from an evolutionary perspective and address key questions in the mainstream demography of fertility. Likewise, much of the work in cultural evolution of fertility describes the uptake of contraception through social networks and in relation to individual circumstances (Colleran and Mace, 2015; Alvergne and others, 2011; Colleran, 2016). Although there is significant debate about whether cultural evolutionary theory is better described as proximate or ultimate (e.g. Laland and others, 2013; Bateson and Laland, 2013), this may actually position it quite well for linking these two perspectives in relation to the mechanisms driving fertility decisions, as well as the adaptive value and long-run dynamics of demographic behaviour.

Various intersections between core areas of demography provide additional scope for extensions of traditional realms of inquiry into more complex understandings of human demographic behaviour. As alluded to above, the feedbacks between fertility and mortality create one nexus that will shed light on demographic behaviour in the past, present, and future. For example, the Neolithic transition, which was accompanied by global shifts toward agricultural and sedentary lifestyles some ten to twelve thousand years ago (Bentley and others, 2009; Bocquet-Appel and Bar-Yosef, 2008; BocquetAppel and others, 2006) is often considered paradoxical, in that increased fertility and mortality were simultaneously thought to have accompanied this transition. Recent scholarship testing key premises of this transition in contemporary small-scale populations transitioning to sedentism have revealed how sedentism can in fact produce the hypothesized effect, with overall increases in fertility despite increased mortality (Page and others, 2016). In particular, cooperative breeding has been key to sustaining high fertility despite increased infectious disease accompanying sedentary lifeways.

There is also interest in the interaction of fertility and mortality, both in our evolutionary past and in the modern world where the average life expectancy for humans has increased "linearly at almost three months per year over the past 160 years" (Gurven and Kaplan, 2007: 321) and women now live almost a third of their lives in a post-reproductive phase. Some findings have shown clear trade-offs between high fertility and mortality, a phenomenon known as maternal depletion, with high fertility being associated with higher mortality in some studies (Gagnon and others, 2009) but not in others, and with reviews of the evidence showing complex results consistent with maternal depletion in some settings, including modern settings (e.g. Hurt and others, 2006; Le Bourg, 2007). More recent work in contemporary high fertility populations

suggests, however, that women may often be buffered against trade-offs between health and high fertility, even in high mortality settings (Gurven and others, 2016). An intriguing recent hypothesis suggests that *low* fertility is to blame for the uptick in female morbidity (especially auto-immune conditions) in many contemporary settings (Natri and others, 2019). Much remains to untangle about the relationship between ecology, fertility and longevity in this complex relationship.

Expanding methodologies provides further scope for integration across the subfields of demography. One means by which this is already occurring is via the use of new methods that provide information on proxies of health and demographic behaviour. Central among these are biomarkers that provide information on endocrine and immune function (e.g. McDade and others, 2007; Worthman and Costello, 2009; Valeggia, 2007). The adoption of the use of mobile phones and other devices such as motes (wireless sensing devices) in data collection facilitate tracking of complex social networks (e.g. Page and others, 2017), migratory patterns and other microdemographic data (e.g. disease transmission (Marcel Salathé and others, 2010) that can be challenging to collect via observation or survey. The use of such methods connects ecological evolutionary demographers with practitioners of applied health and demography, showcasing and calling for more work in applied evolutionary demography (Gibson and Lawson, 2014, 2015) and for demographically relevant work in evolutionary medicine and public health (Nesse and Stearns, 2008; Wells and others, 2017). Work in this area has included an explicit focus on population change (Gibson, 2014), family structure and health (Lawson and Uggla, 2014), social disparities in health (Pepper and Nettle, 2017) and nutritional transition (Wells, 2014). A parallel focus on gender and female autonomy has also provided counterintuitive reasons for undesirable social behaviour, including domestic violence (Jones and Ferguson, 2009; Stieglitz and others, 2018), crime and social violence (Schacht and others, 2014; Schacht and Kramer, 2016), dowry harassment (Shenk. 2007), biased sex ratios (Shenk and others, 2014), sex-biased parental investment (Mattison and others 2016a) and the effects of adoption on mortality and investment in children (Mattison et al., 2015; Mattison et al., 2018; Perry, Daly, and Macfarlan, 2014; Prall and Scelza, 2017), and even female genital cutting (Howard and Gibson, 2017). Such insights suggest different targets for intervention by focusing on the evolutionary benefits of socially undesirable behaviours (see also Hill, 1993). Many policy-relevant ideas brought forward by ecological evolutionary demographers simply would not be identified without an evolutionary perspective; such ideas are especially crucial in areas of policy where problems persist, and new thinking is sorely needed. For example, Gibson and colleagues' work on how the installation of water taps affected women in a low-resource setting was informed by life history theory, which highlights how health and fertility are connected. They found an increase in fertility after the installation of this labour-saving technology, which would not have been predicted under standard public health models (Gibson 2014). Equally, EED should consider topics of core interest to mainstream demography, such as the end points of fertility transition and how best to support ageing populations. More generally, these methods and applied topics should open more integrated research, with the potential to reconnect work in ecological evolutionary demography with mainstream demography, as both increasingly emphasize health and improved forms of data collection and population monitoring.

Lastly, even a relatively lengthy overview of ecological evolutionary demography necessarily omits interesting work in areas that don't quite fall within the core of the field. Given the

breadth of work in life history theory and parental investment, ecological evolutionary demography provides theory for understanding patterns in many related areas, including the upstream regulators of fertility and spacing behaviour, such as marriage (e.g. Chagnon and others, 2017; Marlowe, 2000), conflicts of interest between the sexes (Leonetti and others, 2007; Moya and others, 2016) and downstream consequences of behaviour, such as social (Mattison and others, 2016b) and health inequality (Pepper and Nettle, 2014). While we have not dedicated the same attention to all of these and many more interesting areas of research, we hope that this review has demonstrated the importance of the ecological perspective to evolutionary demography and, conversely, the usefulness of demographic methods and practice to the ecological perspective. Integration of related methods and theory lies at the heart of the initial founding of the discipline of ecological evolutionary demography (Low et al., 1992). We reiterate here that such integration is critical for recognizing the causes and consequences of well-established demographic patterns, and for identifying new patterns and departures from established theories that may be in need of refinement. In other words, ecological evolutionary demography necessarily comprises threads of diverse disciplines. The task for future work is to interweave these for a fuller and more robust science of demography.

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7. Contextual Effects on Fertility and Mortality: Complementary Contributions from Demography and Evolutionary Life History Theory

Caroline Uggla

In this chapter, I explore the influence of the local ecology, also known as contextual or area effects, on two focal demographic outcomes: fertility and mortality. I start by outlining why ecological effects have been of interest to evolutionary scholars, provide a brief overview of life history theory as a theoretical framework, and examine the type of data that have been used to test predictions in traditional, small-scale populations. Key evolutionary concepts such as extrinsic mortality risk and phenotypic plasticity are explained. I then compare and contrast this perspective with how contextual effects have been tackled by non-evolutionary scholars within demography and related disciplines, drawing on studies mainly from high-income contexts based on broad population register data. In the final part of the chapter, I lay out some challenges for this research area, which include addressing selection biases, and attaining a greater understanding of underlying causal mechanisms. Future research is likely to be more fruitful if evolutionary and non-evolutionary lines of enquiry become increasingly integrated.

Introduction

It is widely acknowledged that reproduction and health are not determined by individual characteristics alone, but linked to the social and geographic context where people live (Diez Roux, 2001; Diez Roux and Mair, 2010; Pickett and Pearl, 2001). Health determinants associated with the local context are diverse, but evidence is clear that they are associated with marked differences in health outcomes and, ultimately, mortality. In a high-income country such as the UK, differences in life expectancy are vast; on average, a boy born in Kensington can expect to live until 83.4 years of age, whereas a boy born in Blackpool only has 74.7 years on average (ONS, 2015). The difference in these values is comparable to the life expectancies of boys born in Switzerland and Saudi Arabia, respectively (WHO, 2016). The discrepancy in the number of healthy or disability-free years in the UK is even greater — approximately two decades (ONS, 2015). These within-country differences are not unique to the UK, but exist in many regions around the globe. Moreover, fertility and age at first birth vary greatly depending on context (Balbo and others, 2013). In 2017, the total fertility rate (TFR), i.e. the number of children born per woman, ranged from 7.2 in Niger, to just above 1 in Singapore (World Bank, 2017). While both childbearing and

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length of life are clearly determined in part by genes, these patterns cannot be accounted for by genetic differences alone, but also result from changes to physiology and behaviour.

Contextual influences on behaviour and subsequent demographic outcomes can take many forms. Individuals grow up, live and die surrounded by others who impact their lives in various ways. In high-income contexts, the place where an individual lives and works may structurally define education and employment opportunities, social networks, options for health, behaviour and diet, health care access and the type and magnitude of environmental stressors he or she is exposed to in daily life, from pollution to the risk of being a victim of crime. Individuals who grow up in deprived neighbourhoods are less likely to complete secondary education, are more likely to engage in sexual risk-taking behaviour, have physical and mental health problems, have a less stable family formation and are more likely to be involved in, and arrested for, crime (for review, see Pepper and Nettle, 2017). Variation in ecological conditions and behaviours is great also in traditional populations and lower- or middle-income countries. Anthropologists studying small-scale societies have demonstrated notable differences between populations in terms of daily net caloric intake, energy expenditure, climatic stressors, disease pressures and mortality regimes. All these factors taken together, it is perhaps unsurprising that the context in which an individual lives can predict a multitude of behaviours that are linked to fertility and mortality.

Ecological, neighbourhood or area effects — what I, in this chapter, refer to as "contextual effects" — have long been integral to both demography and evolutionary sciences, even if they sometimes have been studied under different terminologies and frameworks. Until the mid-twentieth century, with few exceptions, all demographic research involved spatial areas (Voss, 2007). Data collection of national censuses based on small geographical areas were integral for early estimations of local fertility and mortality rates and for the foundation of demography as a discipline. Today, most studies concerned with contextual effects revolve around this question: are two individuals who reside in the same area more likely to be similar in terms of their reproductive behaviour and health outcomes, all else being equal, than any two individuals from that population chosen at random? If so, are these effects causal, and what is it about a given context that makes individuals act in a given way? Demographic approaches to contextual effects share similarities with other social sciences such as sociology, geography, epidemiology and public health, where neighbourhood factors have generated interest due to their purported role in shaping health inequalities. Within evolutionary life history theory, the question of how the local ecology is tied to behaviour is a central tenet that has been explored with data from small-scale populations, and, more recently, with register and survey data from high-income contexts.

Chapter Outline

In this chapter, I review how scholars working within the framework of evolutionary life history theory and demography have explored contextual effects on fertility and mortality. There is a vast number of studies on this topic, and this chapter is not intended to be a comprehensive review. Rather, my aim is to highlight the main motivations and methodologies of each discipline in relation to contextual effects. In particular, I describe the framework used in evolutionary sciences known as life history theory, and approaches used in demography to highlight areas where integration between the two has occurred or would be well-placed. The structure is as follows: part one introduces the topic and lays out the structure for the rest of the chapter; part two outlines the principles of evolutionary theory and the empirical research concerned

with fertility, and, more recently, health and risk-taking behaviours associated with mortality. These two sets of outcomes might deserve separate extended reviews, but both form part of this chapter because of the theoretical foundation that unify predictions for reproduction, health behaviours and mortality. The third part describes demographic approaches, first on fertility, and then on mortality and insights thereof. I end by discussing some focal methodological and theoretical challenges for the contextual effects literature and how they can be addressed.

Evolutionary Life History Theory

Evolutionary life history theory is a framework that seeks to understand the variation within and between species in the timing of life events in terms of differential energy allocations (Roff, 1992; Stearns, 1992). It posits that all individuals have limited amounts of energy and have to allocate this energy in a manner that maximizes reproductive fitness, i.e. the proportion of genes in future generations. Decisions about how to manage trade-offs between growth, body maintenance and reproduction should depend, among other things, on the conditions imposed by the ecological environment. An individual who lives in an environment where he or she can expect a long life should delay reproduction, and spend a longer time in the growth phase in order to lessen the risk of premature death and to better manage competition with peers for mates and resources. In many animal species, mortality risk declines when a larger body size is achieved (Clutton-Brock, 1991). This means that many organisms, including humans, face a trade-off between either growing for longer and having lower mortality risk for themselves and for their offspring, or commencing reproduction early and facing a higher mortality risk (Low and others, 2008; Allal and others, 2004; Stearns, 1992). Favouring the latter is known as adopting a faster life history strategy with a higher pace of important life events, such as faster growth, earlier sexual maturation and an earlier age at first birth.

Life history theory was developed in biology to understand variation in growth and reproduction in non-human species before anthropologists and human behavioural ecologists started to apply its principles to humans in the 1980s and 1990s. By then it had been demonstrated that life history variation in growth rates, maturation and reproduction between species could be explained by the mortality rates experienced by adult individuals of that species; a higher age-specific mortality rate was associated with faster life histories (Promislow and Harvey, 1990). Life history theorists, whether concerned with humans or non-human animals, take an optimality approach and assume that observed behaviour should be close to the optimal, as predicted by the costs and benefits imposed by the local environment and an individual's state (Gadgil and Bossert, 1970; Parker and Smith, 1990). Importantly, natural selection has favoured individuals who are able to respond flexibly to their environment. The term phenotypic plasticity describes the ability to alter physiology and behaviour depending on the ecological circumstances; fitness payoffs may be maximized by favouring behaviour *a* in one context, and favouring behaviour *b* in another.

It is worth emphasizing that the evolutionary perspective does not mean that all behaviour is fitness maximizing, nor is it assumed that the strategies that maximize fitness are part of a conscious process. But, for the population health sciences, it is an important insight that individuals might be willing to engage in behaviours that are harmful to their health, if such health costs are outweighed by fitness benefits incurred from such behaviours. However, for this to hold, an important assumption is that ecological conditions are at "equilibrium", i.e. stable, so that behaviour can be adjusted to the optimum. This has implications for understanding

behaviour in areas where, for example, mortality rates have risen or declined sharply with little time for the adjustment of behavioural strategies. By default, behaviour is selected for past environments, but the time lag between the environmental conditions and behaviour remains a contested topic within evolutionary anthropology (Smith, 2013).

Extrinsic Mortality Risk

Life history theory scholars working on humans have generally been concerned with testing whether the variation in the scheduling of life events, such as maturation or age at first reproduction, can be explained by the variation in extrinsic mortality risk that adult individuals are exposed to in their environment (Nettle, 2011; Low, 2005). Extrinsic risks are risks that are not linked to mating or parenting and should apply equally to all individuals within a population (Charnov, 1993). Intrinsic risks, on the other hand, are risks that an individual can mitigate through behaviour, for example by the degree of risk-taking behaviour he or she exercises. The extrinsic/intrinsic distinction is a continuum rather than a clear-cut difference, and how to best operationalize extrinsic mortality risk in a given population is a difficult question that has only recently been addressed in more detail (see further below).

Extrinsic risks exist in many domains, but it is extrinsic mortality risk that has been invoked most frequently in life history models. Mortality clearly curtails the time available for siring and raising offspring, and so individuals who are able to respond to such mortality pressures should fare better than those who are not. Mortality rates (or life expectancies) have the additional benefit that they can be calculated for all groups where basic demographic (life table) data are available, and can be compared across populations and even across species. Notably, comparisons of mortality schedules have offered insights into life history differences between our close primate relatives and us; humans have considerably lower mortality rates than chimpanzees, which might explain why humans have both an extended childhood period and slower life histories once maturity is reached (Hill and others, 2001).

Patterns between mortality risk and life history traits have been studied between different human populations to explain the immense variation we exhibit as a species in terms of reproduction and other life-course scheduling. In a study of twenty-two small-scale societies, growth and maturity covaried with life expectancy at age 15 (which ranged considerably, from 27 to 50 years) in that a faster maturation and an earlier age at birth was observed where life expectancy was lower (Walker and others, 2006). High extrinsic mortality rate has also been proposed as an explanation for differences in physiology between human populations, e.g. the short adult stature of Pygmy populations might be a consequence of a growth cessation necessary to secure reproduction in the face of high mortality (Migliano and others, 2007).

Other studies of associations between ecological conditions and fertility have come from historic data and examinations of variation between parishes, or over time with varying crop failures and famines. Historical studies of this kind tend not to estimate extrinsic mortality risk directly, but use food scarcity as a measure of the environmental quality, or examine conditions prior to or at birth as linked to subsequent reproductive success (for review, see Lummaa, 2003). Analyses based on Finnish church records have found that children born during years of low crop yield have a lower likelihood of marriage and marry later than children born during years of higher food availability (Rickard and others, 2010).

Life History Variation in High-Income Contexts and Cross-Culturally

In this section I review studies that examine both fertility and mortality outcomes and behaviours that come from high-income contexts and cross-country work. One of the first studies exploring the associations between mortality risk and reproduction in a high-income context drew on data from neighbourhoods in Chicago. This study showed that when life expectancy in the neighbourhood was lower, birth rates at younger ages were higher (Wilson and Daly, 1997). The study also reported that risk-taking in terms of homicides were higher in these neighbourhoods where life was comparatively shorter (excluding deaths from homicides). The same relationship between lower life expectancy and earlier age at first birth has been found across countries (Nettle, 2011; Low and others, 2008) and within countries over time (Quinlan, 2010).

While these earlier studies were ground-breaking and tested a central and intuitive idea, some of the early results had some methodological weaknesses, such as the use of aggregated data with relatively few data points. These can be replicated and improved upon now that demographic data at the individual level have increasingly become available. Total life expectancy (or life expectancy excluding homicide deaths as in the case of Wilson and Daly, 1997) is a rather crude measure of extrinsic mortality rate, because, in high-income populations, causes of death beyond individual control come from many sources and vary between areas and socioeconomic groups (Pampel and others, 2010; Uggla and Mace, 2015). Furthermore, when both the independent and the dependent variables are aggregated, this invokes the ecological fallacy, i.e. inferring individual-level behaviour from group-level data (Robinson 1950). In other words, an observed pattern between life expectancy and fertility rates, whether within or between countries, might be due to some unrelated factor at the neighbourhood level or country level. Cross-country comparisons are problematic both because comparability of data across contexts might be questionable, and because an independent variable measured at country level is unlikely to reflect the variation within countries to which individuals are exposed (Pollet and others, 2014). There is often variation between studies in how spatial units are defined, and within studies, both the size and population densities of administrative areas may vary greatly. This naturally has implications for how results can be interpreted, both in terms of individual studies and when trying to summarize findings from the literature overall. Towards the end of the chapter, I return to these issues and discuss some methodologies that address them.

With broad cross-country data increasingly available, it is tempting to test relationships between life expectancy and various indicators of life history variation that may be comprised in aggregate data published by international organizations such as the United Nations, or the World Health Organization. However, evolutionary scholars have to think carefully about when the assumptions of their models do not hold. For instance, when new epidemics arise (as with HIV/AIDS), individuals may not have the resources, nor the correct information needed to act in a manner that would maximize their fitness. Furthermore, the assumption that ecological conditions are at equilibrium may be violated in contexts where there has been rapid development so that life expectancy has increased or is fluctuating. Rapid change in mortality rates might be a reason why extrinsic mortality risk is a rather poor predictor of behaviour in many developing contexts (Anderson, 2010). It has been suggested that life expectancy only predicts reproduction in contexts where life expectancy is 65 or over (Low and others, 2008) though there is not yet enough work on this topic for a conclusive verdict.

The studies described above aim to explain variation in demographic events as a response to variation in the extrinsic mortality risk of the environment. As such, they generally examine the association between ecological conditions and outcomes that are relatively easily captured, such as age at first birth or fertility, on which data are commonly collected and available. However, because natural selection can only act on behavioural strategies that individuals hold, a key interest in the evolutionary life history literature is the *behaviour* of individuals, rather than the outcomes such behaviour might result in. It is also crucial that a range of demographic behaviours should be correlated with individuals with a given life history strategy; for example, individuals who reproduce early should, on average, be more likely to favour risk-taking behaviours that might increase their risk of premature mortality. Engaging in high-risk behaviours and discounting the future may be adaptive if any long-term benefits are less likely to be reaped. More recent efforts have therefore sought to examine variation in risk-taking and health behaviours of individuals. Such studies have been facilitated by other forms of data than those traditionally used by evolutionary life history scholars, namely high-resolution demographic census and register data. In the following paragraphs I outline some of this research.

Life History Theory, Extrinsic Mortality Risk and Beyond

A new strand of life history research is drawing on data sources more traditionally used within demography and non-evolutionary social sciences to map variation in individual strategies with individual level data. These studies form part of a general trend that the number of studies in evolutionary behavioural ecology using data from high-income countries has increased over the past years, and it has become more common to use register, census or survey data to test evolutionary predictions (Nettle and others, 2013). The implications for life history theory studies are several. Importantly, a broader range of life history outcomes have been explored. Health behaviours can be seen as part of a life history trade-off; although not a new idea in itself (Hill, 1993), recent work has incorporated health behaviour into the life history framework in various ways (Brown and Sear, 2017; Virgo and Sear, 2016; Uggla and Mace, 2015; Pepper and Nettle, 2014; Nettle, 2010). Moreover, the increased access to detailed national datasets has allowed testing more fine-grained predictions and acknowledging that other extrinsic risks than mortality may also matter.

Exploring Life Histories with Census Data: The Case of Northern Ireland

Detailed data on causes of death from mortality registers can be used to take seriously the question of how to operationalize extrinsic mortality risk in a given population. Ruth Mace and I set out to test whether extrinsic mortality rate at the local level was associated with age at first birth and death from risk-taking behaviours or behaviours harmful to one's health (Uggla and Mace, 2016a, 2015). We made use of census data from Northern Ireland, where the whole population is included in a longitudinal mortality study linked to the census. To construct a measure of extrinsic mortality rate, a definition from population health sciences was applied. It classified all possible causes of death into those that are preventable and those that are not, based on International Classifications of Diseases (ICD): classifications of death recorded by the medical doctor (Page and others, 2006). ICD codes are very detailed, and so distinctions can be made based on how likely it is that the individual's death was linked to their own risk-taking or health behaviour. For instance, preventable deaths comprise cancers where the role of individual behaviour is deemed to have an impact on disease aetiology (e.g. lung cancer from smoking), and traffic accidents that are due to the driver's own

behaviour (e.g. involve a stationary object rather than another vehicle). A range of causes of death beyond individual control, e.g. genetically determined illnesses, are deemed unpreventable. While a perfect measure of what causes of death are within or beyond individual control is probably impossible to operationalize, this distinction captures all causes of death and whether they were deemed preventable by health care professionals. Extrinsic mortality rates on local ward level were then calculated based on deaths deemed unpreventable and showed large variation between areas.

Using this extrinsic mortality rate as an independent variable, the analysis suggested that mortality risk beyond individual control was positively associated with both reproduction and intrinsic deaths from risk-taking behaviours, but that patterns varied for different individuals. Men living in areas with higher extrinsic mortality rate, i.e. more likely to die from causes *beyond their own control*, had higher risk of death from risk-taking or poor health behaviour, than men who lived in areas with lower extrinsic mortality rate (Uggla and Mace, 2015). However, the same was not true for women. Moreover, the data suggested that the association between extrinsic mortality rate and risk-taking behaviours was greater among men with lower socioeconomic status (SES) than peers with higher SES. Heterogeneity in contextual effects has not received much attention within evolutionary sciences (probably due to a lack of data,) but evidence of this nature is common in demography and population health, albeit rarely with the intrinsic/extrinsic distinction.

Crime, Morbidity and Adult Sex Ratios

Another advantage of having access to detailed census or register data is that other potentially important factors can be explored alongside extrinsic mortality rate to compare their relative effect. Our Northern Ireland studies also explored whether other area factors such as the adult sex ratio (an indication of number of available partners) and the level of crime in the local wards were correlated to individual reproduction and risk-taking behaviour (Uggla and Mace, 2016a, 2015). Interestingly, among men, the same two area effects — extrinsic mortality rate and crime rate — predicted both early childbearing and death from risk-taking behaviours. Among women, the data suggested that earlier childbearing was, in addition to extrinsic mortality rate and crime, associated with a female-biased adult sex ratio (i.e. accelerated in areas where mates were scarce).

Local morbidity rate is another potentially important factor for life history variation. In societies where modern health care facilitates a longer life, healthy life span might matter more than absolute life span. Furthermore, it might be easier to perceive whether people in one's surroundings are healthy (and at what age health imposes constraints on lifestyle) than to perceive extrinsic mortality risks. Some recent evidence underpins the association between morbidity and reproductive behaviour; higher morbidity at the ward level has been linked to a lower abortion rate for women under the age of 25 years, but a higher abortion rate at older ages in the UK (Virgo and Sear, 2016). Virgo and Sear (2016) argue that morbidity might be a more salient cue to reproductive decision-making than mortality in their high-income population, offering a rare comparison between different area-level factors.

Summary

Contextual effects are central to evolutionary life history theory, which has sought to explain variation in important life events such as age at first birth within and between populations. In particular, extrinsic mortality risk is a population level variable that has been at the core of evolutionary life history models. Life history theory emerged from observations based on animal

species, and evolutionary anthropologists were initially concerned with testing hypotheses in small-scale societies. Recently, with the increasing availability of high-quality survey, census and register data (mostly) from high-income contexts, there has been a shift towards data that allow us to test life history predictions in more detail. Simultaneously, the field has broadened its focus from reproduction (e.g. age at first birth), to include other outcomes such as abortion, breast-feeding practices and health-seeking behaviours. Two important insights from life history theory that can be of value in non-evolutionary social sciences are (i) individuals may behave in ways that are harmful to health and wellbeing in order to maximize fitness, and (ii) context should shape behavioural strategies so that an individual's outcomes fall on a trajectory (i.e. some correlation between different demographic behaviours).

Summary of the evolutionary life history framework for understanding variation in reproduction and mortality:

- Primary interest lies in understanding how natural selection has shaped human behaviour, rather than predicting trends in fertility or mortality within a specific population.
- Human behaviour has been shaped by natural selection to be highly flexible, and this so-called phenotypic plasticity is not due to genetic differences between groups.
- Early reproduction and risk-taking behaviours, while in some cases harmful to health and wellbeing, may be seen as adaptive responses to an individual's environment.
- Childbearing behaviours and risk behaviours are likely to be correlated within individuals to some extent.
- The use of demographic and health data has enabled the testing of fine-grained predictions, and the operationalizing of extrinsic mortality risk in a broad range of populations.

Contextual Effects in Demography

In this section I provide an overview of contextual studies on fertility and mortality in demography, comparing and contrasting them with the evolutionary approach to the same questions.

Overview

In contrast to evolutionary scholars concerned with generating and testing hypotheses based on evolutionary theory, demography is a discipline with relatively little or no overarching theory (Tabutin, 2007). This depends, of course, on the definition of theory, and to what extent the bringing in of theories from neighbouring disciplines (such as sociology and economics) is considered "demographic". Demography grew out of analyses of national registers and censuses, and is naturally closely entwined with social policy and advocacy. It has even been called a "wild science", due to its origins in data collection and government organizations, as opposed to academia (Petit, 2013). While there is clearly merit in descriptive demographic research, some demographers have argued that the discipline would benefit from integration of a broader range of theories (e.g. Sigle, 2016).

Given the difference in their origins, it is not surprising that demography and evolutionary sciences have differed in their foci and in the populations most often studied. In Table 1, I lay out some

of the typical key interests, similarities and differences between them. Where evolutionary studies traditionally drew on small-scale populations and data collected by anthropologists, demographers interested in contextual effects often use data from both low- and high-income countries to compare different regions or residential areas. National registers and survey data, which form the basis of much demographic work, have many strengths. They often stretch over long periods of time, have multiple outcomes on the same individual, can track domestic migration, and be tied to changes in policies and society overall. This is valuable considering that one role demographers fulfil is to help nations understand and make projections about their populations and the wellbeing of their people, but is equally useful when testing predictions about human behaviour.

Table 1. Comparison of key features and interests of evolutionary life history theory and demography for contextual effects on fertility and mortality.

	Evolutionary life history theory	**Demography**
Definition	The study of human behaviour within and between societies as understood from an evolutionary framework based on natural selection	The study of populations and the cornerstones of population change: fertility, mortality and migration
Key aims	To generate and test hypotheses to develop understanding of human behaviour and evolutionary theory	To describe and forecast population patterns of fertility, mortality and migration, and improve public health and reduce inequalities
Populations of interest	Typically traditional, small-scale societies, with recent increased interest in high-income contexts	Anywhere data are available, but predominantly populations with censuses, large surveys or registers
Theory	Life history theory (and others) from evolutionary biology	Less reliant on theory, or informed by theory from the social sciences e.g. sociology
Data	Small-scale household surveys conducted by anthropologists, historical records, e.g. parish records, and more recently population registers and census data	Register, census or survey data, sometimes linked to health registers; rarely collected by those who analyze the data
Age groups of interest	Mainly (but not limited to) individuals of reproductive age, or behaviours that can be traced to that age group	Either the whole population, or specific sub-groups that can be defined for interventions, e.g. “the oldest old”
Outcomes of interest	Age at first birth, total fertility, risk-taking behaviours and intrinsic mortality. Multiple demographic behaviours are expected to fall on a continuum of a life history strategy	Measures of fertility, life expectancy, all-cause mortality or specific causes of death, often studied separately, migration
Focal area/ contextual predictors	*Extrinsic* mortality (often proxied using measures of deprivation) and more recently other indicators e.g. morbidity rate	Neighbourhood poverty, deprivation indices

Contextual Effects on Fertility

Much demographic research on tempo and quantum of fertility (when people have children and how many they have) has explored individual determinants, such as women's labour market participation and education (Hoem and others, 2006), rates of cohabitation, marriage and separation (Kohler and others, 2002) and the impact of various family policies (Neyer and Andersson, 2008). However, it is widely recognized that to predict an individual's or couple's childbearing behaviour, considering the context in which people live is important. In early demographic studies that sought to explain the first demographic transition, spatial analyses were integral as fertility decline was propelled by urban dwellers before rural family sizes followed suit (Coale and Watkins, 1986). The decisions of whether or not to have a child, when to start, how to space children and when to stop all impact fertility rates, and are complex decisions with multiple determinants governed by the norms, institutions and policies at country or regional level (for review see Balbo and others, 2013).

Many insights into, between and within country variations in fertility come from studies on the first demographic transition. One example of the latter is Belgium, a small country geographically, but diverse in terms of religion, culture, language and development: it demonstrated variation between two neighbouring provinces that could be comparable to a lag of fifty years of fertility decline (Lesthaeghe, 1977). From the late nineteenth century until 1960, fertility variation at the national level increased in Western Europe, and at the subnational level it decreased with increasing homogenization within national states (Watkins, 1990). Then in the 1960s and 1970s, Europe underwent a second demographic transition, where a gender revolution and a resurgence in female participation in the labour force that was incompatible with childcare led to the postponement of first births and an overall decrease in fertility (Lesthaeghe, 2010). Nevertheless, substantial variation in fertility has persisted between European countries and regions (Billari and Kohler, 2004).

In the debate on whether country differences in fertility will persist or whether convergence is likely to continue (Frejka and Calot, 2001; Wilson 2001), some have argued that it is surprising that there is not a greater body of work on *within*-country fertility variation (Kulu and Boyle, 2009), and the local residential context. Much of regional fertility variation has been analysed at the national or sub-national level perhaps because variables of interest, such as labour market conditions and level of economic development (Fox and others, 2018), or the impact of family policies on fertility are easily operationalized on the national or sub-national level, but make less sense at the neighbourhood level. Moreover, it is possible that the objective to avoid very low fertility *at the population level* contributes. That is to say, from a policy standpoint, very low levels of fertility are problematic at a national level, but less alarming in smaller areas of resolution, both because of how nation states are organized, and because people move between areas.

The recent surge in spatial analyses of fertility was long overdue and may be related to more refined measures and methods available from geo-coded data and multilevel models (Voss, 2007; Matthews and Parker, 2013). Echoing the early studies on urban/rural differentials, one strand of current within-country research has posed the question of whether fertility differs according to residence type. On balance, these studies tend to show that there is higher fertility in rural and suburban areas than in urban areas, which persists even when the SES composition of such areas is controlled (Kulu and Boyle, 2009). However, because some areas are seen as more suitable for bringing up a child, the "migrant selection" effect is a likely confounder (Courgeau, 1989). It is also vital that studies comparing different geographical areas (e.g. regions, census tracts, wards or some other spatial unit) separate the contextual from the compositional, the latter referring to the fact that

fertility rates of a given region may be due to the composition of individuals who live there, rather some other property of the area. Evidence that fulfils these criteria is mixed; some studies suggest that context does not matter for fertility once differences at the individual level are accounted for (Hank, 2002), while others demonstrate evidence of clear regional differences that are robust even after adjustment for individual characteristics such as women's employment and civil status are made (Kertzer and others, 2009). There might also be differences with the type of outcome examined. For example, in the UK the influence of the residential area level has been found to be relevant for the transition to first birth, but second- and third-birth progressions are correlated to the characteristics of the couple, and not the area where they reside (Fiori and others, 2014).

Some contextual studies examine fertility behaviours of individuals with different language identities, or who have a different ancestry or ethnic background. Recently revisiting the subject of Belgian fertility variation, Klüsener and colleagues (2013) have demonstrated that individuals living in the German-speaking regions of Belgium, which bear the influence of German family norms but enjoy Belgian family policies, have fertility profiles that are more similar to the Belgian than the German. The authors argue that institutional context is more influential than cultural norms, although how these might be fully disentangled remains a sticky point. Another perspective on local influence comes from studies of the fertility of immigrants and their descendants, where residential segregation is used as a proxy for exposure to norms of the destination country (Kulu and González-Ferrer, 2014). For instance, child migrants to England and Wales who grow up in areas with lower levels of residential segregation have fertility levels that are more similar to those of natives, as compared with peers who grow up in more segregated areas. This points to the fact that residential context during childhood is important for future fertility behaviour (Wilson and Kuha, 2018).

Further efforts to focus on the role of context include research that goes beyond the arbitrary residential areas and investigates the influence of nearby neighbours and colleagues on fertility behaviour. Recent evidence from Norway suggests that neighbours influence couples' transition to a third birth, even after couple confounders were adjusted for (Bergsvik and others, 2016). The results held when varying the area sizes between the five hundred and the twelve nearest neighbours. In the same vein, but examining the social influence of colleagues in the workplace, data from Germany suggest that women are influenced by female colleagues; odds of progression to first birth doubled the year after a peer gives birth, after which the odds decreased and were diminished after two years (Pink and others, 2014). A recognition that meso-level factors (such as social network and family-level factors) are important, sandwiched in between micro-level (individual) and macro-level factors (institutions and norms), is growing, and provides a link between small areas and the larger contexts in which they are embedded (Balbo and others, 2013).

Teenage Childbearing and Neighbourhood Deprivation

Teenage childbearing is one aspect of fertility that has been studied extensively from a contextual perspective; these studies show that neighbourhood deprivation is associated with earlier onset of childbearing (Harding, 2003; Imamura and others, 2007). Teenage childbearing has been seen as an undesirable behaviour by policy makers, often linked to adverse birth outcomes and sexual risk-taking and, as such, subject to many policy interventions (Dickins and others, 2012; Allen and others, 2007). Nevertheless, neighbourhood deprivation studies have not generated conclusive evidence on what it is about deprivation that is associated with earlier

childbearing. Some evidence suggests that the risk of teenage pregnancy is higher if a high-poverty neighbourhood is adjacent to a more prosperous neighbourhood, hinting at an effect of inequality (see also Gold and others, 2004; Wilkinson and Pickett, 2007). Contextual effects on early fatherhood have not received much attention, echoing the general trend of more emphasis on female than male fertility. Where female and male childbearing at young ages have been compared, there is some indication that different contextual effects matter for early motherhood and early fatherhood (Uggla and Mace, 2016a), however this area remains underexplored.

Covariation in Fertility and Mortality

While demographic work has established that fertility and mortality often change in concert, demographers have primarily been concerned with trying to describe shifts in fertility and mortality at the population level, rather than examining variation at the local and individual level. Even less work has attempted to ascertain the effect that population mortality rates have on individual reproductive decision-making. In contrast, the opposite relationship, i.e. the impact of an individual's childbearing on longevity, has generated interest in both demography- and evolutionary-informed work (Chereji and others, 2013; Doblhammer and Oeppen, 2003). That is not to say that the idea that mortality rates can impact fertility is a foreign one in demography. Conversely, it is well known that a decline in child mortality on the population level, together with economic development, predated the decline in fertility of the first demographic transition (Kirk, 1996). To consider children as part of an economic quality-quantity trade-off, where child quality is favoured over quantity when mortality rates decline (Becker, 1981), is an example of how economic theory has been integrated into life history models, and is central to the evolutionary study of human fertility (Hill and Kaplan, 1999; Kaplan and others, 2002).

Work on so called "mortality shocks" and their impact on fertility are analogous to evolutionary work outlined in previous sections of this chapter. For example, Nobles and colleagues used data from regions affected by the 2004 Indian Ocean tsunami, whose impact was considered to hit communities randomly, to study the impact of mortality rates on fertility (Nobles and others, 2015). They found evidence of replacement fertility (women who lost children were more likely to have another birth) and that women without children before the tsunami commenced childbearing earlier in regions affected by the tsunami (Nobles and others, 2015).

While evidence linking local mortality risk and reproductive behaviour remains rare, other factors such as economic insecurity and uncertainty have been scrutinized in detail by demographers. Economic downturns and increases in regional unemployment rates tend to be negatively associated with fertility (Sobotka and others 2011, for review). A recent study showed that, following the recent recession in 2008 in the US and Europe, both unemployment rates and overall uncertainty (measured as drop in consumer confidence and sovereign debt risk) were negatively associated with childbearing (Comolli, 2017). While the type of data and operationalizations of uncertainty differ, this type of work has much in common with evolutionary studies that have also explored the associations between uncertainty and reproductive behaviours (Nolin and Ziker, 2016; Davis and Werre, 2008). Thus, central research questions about the role of extrinsic mortality risk and uncertainty for reproduction exist in parallel, and greater cross-disciplinary integration would be beneficial.

Contextual Effects on Mortality

Recent years have seen an explosion of studies on how neighbourhood factors are associated with health and mortality. Several converging trends are likely to be responsible for this influx, including better statistical methods that allow isolating individual and area effects, developments within geo-referencing technologies, a renewed interest in health inequalities, and the idea that individual characteristics are insufficient to explain health outcomes (Diez Roux and Mair, 2010). As with fertility, the processes leading to mortality are complex. Mortality is determined by a combination of diet, physical activity, health behaviours, genetic predisposition, social support, access to health care and physical barriers such as pollutants or toxins that may vary between areas.

A key reason mortality risk is examined at the local level is because it is thought that many health interventions can be implemented at this level. However, with indications that the magnitude of the area effects are sometimes negligible, there has been debate about the usefulness of health policy implementations at the neighbourhood level (Lupton, 2003). While some studies map contextual effects on life span or all-cause mortality, others are conducted by medical experts who have an interest in a particular outcome, e.g. ischemic heart disease or suicides. When a particular disease or cause of death is of interest, it may be easier to hypothesize about the potential impact of the local context. However, rarely are different causes of mortality that could be considered under individual control categorized together. In part, this might be due to the fact that the types of death that are preventable varies over time and space (Page and others, 2006).

Most contextual studies examine the association between some form of aggregated SES measure or deprivation and mortality. These studies generally find that higher deprivation is linked to higher all-cause mortality risk after controlling for individual SES and other factors. An early example from the US reported that, after controlling for age, sex, race and health status, individuals in deprived areas had a 50% higher risk of death (Haan and others, 1987). This association is remarkably consistent across countries (in European and US datasets) (van Lenthe and others, 2005). A different set of studies examine variation or inequalities without having *a priori* predictions of why there is variation between areas. Area variances — and not just the means — are important in order to accurately understand differences between areas (Merlo and others, 2009) and provide an indication of the magnitude and change over time of health inequalities.

Heterogeneity of effects is central to understanding the plethora of results of contextual effects of mortality. There is evidence that the association between area SES and mortality is stronger for men, and for older individuals (Meijer and others, 2012). Winkleby and colleagues (2006) report that in the US, the benefits associated with residence in a more affluent area do not extend to men and women with a lower SES. Furthermore, the effect of the local area on mortality may vary depending on the individual's life stage. For instance, multiple waves of census data from Norway suggest that, for young individuals, only the most recent area of residence was linked to mortality from violence and mental health issues, whereas for older individuals, areas from previous stages of life had additional effects (Naess and others, 2008). However, because different outcomes might have different relationships with different neighbourhood characteristics, caution should be exercised when generalizing from one dependent variable (Roos and others, 2010).

Deprivation and Extrinsic Risks

Many insights gained from demographic and epidemiological studies on contextual (or neighbourhood) effects on mortality can be linked to evolutionary life history theory. Arguments related to deprivation and death are congruent with extrinsic mortality risk, because deprivation is in many cases associated with higher extrinsic mortality (Pampel and others, 2010; Uggla and Mace, 2015). Thus, the vast amount of evidence on associations between area SES and behaviours constitute a rich source that underpins arguments of extrinsic mortality risk for health and risk-behaviours. However, conclusions based directly on extrinsic mortality risk and its impact on particular health behaviours are almost non-existent. This may be because it appears circular that population mortality rate — even if it is extrinsic — at the area level would predict risk of death of individuals. One exception comes from work based on Demographic and Health Survey (DHS) data from fourteen African countries (Oster, 2012). Oster found that reductions in HIV risk-taking were higher where life expectancy (excluding HIV deaths) was higher, that is, where individuals' sexual risk-taking had greater impact on their life span. This is one plausible explanation for why behavioural response to HIV has been much slower in this context than the HIV response in some high-income contexts (Oster, 2012).

Proximate Determinants and Constraints

Demography and other data-driven disciplines are well positioned to measure proximate mortality determinants, such as healthcare access or availability of close kin, on mortality. The emphasis on proximate determinants of health and mortality outcomes is a good complement to the life history approach that has been less concerned with proximate causes. As a useful starting heuristic, evolutionary-minded scholars assume that individuals are able to make cost-benefit analyses about mortality risk because they are able to gain accurate information on the costs and benefits of their decisions (not necessarily consciously), and can respond to contextual factors largely without constraints (Borgerhoff Mulder and Schacht, 2012). This might not always be true and provides reason to think about how constraints to invest in health vary between different sub-groups. In the UK, individuals in more deprived areas report feeling less safe using green spaces for physical activity (Jones and others, 2009) and are exposed to more fast-food advertising than individuals in more affluent neighbourhoods (Adams and others, 2011). Such structural differences, coupled with physiological pathways, e.g. that women in deprived areas are more likely to have a blunted cortisol response, might make it difficult to favour day-to-day health choices that are beneficial in the long term over those that offer short-term gratification (Barrington and others, 2014).

Summary

Within demography, much emphasis is placed on describing variation in fertility patterns, though spatial differences are often examined on national or sub-national level, and few examples exist of how mortality risk influences fertility. Indirect measures such as urban versus rural residence, area deprivation and proxies for uncertainty are nevertheless insightful and often overlap with evolutionary perspectives on fertility variation. With regard to mortality, in recent years the number of studies on contextual effects on mortality has grown rapidly. Individuals in areas with high deprivation generally have higher excess mortality, even when individual characteristics have been controlled (or "accounted for"). However, there are notable

differences in how determinants are operationalized, and in heterogeneity based on, for example, sex and life-course stage. An increased understanding of the feedback loops between socioeconomic factors, health and reproduction, along with broad interest in proximate pathways, is promising for the aim to decrease inequalities in mortality.

Summary of contextual effects on fertility and mortality within demography:

- Motivated by understanding determinants of fertility and improving health and wellbeing of populations.
- Takes a "bottom-up" approach and is not always strongly theoretically motivated and/or draws on theories from other social science disciplines.
- Individuals in deprived areas have higher rates of teenage childbearing and higher mortality risk, but these topics are seldom studied in unison.
- Contextual effects on mortality are part of a burgeoning literature on health inequalities which has documented differences according to type of mortality and individual characteristics.
- Demographic studies on fertility and mortality are often characterized by an emphasis on methodological quality, including selection biases, and methodologies that attempt to isolate influences.

Challenges and Future Routes of Research

In this section, I discuss some challenges to research on contextual effects, including selection biases, how to define areas, and understanding the underlying mechanisms. I offer some suggestions for future research and stress the complementary insights of evolutionary theory and demography for these questions.

Selection Biases

Both studies rooted in demography and evolutionary life history research overwhelmingly rely on observational data. While experimental study designs have been used to test life history predictions in both humans and other species, when it comes to factors influencing actual behaviour rather than preferences for childbearing, the experimental method is neither feasible, nor ethical for human subjects. Physiological experiments, including priming methods, are an exception (for review, see McAllister and others, 2016). Inherent to observational data are issues of selection biases, which are problematic when trying to make inferences about the impact of an area on the behaviour of individuals who live there. Even with longitudinal register data, factors that might be associated with a propensity to move to a certain area often cannot be adjusted for.

One way to address issues of self-selection is through randomized controlled trials. These are commonly used in medicine and for public health interventions, but are more complicated when applied to questions related to contextual effects. A rare example of experimental data on this topic is the Moving to Opportunity (MTO) project, which was implemented in five large US cities in the 1990s to test the effect of areas on individuals (Leventhal and Dupéré, 2011). In this project, randomly selected participants in high-poverty neighbourhoods were offered vouchers and support to move to less impoverished areas. There have been many studies on the MTO project, one of which reported that young girls who moved to a less deprived area had fewer mental health

problems and more benefits in terms of education than non-movers (Leventhal and Dupéré, 2011). However, even if it is considered ethical, moving people to a new area is difficult, expensive and impractical and does not necessarily eliminate doubts about causation (Oakes, 2004). Thus, we are often left attempting to infer causality from observational data. Some have argued that the statistical issues such as endogeneity — the difficulty of defining appropriate geographical borders and extrapolation in multilevel analyses — mean that contextual effects are better investigated through qualitative approaches (Cummins and others, 2007). While they cannot alleviate the problem of selection biases, mixed-methods studies that draw on both qualitative and quantitative data could help to understand the experiences that produce behavioural variation between areas.

How Should Spatial Units Be Defined?

A common assumption of contextual studies is that the area where an individual lives functions as a cue to what life history strategy he or she should adopt, and is where childbearing intentions are formed. Neighbourhoods are often relatively small areas in which individuals may be familiar with the local conditions. But when areas are larger, e.g. census tracts or regions, the area might poorly capture what an individual encounters in his or her daily life. A study on area effects on all-cause mortality in Finland found an effect of residential area SES when the area was 250 x 250 meters, but this effect was attenuated, or completely absent when larger areas were used (Halonen and others, 2013). Another complication is that, in high-income countries, many individuals move between the home and the workplace and encounter multiple areas on a daily basis. Multilevel models with multiple memberships that allow simultaneous incorporation of family and work environments alongside wider contextual effects (Fielding and Goldstein, 2006), may go at least some way towards addressing this bias statistically. Where possible, areas based on a given number of nearest neighbours, applied, for example, in geography (Malmberg and Andersson, 2019), will help to fine-tune research designs so that they are congruent with the research question at hand.

Heterogeneity of Effects

From a population health perspective, heterogeneity in contextual effects is important to identify vulnerable groups, and to understand better the pathways to good health. From an evolutionary perspective, heterogeneity in contextual effects may help test detailed predictions for how costs and benefits of the local environment vary with age, sex or sociocultural context. For example, we may predict that the relationship between extrinsic mortality risk and health behaviour should be stronger among young individuals if local mortality rate has stronger fitness implications for younger than older individuals. However, the evolutionary perspective can generate predictions in different directions depending on the particular assumptions and the outcome in question. For this purpose, broad population data are required to compare different groups within populations, especially when the outcome of interest is premature mortality or other rare events. Anthropologists have often lacked data to test such effects, because, in small-scale societies, entire populations might yield sample sizes that are too small to test interactions. Thus, existing evidence from demography on contextual effect heterogeneity is useful, as it can both help to test the assumptions and to think clearly about the theoretical reasons for why effects may vary.

What Are the Underlying Mechanisms of Contextual Effects?

A major challenge for future research is to understand the pathways by which context is associated with variation in fertility and mortality. One development is the application of biomarkers that can capture how physiological characteristics differ between deprived and affluent neighbourhoods. Allostatic load, a measure of the "wear and tear" of the body has been linked to cumulative neighbourhood disadvantage (i.e. the longer the exposure to a deprived neighbourhood, the worse the condition) (Gustafsson and others, 2014). But even with such new knowledge of biomarkers, we may not be able to assert how various indicators are related to behaviours within the same areas.

Other types of data, e.g. from surveys, can help us to understand people's perceptions of local conditions, which might be as important as the observable local characteristics. James Gilbert, Ruth Mace and I tested individual perceptions in eight neighbourhoods of Belfast, Northern Ireland, and compared these to the census data of these neighbourhoods (Gilbert et al., 2016). The data suggested that while individuals had an accurate perception of neighbourhood age at death and morbidity, the discrepancies between actual and perceived levels of crime were high. Most individuals across the eight neighbourhoods reported high perceived personal safety, even though these neighbourhoods had been chosen to include both high and low ends of the crime rate distribution. One interpretation is that crime may affect only some individuals in an area and most individuals have reason to feel safe. There are clear parallels to the difficulty of perceiving local conditions accurately, which has been discussed with reference to mortality decline in low-income countries. It has been argued that perceiving mortality decline is difficult due to our tendency to acknowledge events, such as child deaths, more than non-events (child survival) (Montgomery, 2000). Future research will have to deal with the semantics, i.e. that individuals might respond in a manner that fits with the narrative of whether their area is "good" or "bad", and whether perceptions extracted verbally are meaningful.

Incorporating Life Course Factors into Contextual Effects

Both demographers and evolutionary scholars recognize the importance of the life course in shaping individual health (Ben-Shlomo and Kuh, 2002; Stulp and Sear, 2019), and it will likely continue to be integrated into new work on contextual effects. One question of interest is whether some periods during early development and childhood have more bearing on adult reproductive decision-making and health outcomes than do later periods in the life course. Whether developmental trajectories are mostly determined by the cumulative exposure to poverty, or whether certain periods, e.g. early childhood or adolescence, constitute a "critical window" is debated in both developmental biology and psychology, and within the health sciences (e.g. Braveman and Barclay, 2009; Murray and others, 2011). Another life-course question is to what extent individual reproductive, socioeconomic and health outcomes will correlate. Despite the strong theoretical motivation of life history scholars to predict multiple outcomes over the life course, they lag behind demographers who have long dealt with the issue of anticipatory analyses, i.e. that individuals may schedule life events (such as having a child) with the anticipation of other future events (such as completing higher education) (see e.g. Hoem and Kreyenfeld, 2006). This is a clear example of where theory and methodological insights from respective discipline could be successfully integrated.

Conclusion

Demographers and evolutionary life history scholars alike are interested in understanding why fertility and mortality vary between contexts. In so doing, scholars from these two fields have different aims, motivations and disciplinary origins that explain why their respective approaches to contextual effects differ — and where they overlap. While demography takes a bottom-up approach, driven by data, evolutionary sciences are top-down where data is a necessary means to answer questions that develop theory (Sear, 2015). Evolutionary anthropology has been characterized by the application of survey data from small-scale populations to understand fertility variation, rather than samples with a large number of data points that lend themselves to complex statistical techniques. The application of demographic data and methodology is now seen, for example, within anthropology, where studies have used a multi-level framework with Demographic and Health Survey data, or teamed up with local NGOs to collect rich data in different spatial areas (Lawson and others, 2015; Howard and Gibson, 2019; Uggla and Mace, 2016b). This development is relevant to area effects because it denotes a shift from the anthropological tradition of comparing populations in different contexts and with distinct cultural attributes, to comparing individuals residing in different settings while holding constant various characteristics that have been collected in a uniform manner.

Despite the differences between the disciplines, there are many shared elements and areas where integration is occurring or where continued interdisciplinary exchange seems promising. Demographers and evolutionary scholars interested in topics related to reproduction and mortality share the quest to understand the pathways that can lead to improved population health. In so doing, a better grasp of the role of life course factors, proximate physiological mechanisms and the role of structural constraints at the local level are key challenges. Identifying the determinants that link health, wellbeing and reproduction is a tall order and an endeavour that necessitates integration of different approaches and theories. The wealth of data from different populations is among the contributions of evolutionary theory to the understanding of human fertility, and it provides an extra layer of explanation that can unify existing frameworks (Sear, 2015). With the continued exchange of novel methodologies and the increased sharing of data from different contexts, further integration between these fields has great promise to enable us to better understand how the local context influences fertility and mortality.

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1 Note this chapter has been posted on the Open Science Framework website since 08/04/2020, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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SECTION 3: EVOLUTIONARY DEMOGRAPHY THROUGH TINBERGEN'S EYES

Nikolaas Tinbergen was a famous Dutch biologist who was recognized with a Nobel Prize in 1973 for his work on animal behaviour. As mentioned in the preamble to the section on evolutionary ecology and demography, many motivating factors for human evolutionary demography grew out of the biological study of animal behaviour: ethology. Tinbergen's research and writing have been very influential throughout areas of biology, but particularly for those who study behaviour. Tinbergen suggested that behaviour can be best understood within an evolutionary framework by considering four different levels of explanation. A behaviour wasn't truly "explained" until each of the four levels was understood. The four levels are:

1. Function – what is the locally adaptive function of the behaviour? This is often, but not always, understood as a cost-benefit analysis of the relationship between the observed behaviour and a possible proxy measure of reproductive fitness, such as the number of surviving offspring. For example, the number of eggs produced by a bird may be a behaviour of interest and the number of those hatched fledglings that go on to reproduce themselves used as the proxy measure of fitness: produce too many eggs and none of them live; produce too few and the strategy is replaced by those who produce more. As the name suggests, many explanations that we as social scientists think of broadly as "functionalist" would be at this level, but of course with the caveat that the "function" here is with respect to a measure of evolutionary fitness (see the chapters by Moorad, and by van Daalen and Caswell for discussions of fitness and its measurement). The power of the evolutionary approach is that the functional goal is implicit in the process of natural selection. Functional explanations in demography would be, hypothetically speaking, statements like: "in population X, mean age at first birth is five years later than it used to be, because those who wait five more years have lower child mortality rates due to their higher incomes, which ultimately leads to higher fitness."
2. Phylogeny – what is the history of a behaviour in the lineage of the species? In short, where did it come from and how did it change over long stretches of time? Did humans simply inherit this trait or behaviour from ancestral species, for whom it was adaptive in a different environment, and have retained it because it is not (very) harmful? Or is this a recently evolved trait that was actively selected for in the human lineage? Across species of human ancestors, we see a series of gradual changes from a tree-living ancestor to one whose spine and body proportions changed considerably, until we have the long-legged bipedal folks with large heads

who read about human evolutionary demography. The phylogenetic perspective would look at these characteristics and how they changed over long periods of time, typically across species. Demographers sometimes wonder why we in evolutionary demography are so interested in cross-species patterns, and this is part of the reason. Stages of the life course, such as childhood or the post-reproductive lifespan, are good examples of demographically-relevant traits that we examine from a phylogenetic perspective. This is why evolutionary demographers will try to understand the post-reproductive lifespan in humans by comparison with late-age fertility and longevity across primates, or why they sometimes compare with other species, like whales, that slow down or stop reproduction at later ages. Examining instances of "convergent evolution", the evolution of similar traits in phylogenetically distant species, such as post-reproductive life in humans and some whale species, helps provide evidence that particular selection pressures may have led to such traits. Interactions among social behaviour (including play, learning and teaching), mortality and the length of childhood are also frequently addressed, and for good reason, from a phylogenetic perspective.

3. Development – how did the behaviour form as the animal grew from an embryo to a new-born to an adult? Psychology has a large branch of research dedicated to how cognitive abilities change along with brain and body growth in children. The degree to which some behaviours are learned vs. "encoded" is a common theme at this level, as are questions about how the environment interacts with genetically influenced physiological traits. Demographers are well acquainted with how early-life effects can have late-life consequences on health or morbidity, which is akin to studying the late-life consequence of environmental interactions with developmental processes. Evolutionary researchers have paid more attention to the consequences of early life on the timing and rate of reproductive maturity, including age at first birth, though they have also highlighted the dearth of literature on how development influences reproductive outcomes in later life, such as the overall number of children born.
4. Mechanism – how does the animal's physiology make the behaviour possible? For instance, are hormones needed to trigger neuronal cues that increase its aggressiveness, slow its metabolism or cause it to engage in a dance to attract mates? Most of the work at this level of explanation is about physiology, neurons and the features of our anatomy that make the behaviour possible and/or allow it to occur. This includes the sensory mechanisms that allow organisms to track information from the environment to which they react. For social species, including ours, such work can also include understanding the social mechanisms through which behaviours spread. Many sociologists, demographers and anthropologists are also interested in mechanisms of social transmission and how these affect choices that map onto the timing and spacing of reproduction. In fact, as Colleran shows in a later chapter, the study of cultural transmission is ripe for bridging demography with evolution, as there are already many overlapping research questions and methods.

With Tinbergen's four questions we see how, in principle, integrating levels of explanation for an observed behaviour or demographic pattern can lead to complementary perspectives on, and a deeper understanding of where patterns come from, and how and why they change. The perspectives of individual researchers, or their respective disciplines, may be more focally aligned with some of these levels than others. For example, evolutionary demographers will often include consideration of functional explanations in their work, sometimes alongside mechanistic and/or developmental explanations. Social science, on the other hand, focuses on mechanistic or developmental explanations. This may inadvertently lead to the perception of conflicting viewpoints between those who are interested in evolutionary explanations of demographic patterns and those who are only interested in those stemming from sociological factors: the questions and explanations may seem different, and even mutually exclusive, because they are focused on different levels. But this "four questions" approach clearly illustrates the importance, and complementarity, of the kind of cross-disciplinary approach for which we advocate in this volume.

The following four chapters give a demographic case-study for each of these levels. Mace, who firmly established human behavioural ecology in the British academic landscape, sets the stage with a discussion of function, demonstrating how difficult it can be to disentangle ultimate (those relating to function and phylogeny) from proximate (mechanistic and developmental) causes for a behaviour, and outlining the methods needed to test hypotheses about function. This is a helpful chapter for those unsure about how evolutionary approaches fit into social science.

Jones et al., a team of evolutionary biologists who have made ground-breaking contributions on the evolutionary demographic study of lifespan and ageing, use a cross-species analysis to construct an "architecture" for the human life pattern, which helps identify which demographic patterns are divergent from other mammals and which are not, finding two key dimensions that distinguish humans in novel ways. Thus, the phylogenetic approach places human patterns in a broader context, provides scales for evaluation variance within and across species, and identifies characteristics of humans that are truly unique, rather than just assumed to be.

Sheppard and Coall, representing a collaboration with substantial experience in anthropology, behavioural ecology and physiology, and who, individually and together, have produced significant work on development and its consequences for later life outcomes, here provide a useful review of the many ways that variation in demographic behaviour stems from influences in early life, including environmental stresses that have consequences at later ages. This chapter covers ground more familiar in social science, but also illustrates the benefits of taking an evolutionary perspective on development, highlighting some areas where demography has not focused so much on developmental explanations.

Lastly, with a strong tie to demographic history, Vitzthum uses her considerable interdisciplinary expertise in anthropology and reproductive biology to provide an overview of the mechanisms of reproduction and how they influence broad fertility patterns. Given demography's place in the social sciences and the weakening of the link between physiology and reproductive output that happens during the demographic transition, demographers tend not to pay too much attention to reproductive biology when considering fertility outcomes. Vitzthum's chapter contains a wealth of insights into reproduction, however, and deserves to be widely read by those who work on fertility.

8. Why Do We Do What We Do? Analysing the Evolutionary Function of Reproductive Behaviour

Ruth Mace

In this chapter, I describe the reasoning behind dividing explanations for the evolution of behaviour into four different levels: two proximate explanations relating to mechanism and development; and two ultimate explanations relating to evolutionary history and function. I outline the basic methods we can use to test functional hypotheses about the evolution of behaviour. I note that in natural populations, we often find ourselves studying proximate mechanisms even if our central interest is evolutionary function. I conclude that a distinction between proximate and ultimate explanations for behaviour can be a useful heuristic tool in many situations, even if, in some real-world studies of human behaviour; this distinction is sometimes blurred.

Proximate and Ultimate Questions About Behaviour

How to ask questions about the evolution of behaviour is not quite as simple as it may appear. Social scientists and evolutionary biologists have been arguing about this for some time. Even evolutionary biologists only formulated a framework with which to have these kinds of discussions back in the 1960s, when Ernst Mayr (1961) made a crucial distinction between questions that relate to mechanisms and those that relate to evolutionary function. A couple of years later, Dutch ethologist Niko Tinbergen published a paper on his "four whys" (Tinbergen, 1963), which broke down the categorization of why animals (or humans) do what they do further. He identified mechanistic and development explanations as two "proximate" questions — the how questions. Evolutionary history and evolutionary function were what are usually described as "ultimate" explanations — the why questions. Evolutionary function is how a behaviour contributes to the Darwinian fitness of an organism; that is how it contributes to either or both of survival and reproduction. It involves identifying the fitness costs and benefits of behaviours, and is central to the interests of behavioural ecologists, including those studying human behavioural ecology (HBE). Tinbergen's insight helped everyone, in biology at least, to clarify what kind of evolutionary question they were asking. The understanding that these questions are not mutually exclusive obviates the need to argue about whether proximate or ultimate explanations are the correct ones.

So, for example, why does the baby cry? Because she is hungry (mechanistic); because crying is an innate behaviour that babies do not need to learn (developmental); because all primates have some kind of distress call (phylogenetic); because it makes her mother feed her more, so

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she is more likely to survive (evolutionary function); all are potentially correct answers. Actually, the last question on evolutionary function can be unpacked further. There are a number of more precise reasons as to why, in the sense of evolutionary function, a mother might respond to a crying baby by feeding or holding her infant more; these include the possibility that the infant is manipulating the mother, or that she is signalling her vigour to discourage her mother from infanticide (Lummaa, Vuorisalo et al., 1998). There is a surprisingly complex literature on the evolution of begging in chicks in a nest, because the theoretical underpinnings of parent-offspring and sibling conflict can all shape which patterns of offspring begging for food will be favoured by natural selection.

Although Darwin had considered functional explanations for behaviour in depth in his works, ethologists in the twentieth century largely ignored much of his theory of how adaptation arose by natural selection, preferring to focus mainly on proximate explanations for behaviour (many with a particular interest in the developmental questions of nature vs. nurture). However, this did not prove to be a particularly fruitful line of enquiry. It was only after Tinbergen's contributions in the sixties, and E. O. Wilson's textbook on sociobiology in 1972 (Wilson, 1980), and then Krebs and Davies' first textbook on behavioural ecology in 1976, that the fields of sociobiology, and then behavioural ecology were born (Laland and Brown, 2002). Behavioural ecologists have rapidly grown in number since that time, and the scientific agenda began to swing firmly towards trying to understand the evolutionary function of behaviour. Behavioural ecologists developed a strong tradition of developing functional hypotheses and testing them through detailed empirical studies of animals in their natural habitats. The application of this functional thinking to human behaviour and culture was appreciated almost from the beginning (Chagnon and Irons, 1979).

Human evolutionary demography is that part of human behavioural ecology that relates to demographic phenomena. Demographers interested in the "ultimate" evolutionary function of behaviour relating to timing and investment in reproduction, growth and survival are basically studying what evolutionary biologists call life history theory. This is a framework that formalises which combination of life history traits can maximize the lifetime reproductive success of an organism. What is the optimal speed to grow at? When is it optimal to start or stop reproducing? When to die? All of these are questions that are answerable, in theory, if we only knew enough about the environment in which an animal or human lives, and the impact of that environment on its chances of survival and reproduction. These life history traits are all influenced by natural selection in somewhat predictable ways (Roff, 1992). An especially important variable in nearly all these life history questions is the risk of extrinsic (unavoidable) mortality at each life stage. When mortality risks in the environment are high, selection favours a "fast" life history, in which it usually pays to mature as soon as possible, and give birth to a large number of offspring in the hope that some offspring get through and breed themselves before death takes them. Only when extrinsic mortality rates are lower can "slow" life histories, favouring a high level of investment in each of a small number of offspring over a long lifespan, win out, in fitness terms, over the more productive strategy.

Testing Hypotheses About the Ultimate Function of Behaviour

How do we test whether any particular functional hypothesis is correct? There are actually several tools from behavioural ecology at our disposal (Krebs and Davies, 1993). Firstly, of

course, the hypothesis has to be formalised. A verbal model may suffice, but usually evolutionary hypotheses can be modelled formally, to generate qualitative and quantitative predictions. The currency of such models is either Darwinian fitness, or some proxy measure that is thought to correlate with Darwinian fitness (perhaps calories obtained, or babies born that survived). It may also be necessary to include constraints (which may be trade-offs) within the model. In an ideal world, the model is formulated, tested against data, and, if found wanting, our hypothesis can be updated in the light of the empirical evidence.

The simplest method of testing an evolutionary hypothesis is inference from design. This is a weak method, but it is widely used in many areas of evolutionary biology and anthropology. If wings look like they are useful for flying they probably are, and it may not seem to be necessary to design an experiment to test that. But this can lead to "just-so stories" — named after Kipling's fantastical tales — stories that make a kind of sense but are not scientifically formulated, and for which there is no particular evidence! Such storied are untested, and of little use. Therefore, the more formal, quantitative models that can generate more precise and testable predictions are far preferable. There are three main mechanisms for testing these evolutionary hypotheses about function. These are: experimentation, comparative studies comparing individuals within populations and comparative studies across populations or across species.

Experiments

Experimental manipulations are what, ideally, we would like to do to understand evolutionary processes. However, experimentation that influences the reproductive success of real people is usually not possible in human populations, for obvious ethical reasons (plus humans have a very long generation time). We can seek natural experiments, such as political changes or ecological disasters that have happened to influence part of a population and not another part. For example, we compared one area of rural Ethiopia where water supply was improved and another where it was not, and found that the resulting decrease in the energy that women had to spend on collecting water had the effect of decreasing mortality, and increasing their fertility rate (Gibson and Mace, 2006). It also increased infant malnutrition, unexpectedly.

Another example that could be considered a "natural experiment" is the imposition of child policies in China that have forced individuals to severely restrict their fertility since the 1980s, and also the invention of prenatal gender testing, which led to dramatic changes in sex ratios in some parts of Asia (Hesketh and Xing, 2006). Such accidents of history were used to help us understand the nature of sex-biased parental investment, albeit not necessarily in an explicitly evolutionary context. A jump in sex-biased abortions in Asia showed us how behaviour can be apparently maladaptive in the evolutionary sense, at least in the short term; every baby has one mother and one father so a male bias in the sex ratio caused by female infanticide or prenatal sex-specific abortion will actually favour the Darwinian fitness of female children and damage the fitness payoffs from boys, as they became the sex less likely to find a mate in future years. The recent relaxation of the China's one-child policy also provides opportunities for understanding reproductive decision-making in evolutionary contexts (Liu, Duan et al., 2017). Such recent changes can only really tell us about proximate mechanisms in reproductive scheduling, although we may then use inference from those mechanisms to better understand the evolutionary underpinnings of the system.

Comparing Individuals Within Populations

Often, natural variation in a population can help us to infer the causes of fitness differences, which itself gives strong clues about function. For example, a range of predictions regarding the evolutionarily-informed predictions on the effects of sex ratio on divorce, parental investment and risk taking are now being tested on the basis of historical and natural variation in sex ratios across a range of populations where enough data is available (Grosjean and Brooks, 2017, Schacht, Kramer et al., 2017; Uggla and Mace 2017). Such tests are not as powerful as a real experiment (that might include randomisation and careful controls in populations where sex ratio was and was not manipulated, but would obviously not be possible). However, because such comparisons of naturally occurring variation between individuals and within populations directly concern reproductive success, they are getting close to directly testing functional hypotheses. Longitudinal demographic data is especially useful for this task. In the case of the evolution of menopause and post-reproductive life, longitudinal demographic data has been used to show that those children with grandmothers survive better (Sear, Steele et al., 2002), and that those individuals who survive to be old enough to become grandmothers have higher lifetime reproductive success than those that do not (Lahdenpera, Lummaa et al., 2004).

Of course, there are possible confounds, as correlation does not mean causation. It is possible that only high-quality mothers live long enough to become grandmothers (where "quality" is a technical term used in evolutionary biology as shorthand for features that promote survival or reproduction); therefore, whilst it appears that grandmothering enhances a woman's inclusive fitness, actually having a grandmother may not be causing grandchildren to survive; it may be simply that those women who survived into grandmotherhood were those who were the strongest mothers in the population throughout their earlier life and who gave birth to the most surviving offspring. A third variable (such a household wealth or immunity to disease) could explain both variation in offspring number and mother's longevity, resulting in what is known as a "phenotypic correlation". Such phenotypic correlations are the bane of those testing hypotheses about evolutionary function in natural populations. The evidence is more convincing that menopause, or at least post-reproductive lifespan, has to be about grandmothering when it is shown that only those grandmothers living near to their grandchildren had a positive effect (Engelhardt, Bergeron et al., 2019).

A more formal approach to examining variation between individuals within one population is to test to what extent observed phenomena fit the predictions of a mathematical model that optimises reproductive success. Sometimes a verbal model is not precise enough to isolate complex life history trade-offs, and a formal optimality model is required. Life history theory, as mentioned above, concerns how the timing of events such as birth and death, as well as growth, can maximise reproductive success. A model of an optimal life history ideally encompasses all the costs and benefits of certain actions across an entire lifetime, taking into account any constraints that may be relevant. A change in the risks or benefits at one point in the life cycle can alter optimal decisions at another point in the life cycle, making the optimal strategy hard to compute. It could be very specific to a particular environment, making generalisations difficult. Optimality models have been used to address issues of human family size, going back to Blurton-Jones's early analysis of birth intervals in !Kung hunter-gatherers (Blurton-Jones, 1986); this drew on insights from ornithologist David Lack's work on optimal clutch size in passerines (Lack, 1954). Blurton-Jones calculated that short interbirth intervals

increased infant mortality, but there was a trade-off, as very long birth intervals lower lifetime fertility. He calculated that the best trade-off between infant mortality and lifetime fertility (i.e. the decision that maximised lifetime reproductive success or LRS) was for !Kung mothers to have a baby about every four years (Blurton-Jones, 1986). This did represent the median birth interval observed by Nancy Howell's demographic data from this foraging population. Thus, he concluded that the ultimate explanation for !Kung long birth intervals is to optimise the fertility/infant mortality trade-off and thus maximise LRS.

He hypothesized that the proximate mechanism that drove birth spacing was the energetic costs suffered by mothers who had to carry two young infants at the same time (as well as any food they had gathered and wanted to take to camp), which he called the "back load model". The proximate explanations for these long birth intervals were that the energetic burden of carrying and breast-feeding any infants caused lactational amenorrhea, and that cultural taboos against resuming sexual activity for a couple of years after giving birth help to space births, and hence helped to reduce the mortality that would arise from shorter interbirth intervals. Interestingly, those !Kung who settled in cattle camps, giving up their nomadic lifestyle, did not suffer such high energetic costs of child care, as they could leave children in camp; so they had fewer constraints, and their birth intervals shortened. Whether this might in fact also be due to other factors such as increased food supply or a reduced burden of sexually transmitted infections is not entirely clear (Pennington, 1992).

One of the problems with a simple model predicting a single optimum, as in the birth interval example just given, is that real data displays an array of birth intervals. Why do some women consistently reproduce too fast or too slowly to optimise that predicted LRS? There are many important reasons — and deviation from a simple model can help us identify them. The process of science in general, and behavioural ecology in particular, works by proposing simple models and rejecting those aspects that fail to predict the data; then going back and improving the model to help us to understand the system better. Women may all have different optima based on their own individual costs of reproduction, as mentioned above; or, women may fail to reach their optimum due to constraints.

Optimality models do have to take constraints into account. Many of the debates in evolutionary demography concerning apparently maladaptive behaviour revolve around whether or not a behaviour is the result of hidden costs and benefits or simply constraints (cognitive, behavioural, physiological or time constraints). For example, some have argued that the span of primate female fertility (including in humans) is constrained to a maximum age of about fifty years, so no functional explanation for menopause is required (Kim, Coxworth et al., 2012). Others, including myself, disagree, but unfortunately the testing of hypotheses about constraints is rather hard. Resorting to the argument that evolution simply cannot solve certain problems can shut down the evolutionary debates, as it is hard to prove a negative. Failure to find a mate or infertility caused by an STI could constrain female fertility below the optimum in individual cases. But constraints seem unlikely to provide a satisfactory explanation for population-wide or species-wide phenomena like menopause, or the demographic transition to low fertility.

Lack of control over decision-making could also push fertility above the optimum, as might be the case in some societies where there is a conflict of interest between male and female optimal fertility, with males paying lower costs of high fertility than do females, and thus

favouring faster reproduction and perhaps not observing adequate post-partum sexual taboos. Females may have to bow to male optima if they do not have bargaining power.

This may apply in the Dogon in Mali, where rates of polygynous marriage and high fertility appear to be associated with high infant mortality and are not beneficial for female reproductive success (Strassmann, 2011).

Alternatively, the diversity may reflect phenotypic variation in female quality, or variation in other aspects of her state (such as whether she has help from allocarers). Simple optimality models effectively assume everyone is the same. However, some mothers have physiological or social advantages that will enable them to reproduce at a much higher rate than others without much increase in mortality risk. Each mother has her own optimum, depending on her state, with the result that most data on birth intervals reveals that those mothers with the highest birth rate usually have the highest reproductive success. This does not mean there are no trade-offs; it is simply that we may not see them because phenotypic correlations are also present. High-quality mothers are more successful than low-quality mothers. Experimental studies in birds, where the number of eggs in the nest can be manipulated experimentally, so that clutch sizes are random with respect to female quality (Gustafsson, 1994), do reveal these correlations. Humans obviously cannot be manipulated in the same way. Of course, chance events (in each individual history) can also generate variation — mortality, for example, is highly stochastic.

A more realistic and powerful framework for modelling optimal reproductive decision-making is stochastic dynamic programming, in which reproductive success over the lifetime is modelled, in a stochastic environment and where decisions are state-dependent (Houston, Clark et al., 1988). This is, of course, much more realistic. For example, birth rates depend on the characteristics (state) of both the woman herself and of the family (environment) that each woman finds herself in. Whether or not it is optimal to have another baby could depend on many features of the mother's current state, such as when her last birth was, did the child survive, the size of the family and how much food or other resources are available. Dynamic programming of such decisions has been used to model reproductive scheduling in the !Kung, enabling the risk of mortality for both mother and infant to be included, and the model generated realistic reproductive schedules (Anderies, 1996).

The same approach was used to see how wealth and the sex of the existing offspring, as well as mortality risks, influenced optimal reproductive scheduling in the Gabbra, who are camel pastoralists in Kenya. Here, most of the costs of setting up offspring in their own new families fell onto the parents of sons (Mace, 1998). Decisions to reproduce depended on wealth and the number of sons a family already had. The model showed that mortality risk had little influence on optimal family size but had a large influence on the number of births (as it is optimal to quickly "replace" births when the infant does not survive, but the higher mortality balances out the effect of the higher birth rate generating little effect on final family size). The model further showed that marriage costs (bride price) also had a big influence on optimal reproductive decisions, with smaller family sizes becoming optimal when marriage costs were high. This optimality framework shows how high costs of raising children co-evolve with low fertility.

Thomas et al. used this approach to investigate the relationships between sibling helping and sibling conflict, and their effect on birth intervals (Thomas, Shanley et al., 2015); they show that sibling competition and mortality risk interact, in that sibling competition only has a serious influence on optimal birth intervals when mortality is low. This captures one of

the essential features of the demographic transition to low fertility, which is that, as mortality has declined, parents appear to invest more in each offspring at the expense of high fertility (a trend that has occurred worldwide over the last 200 years). This model also showed that these factors alone, which only considered the two generations of parent and offspring, could not generate the evolution of menopause. The current evidence suggests that any model that predicts the evolution of menopause requires the inclusion of the grandparental generation and grandmother benefits in its evolutionary dynamics (Thouzeau and Raymond, 2017). This study examines the evolutionary dynamics of menopause, hence addressing both the selective forces that maintain it and those that drive its evolution in the first place, showing that the important selective pressures at these two levels of explanation can differ.

Comparison Across Different Populations

One important way to test hypotheses about how the risk of mortality shapes animal behaviour is comparison across species. This is widely used to test hypotheses about evolutionary history and function in biology. If species living in certain environments are more likely to do one thing than another, then a statistical case can be built for a functional association (in Tinbergen's sense of "why did natural selection favour that?"). For example, testes size is a measure of a mating system, as it is reveals how important sperm competition is. Sperm competition occurs when females mate with several males, and, in circumstances where males cannot prevent this by mate guarding, their best chance of being the one that fertilizes the egg lies in delivering a large amount of sperm with each mating. Chimpanzees (*Pan Troglodytes*) have huge testes by comparison with humans, telling us instantly that rates of female polyandrous mating (females mating with multiple males) are much higher in chimps than in humans (Harcourt, Harvey et al., 1981). In gorillas, however, where males tend to have little competition for access to females in their family groups, testes are small. Human testes are small compared to chimps, but slightly larger than gorillas, suggesting we are only a mildly polygamous species. This fits with the observed patterns of marriage norms cross-culturally, where some form of polygynous marriage is the most common marriage system, and even in human populations in which marriage is monogamous, serial monogamy is common (Murdock, 1967). In theory, were we to discover a new ape species previously unknown to science, we could immediately guess something about the mating system of this new species based on physiology alone.

Although there used to be quite a range of human-like sister species or ancestors, *Homo sapiens* (ourselves) is the only human species that remains extant. Life history does not fossilize well, so comparison across very closely related species is not really possible in the hominin line. However, we can do comparative studies across human cultures. Languages rarely hybridize and thus can act as badges of demarcation, broadly dividing the world into ethnolinguistic groups. Such groups often have different cultural norms with respect to customs such as mating system, marital and residence arrangements, the role of women, who cares for children and who works in the fields or contributes to other forms of subsistence, whether there is warfare within or between groups. These variables can all generate different demographic profiles.

We can borrow statistical tools from evolutionary biology to try to clarify the evolutionary processes that have given rise to this variation (Pagel, 1999; Mace, Holden et al., 2005). Phylogenetic comparative methods are powerful and accurate for trying to understand evolutionary processes. They can be used to infer ancestral states from data on extant cultures

or species (in this case cultures), if we have information with which to map the evolutionary relationship between these groups (cultures) onto phylogenetic trees. In cross-cultural studies, these phylogenetic trees map the historical relationships between ethnolinguistic groups through linguistic similarity, as words have the properties of a culturally transmitted replicator (Pagel, 2009). Because cultures usually mix to some extent through migration and intermarriage, the genetic history of neighbouring groups is not necessarily very tree-like. However, languages do not usually blend in the way that genetic material does. Children of mixed parentage with respect to language tend to learn the dominant language of their population, or both languages, but not a blend of the two. This has meant that languages retain signals that can be used to infer cultural historical relationships.

This not to say that words are never borrowed. Clearly many of them are, especially when the beliefs or technology that those words refer to are also borrowed; but on the whole the strong frequency-dependent nature of linguistic communications means that differences in core vocabulary can usually be very helpful for inferring phylogenetic trees of culture (Mace, 2005). Statistical methods can then use a combination of the phylogenetic tree, and the data on the extant cultures that we observe, to infer what cultural states may have looked like at various nodes on the tree that represent ancestral cultures (Pagel, 1999). Anthropology is generally confined to the present, but these methods for estimating ancestral states potentially enlighten us as to the likely patterns in prehistory that led the past to generate the diversity that we see in the present.

Phylogenetic comparative methods are just statistical approximations of course, and as with all statistics, and all science, the conclusions are only as good as the data and the assumptions on which they are based. However, they do provide a principled and repeatable scientific framework within which to ask questions such as: what were the ancestral states of certain cultural traits? What were the rates of evolutionary change in each direction? Does one cultural trait co-evolve with another cultural trait, or with a particular feature of the physical environment? What was the most likely order in which two traits evolved over time? These are especially important questions for understanding evolutionary function, because the co-evolution of two traits in cultures that have ancestral relationships is almost impossible to infer unless the phylogenetic relationships are known (Mace and Pagel, 1994). Uncertainty in phylogenetic trees, and in the path of evolution along the branches of those trees, can now be estimated using a range of Bayesian phylogenetic comparative methods (Huelsenbeck, Ronquist et al., 2001; Pagel, Meade et al., 2004; Pagel and Meade 2006). Hence, phylogenetics is very important with respect to both of the two "whys" that Tinbergen described as ultimate: function and evolutionary history. Ancient DNA is another technology that is generating a lot of tangentially relevant information on our recent (i.e. from the last 10,000 years) prehistory, and that will help to inform our knowledge of some (but of course not all) aspects of prehistoric populations and their movements, which will constrain and improve cultural phylogenetic models. The prospects of understanding our cultural evolutionary history are thus improving all the time.

One simple example of this approach, relating to human social organisation, is the evolution of matrilineal kinship (where names and property are inherited down the female line). Anthropologists had long noted a correlation between cattle keeping and patrilineal social organisation in Africa (Aberle, 1961). This example used a method borrowed from evolutionary

biology, designed to investigate the co-evolution of two discrete traits (Pagel, 1994) (in this case matriliny/patriliny and cattle/no cattle). There are examples of Bantu cultures in all four of the possible resulting combinations of presence/absence of cattle and presence/absence of matriliny. Phylogenetic methods, based on a language tree of the Bantu-speaking populations, were used to show the likely directions of such changes, and thus help test the hypothesis that the adoption of pastoralism generated patriliny (Holden and Mace, 2003). It is possible to test whether the state of one variable influences the rates of change in another (Pagel, 1994). Analyses showed that transitions between all of the four different states occurred and could go in all directions, but some transitions are more likely than others. In populations without cattle, transitions between matriliny and patriliny were quite common; but, once a population adopted cattle, a combination of cattle keeping and matriliny was very unstable, and transition rates out of that state were high. In contrast, pastoralism combined with patriliny is a much more stable and consistent state, with high rates of transition into it and low rates of transition out of it. Note that it was unlikely, however, that patrilineal groups without cattle then acquired cattle to become patrilineal groups with cattle; an evolutionary pathway via an intermediate matrilineal state appears to have been more common (at least in Africa, where the data for this study were from).

Phylogenetic comparative methods can also be applied to understanding the evolution of quantitative rather than discrete traits (although such methods are not quite as powerful with regard to understanding evolutionary history as they do not involve an explicit evolutionary model of the direction of change). Sex ratio is one example of a quantitative trait that has been examined in this way, using comparisons across Old World human cultures (Mace and Jordan, 2005). As language trees are only good at inferring relationships within language families, and less good at deeper historical relationships (as signal tends to be lost after about 10,000 years of time depth), a genetic tree was used in this case. Sex ratio at birth did not correlate with sex ratio in adulthood. Male-biased adult sex ratios (which may reflect higher female mortality) appear to be associated to some extent with bride price (that is a transfer of resources at marriage from the family of the groom to the family of the bride) across cultures. Hence in cultures where there is heightened competition between males for scarcer females, parents of girls are enabled to demand bride price. Other demographic traits that have been the subject of hypothesis testing, using various phylogenetic comparative methods, include correlations between social structure and the evolution of post-reproductive life across mammals (Nichols, Zecherle et al., 2016). Some models of the grandmother hypothesis for menopause argue that competition between generations of females for reproductive resources in communally breeding species (like humans) is more intense in patrilocal species, where females disperse at reproductive age and are therefore not related to each other (Johnstone and Cant, 2010). Other models argue that genomic conflict will result in worse menopause symptoms in matrilocal groups where females compete with their own relatives (Úbeda, Ohtsuki et al., 2014). However, there is no evidence from comparison across patrilocal and matrilocal cultures that either the timing of menopause or the severity of symptoms fits either of these models in human cultural groups (Snopkowski, Moya et al., 2014; Yang, Arnot et al., 2019). Yang et al. (2019) actually found worse menopause symptoms in patrilocal (female dispersing) groups, contrary to the predictions of the genomic conflict model.

Proximate and Ultimate Revisited?

The methods I have described for testing the function of aspects of human behaviour, be they reproductive scheduling or social organisation, are borrowed, perhaps with some modifications, from those methods used by behavioural ecologists and evolutionary biologists studying natural diversity in a range of species. If we are using cultural groups for comparison, that means that we are most likely studying the evolution of variation in cultural traits that are socially transmitted and inherited, rather than genetic traits; this is known as cultural evolution, or gene-culture evolution in some cases. (The definitions of cultural evolution are various, but one of the most common definitions of a cultural trait is just a trait that is transmitted socially; however, this is of little use in human studies as almost all traits fall into that category). There has been some debate as to whether those of us studying cultural evolution are studying proximate mechanisms or ultimate function. Those who have defined themselves as human behavioural ecologists, such as myself, tend to use the methods of behavioural ecology described above to study what we consider to be the functional basis of human behavioural and cultural diversity. We often seek patterns of natural or experimental variation that fit the predictions of an optimality model or other hypothesis, paying less attention to the proximate mechanism that drove that behaviour to develop. It is enough to concentrate on the phenotype and its distribution in nature or society. Diversity in human norms of behaviour is usually learnt, so much of it is usually best thought of as cultural in origin. In that sense, I do not consider studies in human behavioural ecology and in cultural evolution to be very different (Mace, 2014). The phylogenetic comparative study of matriliny and pastoralism just described, for example, can clearly be understood as a study in both cultural evolution and human behavioural ecology.

Studies of evolutionary dynamics, such as those considering how different selective forces or transmission probabilities of cultural traits lead to certain attributes emerging, may be hard to describe as proximate or ultimate explanations. Such models are similar to population genetic models, but focus on cultural rather than genetic traits. They may fit better into Tinbergen's more specific category of evolutionary historical explanation. For example, if populations adopt matrilocal residence and dispersal patterns when those around them are matrilocal because it enhances reproductive success, as we have shown that they do in southwestern China (Ji, Zheng et al., 2016), then I am happy to describe that as a functional explanation, or an evolutionary explanation. However, it is true that no gene frequencies are necessarily altered in this case; the decision by human females to disperse or not to disperse is almost certainly culturally inherited, so only the frequency of cultural traits change. Describing a study or model of mechanisms of transmission as revealing the ultimate function of cultural behaviour traits is not uncommon, as some like to describe transmission mechanisms as determining "cultural fitness". Some have argued that the proximate ultimate dichotomy is not helpful when thinking about certain aspects of cultural evolution in particular, including niche construction (where man-made environments feed back on the selective pressures imposed on future generations) (Laland, Sterelny et al., 2011). West and others have characterised this as an error, a confusion of Mayr's original distinction between proximate and ultimate causation (West, El Mouden et al., 2011). This is probably just a semantic debate, albeit one that has generated some heat. West et al.'s paper met with some hostility on one side, and the news that the Templeton Foundation was pouring a large amount of money into a grant to investigate the need for a new and "extended evolutionary synthesis" met with a somewhat incredulous reaction on social media

on the other side. These are surprising levels of hostility for fairly arcane matters of academic definition.

Definitions are only useful as tools to help understanding. Proximate determinants of reproductive rate are the usual fare of demographic papers, be they aimed at testing evolutionary hypotheses or not. If the proximate/ultimate distinction helps understanding in evolutionary demography, and if its blurring leads to more potential misunderstandings between biologists and social scientists, then these definitions need to be maintained when it is useful to do so. The study of cultural evolutionary processes is not belittled by such processes being described as proximate mechanisms. However, if the distinction is causing confusion with respect to cultural evolutionary models, which look at the dynamics of changes in frequencies of cultural traits in populations, then Tinbergen's full four questions may be needed to avoid confusion. Such models describe the likely evolutionary pathways that drive certain patterns to emerge (evolutionary history). Such models are undoubtedly evolutionary, even if the words "functional" or "ultimate" do not always readily describe these cultural processes.

Thinking about the evolutionary implications and the function of reproductive patterns of behaviour can help set research agendas. The role of grandmothers in childcare, for example, was a minority interest amongst only very few demographers. However, once Hawkes and others had stressed its potential role in the evolution of menopause (Hawkes, O'Connell et al., 1998), the number of studies of the effect of grandmothers and all other kin on child survival, adult reproduction and a range of other outcomes, like education and health, mushroomed, partly by bringing a new breed of researchers from other fields into the study of large demographic databases. Evolutionary function remains an intriguing research framework on which to hang our continued efforts to work out why people do what they do in all realms of our behaviour, including our demography.

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9. My Family and Other Animals: Human Demography Under a Comparative Cross-Species Lens

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Like all species, the demography of humans has been shaped under the framework of natural selection. Our understanding of human demography can thus be enhanced by viewing it through a comparative, cross-species, lens and exploring the position of humans among other animal species. Here we use demographic data in the form of matrix population models (MPMs) from humans and 90 other animal species to contextualize patterns of human evolutionary demography. We conduct an additional analysis using human MPM data derived from raw census data from 96 countries over a period spanning 1780 to 2014. For each MPM, we calculate a suite of demographic variables that describe multi-component life history strategy, and use principal component analysis (PCA) to contextualize human populations among the other vertebrates. We show that, across species, life history strategy can be described by position across two dominant axes of variation, and that human life history strategy is indeed set apart from that of other animals. We argue that life history architecture — the set of relationships among life history traits, including their correlations and trade-offs — is fundamentally different within humans than across all animal species, perhaps because of fundamental distinctions in the processes driving within-species and among-species differences. We illustrate strong general temporal trends in life history strategy in humans and highlight both striking commonalities and some differences among countries. For example, there is a general for traversal across life history space that reflects increased life expectancy and life span equality, but there is also among-country variation in the trajectories that remains to be explained. Our approach of distilling complex demographic strategies into principal component axes offers a useful tool for the exploration of human demography.

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Introduction

A literature search will reveal thousands of articles with titles containing "... *humans and animals* ...", loaded with the implication that humans are not animals, but that they rather belong to a very distinct group. But humans are certain animals: we may be exceptional in many ways, but perhaps not in terms of our demographic traits. Consider life span: the longest-lived human on record (Jean Calment, died aged 122 (Jeune and others, 2010)) was nowhere near as old as the longest-lived invertebrate, or vertebrate. Those records belong to the humble quahog (*Arctica islandica*), a bivalve, (>507 years; Butler and others, 2013) and Greenland shark (*Somniosus microcephalus*) (270 years; Nielsen and others, 2016) respectively. The reproductive output of humans is fairly modest: the highest total fertility rates (~11 children per woman) have been recorded for Hutterite populations of North America (Robinson, 1986), pales into insignificance compared to most non-human animal species, even if some approach our relatively low fertility rates (e.g., elephants, whales). Humans are admittedly unusual in the extended period of post-menopausal survival experienced by women, but there is evidence for this in some other species too (Cohen, 2004; Ellis and others, 2018).

Niko Tinbergen pioneered a multifaceted approach to the study of animal behaviour when he argued that behavioral questions could be addressed at both an ultimate and a proximate level, and in a dynamic or static context (Tinbergen, 1963). Although the answers to questions posed from these four perspectives will be different, they are entirely consistent with each other. In this chapter, we view the demographic behaviour of animals from the dynamic and ultimate perspective: the evolutionary lens. We aim to put human demography and their variable life histories (i.e., key demographic events in their lifecycle) in the broader evolutionary and comparative context by analyzing demographic patterns across the animal kingdom, and exploring the position of humans among other animal species in 'life history space'.

Like all other animals (and indeed species from the other Kingdoms), our characteristics have been shaped under the evolutionary framework of natural selection. Natural selection functions as an optimization algorithm: fitness is maximized under the long-term conditions experienced by the population (Cole, 1954; Gadgil and Bossert, 1970) and differences among species can, in theory, be explained by variation in the conditions in which they live, the conditions experienced in the past, and constraints of ancestry and genetic architecture. Thus, understanding the origins, the current context, and plausible future for human demography is enhanced by an explicit consideration of other animal species, and the relationships among them.

A great deal of variation exists among animals in traits such as somatic growth rate, age at maturity, life span, number and frequency of offspring produced, survival rates at particular ages or stages, and so on. These traits, collectively known as life history traits, describe the life cycle of an organism. These traits are the focus of the field of life history evolution (Stearns, 1992) and can be estimated using demographic methods (Caswell, 2001; Cochran and Ellner, 1992). Two of the most widely used in demography, both of which integrate schedules of survival and reproduction, are the life table (Chiang, 1984), and the matrix population model (MPM) (Caswell, 2001). MPMs have proved most popular among demographers studying non-human animal populations (Salguero-Gómez and others, 2016a).

The use of MPMs as descriptors of life cycles has a long and distinguished history. Their utility was first outlined by Leslie (1945) for age-structured models, and later extended by Lefkovitch (1965) to include stage-structured models, which can describe cycles based on size, ontogeny, and other phenotypic properties. Both types of MPM describe the dynamics of populations over a discrete time interval (often a year): $\mathbf{n}(t+1) = \mathbf{A}^*\mathbf{n}(t)$, where **n** is the population vector containing the number (or sometimes the relative frequencies) of individuals at each (st)age at time t or t+1, and where **A** is the MPM (also known as the population projection matrix), which describe rates of transition among (st)ages (i.e., probabilities of survival, ontogenetic development, and reproductive output). The major reason for the rapid adoption and widespread use of MPMs arises from their tractability and well-understood mathematical properties (Caswell, 2001; Caswell and others, 2018), and the fact that a large diversity of useful analytical outputs can be derived from them. These analytical outputs include metrics of population dynamics, including the rates of population growth, population structure, metrics for transient dynamics (Stott and others, 2011), the evaluation of the absolute (sensitivities) and relative (elasticities) importance of demographic processes and covariates onto population-level metrics (de Kroon and others, 2000) (among others), and a range of life history metrics including generation time, life expectancy, measures of entropy (e.g., equality in the age at death, or in the age of reproducing females), and so on (see Morris and Doak, 2002; Caswell, 2001; Caswell and others, 2018 for more details). The widespread use of MPMs to describe species population dynamics and the broad range of life history metrics that can be derived from them enables meaningful comparisons of life history across a wide taxonomic scope: a task made easier by the release of the COMPADRE Plant Matrix Database (Salguero-Gómez and others, 2015) and COMADRE Animal Matrix Database (Salguero-Gómez and others, 2016a), and associated functionality (e.g., the Rage and Rcompadre R packages (Jones and others, 2022))

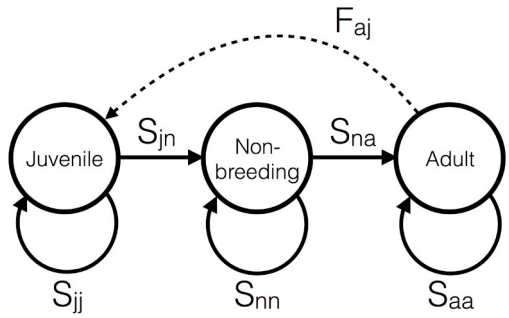
In this chapter, we use MPMs for a subset of vertebrate species from the COMADRE Animal Matrix Database alongside human MPMs derived by applying an iterative scheme that estimates life tables from raw census data from 96 countries (Keyfitz, 1966). We compare these sets of MPMs to contextualize the patterns of human evolutionary demography. We carefully select a set of life history traits that describe the multi-component life history strategy and use multivariate statistical analysis to contextualize the uniqueness (or otherwise) of human populations among the other vertebrates.

We illustrate that, across species, these life history strategies can be understood by examining their distribution across two major independent axes, one associated with the pace of life (in the demographic sense (see Nettle and Frankenhuis, 2019)), and the other associated with reproductive strategy. Demographic strategy, defined by position on these two axes, is not strongly structured by taxonomic affiliation. Humans sit apart from the other mammals, but not exceptionally so. We also show that within humans, the structuring of demographic strategies is markedly different from the cross-species patterns. This difference may reflect differences in the biological processes driving variation.

Methods

Demographic Data

We obtained data on the demography of humans and 90 other animal species across 84 genera, 50 families, 25 orders and 3 vertebrate classes. All of the data included are in the form of matrix population models (MPMs) with a dimension ranging from 2 to 60. MPMs describe the life cycle of an organism as a set of discrete stages, or ages, with transitions from (st)age-to-(st) age expressed as probabilities or recruitment (Figure 1). For our study, MPMs have a distinct advantage over life tables because they are far more widely available in non-human animal studies (Salguero-Gómez and others, 2016a), thus expanding our comparative evolutionary context. Like life tables, the properties of MPMs are well-understood (Caswell, 2001). From MPMs, one can calculate a broad range of metrics ranging from population dynamics measures, such as long-term population growth rate, to short-term dynamics descriptors, such as reactivity and damping ratio which quantify population responses to perturbation (Stott and others, 2011), to measures which may be better described as life history traits such as life expectancy and reproductive strategy (e.g., frequency and quantity of reproduction). Collectively these suites of demographic and life history parameters fully describe the life history demography of the population concerned at the time and place of study.



$$\mathbf{A} = \begin{bmatrix} S_{jj} & 0 & F_{aj} \\ S_{jn} & S_{nn} & 0 \\ 0 & S_{na} & S_{aa} \end{bmatrix}$$

Figure 1. An example of a matrix population model (MPM) and its associated life cycle diagram. This model has three stages defined by ontogeny. In the life cycle diagram, the arrows represent transitions from stage-to-stage in one time step (usually a year). The unbroken arrows represent survival, while the broken arrow represents recruitment to the juvenile stage. The letters next to the arrows indicate the relationship between processes described by the life cycle diagram and the matrix model below it. Thus, for example, Sjn represents a transition probability (survival) from the juvenile stage (j) to the non-breeding stage (n); Saa represents adult survival; and Faj represents the average number of recruits to the juvenile stage produced by an adult.

We obtained the non-human MPMs from the COMADRE Animal Matrix Database (Salguero-Gómez and others, 2016a). This database contains several thousand MPMs from hundreds of species and we use only a subset of these here. Specifically, we filter the matrices to include only tetrapods (mammals, reptiles and birds[1]) partly due to data availability and partly because we wanted to focus on unitary (non-modular) organisms with no clonality or retrogression (i.e., 'rejuvenation'), which pose numerous analytical and conceptual complications. In addition, we only include 'mean matrices' (i.e., the models parameterized with field data that represent the average dynamics expressed by a population across study periods and sites), where the data were collected under unmanipulated and non-captive conditions with a projection interval of one year, and where the matrix could be split into sub-matrices according to demographic processes of growth/survival and sexual reproduction (termed the U and F matrices respectively). We further tested these matrices to ensure they satisfied the mathematical conditions of irreducibility and primitivity, which are necessary for some of the calculations performed (Stott and others, 2012). Application of these criteria resulted in a dataset of 200 MPMs for 87 non-human species. Most species were only represented by a single matrix, but for others, where studies on a particular species have been published from more than one location or time period, there were several matrices available (median = 1, mean = 2.157, range = 1–16).

To this set, we added 1,657 human MPMs obtained from age-specific female population size, birth, and death data.[2] These data were sourced from three population atlases (Keyfitz and Flieger, 1968, 1990, 1971), on the Eurostat server (http://ec.europa.eu/eurostat), as described by Nicol-Harper et al. (2018), or from the Australian Bureau of Statistics, Official Statistics of Japan, Statistics Canada, Statistics New Zealand and the United States Census Bureau. These sources contain different data resolutions (the Eurostat data is age-specific, whereas the others group specific ages into five-year intervals) and different maximum ages, so all were treated using an iterative scheme (Keyfitz, 1966) to generate a 'common currency' of comparable matrices with five-year projection intervals. Keyfitz's iterative scheme takes snapshot data provided in population censuses and infers rates of survival (l_x) and fertility (m_x) assuming a stationary population structure. In other words, 'a life table that agrees with the data' (Keyfitz, 1966). Mueller et al. (to appear as Mueller, M., Packman, D. Townley, S., Hodgson, D., Dooley, C.A., Bijak, J., and Ezard, T.H.G, R and MATLAB functions to convert demographic census data to Life Tables and Leslie Matrices, Wellcome Open Research) give further discussion on the implementation of Keyfitz's iterative scheme (1966) as well as open-source code for MATLAB and R. The R functions are also available within the COMPADRE GitHub repository. These MPMs span 1780 to 2014 across 96 countries. For most countries, just a few MPMs were available (median = 6). For others, multiple years were available (e.g., 90 years for Sweden, 33 years for France, 15 years for Japan), which then allowed us to study how human populations have navigated through time within an organizational framework that contextualizes their demography to that of the other vertebrate species examined here.

1 Note that for convenience we use the traditional taxonomic definitions of class Reptilia and class Aves, rather than placing Aves within Reptilia.

2 Since acceptance of this chapter, the MPM data on the human populations used in these analyses have been made available in the COMADRE Animal Matrix Database (www.compadre-db.org).

We calculate six life history traits from each MPM, which we first list here, but then define more precisely in subsequent paragraphs (Table 1). These life history traits are broadly divided into measures relating to pace and shape of aging (Baudisch, 2011), as well as to reproduction and its distribution throughout the life course. In the former category, we included mean life expectancy (e_0), exceptional life span (Ω), and distribution of mortality risk (quantified by Keyfitz' life table entropy, H). We also included a measure which quantifies the degree of iteroparity: Demetrius' evolutionary entropy, *S*. Degree of iteroparity quantifies the spread of reproduction over the life course. Species with a very low degree of iteroparity reproduce only once before death,[3] while species with a higher degree of iteroparity reproduce numerous times. Finally, we include the basic reproduction number (R_0). Most of these metrics can be calculated directly from the MPMs, but the two entropy measures required that we first calculate a life table from the MPM. We accomplished this using the 'age-from-stage' methods developed by Cochran & Ellner (1992) and Caswell (2001). For the human MPMs, e_0 is a direct output of the iterative scheme implemented to calculate the lifetable (i.e., the conversion to MPM is redundant).

Table 1. The demographic traits used in our analysis. For a fuller description, see the main text.

Symbol	***Name***	***Description***
e_0	Life expectancy from birth	The average age at death of individuals in the population.
Ω	Exceptional life span	The age that only 1% of the population attains.
T	Generation time	The time taken for individuals of a population to be fully replaced by new individuals
R_0	Basic reproduction number	The average number of offspring produced over the lifetime of an individual.
H	Distribution of mortality risk	Quantified by Keyfitz's life table entropy. A measure describing the distribution of mortality risk over the life course. When mortality is constant $H = 1$, when it declines $H>1$, when it increases $H<1$.
S	Degree of iteroparity	The degree of iteroparity, or the uncertainty in the age of the mother of a randomly chosen newborn, is also known as evolutionary entropy (Demetrius, 1974). Organisms reproducing in a single reproductive bout have low values of S ($S \approx 0$) while those that reproduce steadily and evenly throughout the life course have high values of S.

Mean life expectancy (e_0) is a measure of the average time an organism can be expected to live. We calculated mean life expectancy from birth (i.e., from entering the first stage of the life cycle described by the MPM) using a Markov chain approach, which focuses on the time individuals spend in different states (i.e., life stages) as they pass through the life cycle. The primary tool for this analysis is the fundamental matrix (**N**) of the MPM (**A**), which provides a measure of the expected residence time in each state in a Markov chain (see Caswell 2001 for further details). The first column of **N** thus represents time that an individual that was

3 Such species are also referred to as being semelparous or monocarpic.

originally in the first stage spends in each subsequent stage and, therefore, the sum of the column is an estimate of life expectancy across all the stages. We calculated exceptional life span (Ω) as the number of time steps (i.e., years in this case) that it would take a population vector of 1000 individuals in the first stage and zero in all other stages to reach a population size of less than 10 (summation of the population vector $n < 10$) when multiplied iteratively following the chain rule of $n(t + 1) = \mathbf{A}^*n(t)$ (described in Caswell, 2001, and Morris & Doak, 2002). For the human MPMs, the calculation of exceptional life span was not possible because the oldest age class (80+ years) did not include a stasis transition (i.e., surviving within the age class). Thus, our analysis of the human MPMs alone excluded this variable; for our cross-species comparison we used an exceptional life span value of 100 years, based on an examination of life tables from the human mortality database (HMD). The exact value of exceptional life span varies among populations and time periods, and we therefore checked that varying the value used between 85 and 120 did not qualitatively influence our results. There are several measures of generation time (T), but we use the time taken for a population to increase by a factor of R_0, i.e. $T = log\ R_0/log\ \lambda_1$, which is straightforward to calculate from stage-classified matrix models (Caswell, 2001).

We included two entropy measures. First, Keyfitz entropy (H, also known as life table entropy (Keyfitz, 1985)) is a measure that describes the way that mortality risk is distributed over the life course. Where mortality risk remains constant through life $H = 1$, where mortality risk is relatively high early in life and declines with age (as in, for example, teleost fish) $H > 1$, and where and where mortality risk increases with age (as in humans) $H < 1$. In the extreme case where there is zero mortality until all individuals die at the same time, $H = 0$. H can therefore be interpreted as a measure of the shape of the survivorship curve, and of the mortality trajectory, and thus the 'shape' of senescence (*sensu* Baudisch, 2011) (See Figure 2A). In addition, it is useful to note that H is also a descriptor of the distribution of the age at death, or lifespan equality, with low values of H corresponding to high life span equality, where most individuals die at a similar age (Colchero and others, 2016). Note that H is well-correlated with numerous other viable candidate measures of the shape of the mortality trajectory — and consequently the distribution of the ages at death (Wrycza and others, 2015). Secondly, Demetrius' evolutionary entropy (S), quantifies the degree of iteroparity, or the spread of reproduction across the life course and can also be interpreted as the uncertainty in the age of the mother of a randomly chosen newborn (Demetrius, 1974). Organisms that reproduce in a single reproductive bout (e.g., semelparous species) have low values of S ($S \approx 0$), and those that reproduce steadily throughout their life course have high values of S ($S >> 0$) (Figure 2B).

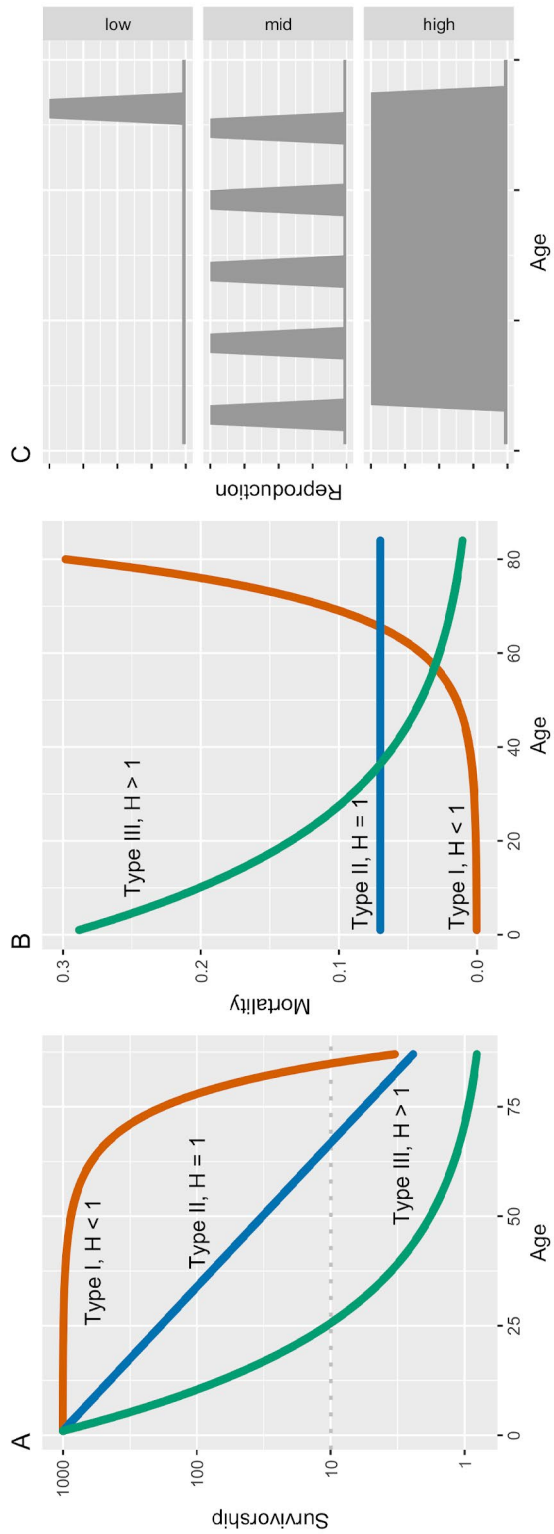


Figure 2. The relationship between survivorship curves and the distribution of mortality risk over the life course as quantified by Keyfitz's life table entropy (A) and the corresponding mortality trajectories (B). In this case, the entropy values are 0.16, 1.00 and 1.28 for Type I, II and III respectively. C illustrates the relationship between distribution of reproductive output and degree of iteroparity measured using Demetrius' evolutionary entropy ranked from low entropy in the top panel to high entropy in the bottom panel.

Finally, to calculate the basic reproduction number (R_0), which is the average number of offspring produced over the lifetime of an individual, we used the methods described by Caswell (2001). Thus, we first calculated a matrix, **R**, as the matrix product of the fundamental matrix (**N**) and the **F** matrix (sexual reproduction). Then we calculated R_0 as the dominant eigenvalue of the matrix **R**. Collectively, these metrics provide a well-rounded description of the life history strategy of the species and populations in our study.

Phylogenetic Data

Phylogenetic trees describe the hypothetical evolutionary relationships of groups of organisms with a single common ancestor. They are necessary in comparative analyses across species, because (i) common statistical approaches assume independence of errors, which is not the case in analysis where each datapoint is related in some structured hierarchical way to all others, and (ii) it is useful to gain insight of how trait values are structured by the phylogeny (Blomberg and others, 2003; Freckleton and others, 2002). We use a species-level phylogeny constructed by Healy et al. (2019) based on available phylogenies for birds (Jetz and others, 2012), mammals (Kuhn and others, 2011) and reptiles (Pyron and Burbrink, 2014).

Statistical Methods

Our statistical methods differed between our central cross-species analysis, which includes all available species data, and the analysis we performed on solely the human data. For the cross-species analysis, we heed the well-known observation that demographic parameters scale with body mass. We therefore obtained body mass for our species from Myhrvold et al. (2015) and regressed each demographic trait against body mass in a phylogenetic generalized least squares (PGLS) regression. In addition, we controlled for matrix dimension, which can potentially confound comparative analyses (Salguero-Gómez and Plotkin, 2010), by adding it (log10 transformed) as a covariate. We used the residuals from this relationship, which describe each trait's departure from expectation given body mass, phylogenetic relationships, and matrix dimension in a principal component analysis (PCA) (Gaillard and others, 1989). For the human-only analysis we did not need to account for phylogeny, body mass or matrix dimension in this way since the subjects are all populations from a single species, with approximately the same body mass and from matrices of equal size. Nevertheless, we acknowledge that the human data has numerous structural dependencies that may influence mortality and fertility patterns, and these are the subject of ongoing work.

Principal Component Analysis (PCA)

Principal component analysis (PCA) is a statistical technique that reduces complex, multidimensional data to a smaller number of dimensions (hereafter, 'axes') that are linearly uncorrelated (Legendre and Legendre, 2012; Mardia and others, 1979). We use PCA to characterize our complex life history data by using either the residuals from our cross-species regression models or the actual demographic estimates for the analysis of humans alone. For the cross-species analysis, to avoid swamping the analysis with human data, we used representative data from a subset of the human MPMs. Specifically, we used the mean demographic trait values for each of the 84 countries for which we had data. We fitted the PCA using the prcomp function in the stats package of R, and took the standard approach of z-transforming the data (mean centered at 0 with a standard deviation of 1 (Legendre and Legendre, 2012)). We determined the number of principal component axes to retain using Horn's parallel analysis (Horn, 1965; Dinno, 2009). We then used those retained axes to define the life history strategy space onto which we will contextualize the demographic behaviour of the species and populations included in our analysis.

We conducted several PCA analyses to explore life history variation both across species and within humans. First, in order to define the framework for comparisons between humans and other vertebrates, we carried out a PCA across all species (including humans). A large body of previous work (Stearns, 1992; Jones and others, 2008; Gaillard and others, 2005) has suggested the existence of a 'fast-slow continuum' of life history. In this putative continuum, species' demographic strategy can be adequately described by position along a single axis of variation with species with high allocation to reproduction at one end, and those with low allocation at the other end. We therefore hypothesized that there would be clear evidence of a major fast-slow life history continuum, with traits relating to time aligning with the first PC axis. We also expected to see an orthogonal axis related to the range of reproductive strategies available, based on previous work in plants and animals (Salguero-Gómez and Jones, 2016; Salguero-Gómez and others, 2016b). In this second axis, at one extreme are highly-fecund, highly-iteroparous species while at the other extreme are semelparous species that reproduce only once.

Second, we conduct a PCA focused on human populations to evaluate how the life history structuring of our species differs from life history structuring across the vertebrates. For three human populations with high temporal replication, we examined how their relative positioning within the PCA life history strategy space has shifted through time. Broadly we expect to see a similar structuring — with pace of life being a dominant axis. When looking at changes through time in particular populations, we expect to see clear traversal through life-history strategy space reflecting the well-known increases in life expectancy and life spans alongside a reflection of changes in life table entropy that have accompanied changes in the distribution of age at death.

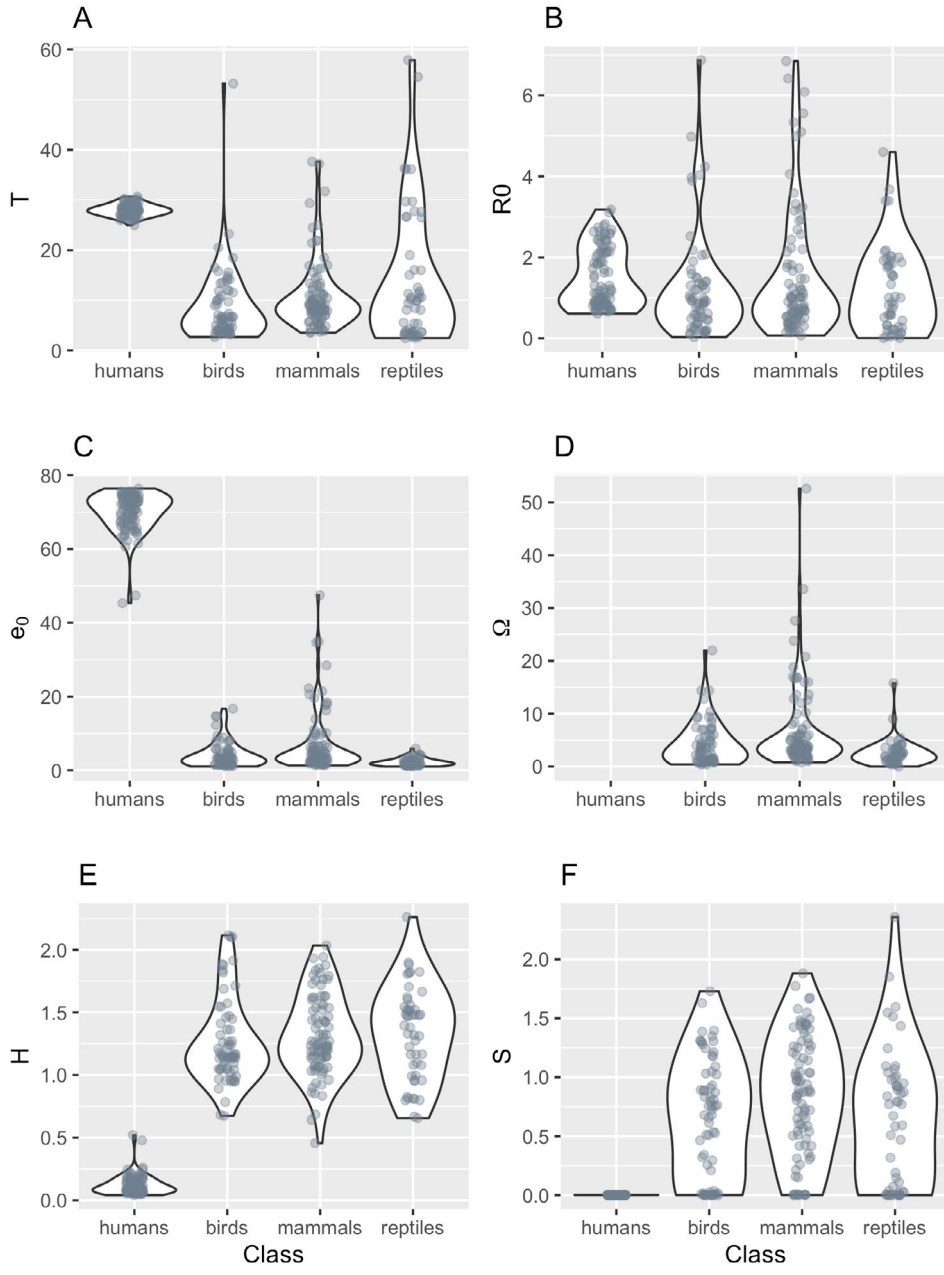


Figure 3. The distribution of six demographic trait values in MPMs used in this study for humans, birds, non-human mammals, and reptiles. A. generation time (T); B. basic reproduction number (R_0); C. mean life expectancy (e_0); D. exceptional life span (for non-humans only) (Ω); E. distribution of mortality risk (H); F. degree of iteroparity (S) — see text for details. Each point represents a single estimate derived from an MPM. Non-human species are represented by between 1 and 16 estimates (mean = 2.157, median = 1), humans are represented by 96 measurements which are the arithmetic means for each country in the available dataset. The values for S in humans were all very low (range: 0.0012–0.0030).

Results

Demographic Measures and Phylogenetic Signal

Across the taxonomic groups the demographic trait distributions overlap for the non-human animals, though it is clear that the longevity and life expectancy of some mammals greatly exceed those of the other two classes (Figure 3). It is also very clear that humans are rather exceptional when considering these life history trait values, with trait distributions that are far-removed from the other mammals in most cases (Figure 3).

The strength of phylogenetic signal in the relationships between body mass and the demographic traits varied considerably, and the estimates were rather uncertain in many cases. In descending order of signal strength, the values were: generation time (0.96, 95% CI = 0.82–1.00), life expectancy (0.64, CI = 0.36–0.82), life table entropy (0.51, 95% CI = 0.04–0.83), exceptional life span (0.47, 95% CI = 0.04–0.73), R_0 (0.10, 95% CI = 0.00–0.63), and evolutionary entropy (0.00, 95% CI = 0.00–0.71).

Principal Components Analysis: All Species

The PCA analysis (Figure 4) revealed that the life history strategy of the animals in our dataset is adequately described by two principal component axes, according to Horn's parallel analysis (Horn, 1965; Dinno, 2009). Together these two axes explain 70.59 % of variation in demographic traits (45.85 and 24.74 % for PC 1 and 2 respectively). The analysis also revealed that all taxonomic classes (birds, reptiles, and mammals) cluster together in demographic PCA-space (Figure 4), and that the humans are to be found outside the 95% CI bivariate ellipse that represents the limits of mammalian life history strategy. The loadings (Figure 4 and Table 2) indicate that three of the six variables (exceptional life span (Ω), mean life expectancy (e_0) and generation time (T)), align well with PC1 and two align well with PC2 (distribution of mortality risk (H), degree of iteroparity (S)). Basic reproduction number (R_0) appears to align approximately halfway between the two major PC axes. Since generation time, life expectancy, and exceptional life span are related to the timing of key life events we interpret this first major axis as being strongly associated with the fast-slow continuum (Stearns, 1992; Jones and others, 2008). The second axis (PC2) can be interpreted as representing the probability distribution of key events in the life course: reproduction (indicated by S) and mortality risk (indicated by H). For example, mortality risk may be distributed evenly (negligible senescence), or may increase or decrease with age (senescence or negative senescence). Likewise, reproduction can be concentrated within a particular part of the life course (as in humans) or distributed more evenly.

Table 2: Variable loadings for the first two principal components of the PCA analyses. e_0 = life expectancy; Ω = exceptional life span; T = generation time; R_0 = basic reproduction number; H = distribution of mortality risk; S = degree of iteroparity. SD = standard deviation. All variables were mean standardized before analysis. See Table 1 and main text for details.

Analysis	*PC*	*SD*	e_0	*Ω*	*T*	R_0	*H*	*S*
Cross-species	PC1	1.659	0.525	0.566	0.390	0.300	0.119	0.384
	PC2	1.218	0.359	0.178	0.102	−0.140	−0.736	−0.518
Human (all)	PC1	1.750	0.514	–	−0.006	−0.481	−0.505	−0.500
	PC2	1.014	−0.131	–	−0.940	−0.214	0.195	−0.138

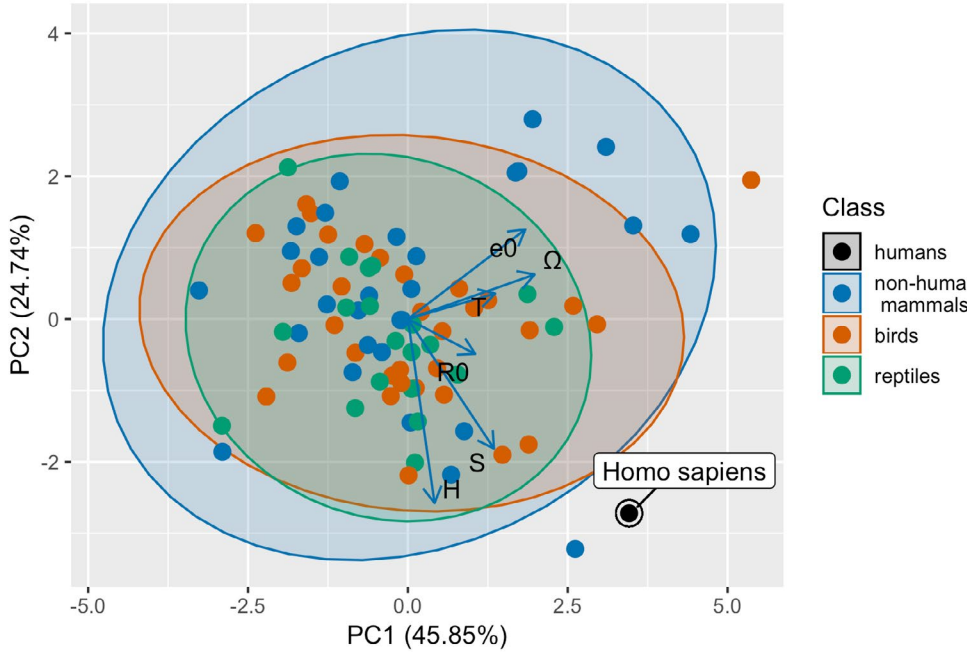


Figure 4: Principal components analysis of demographic behaviour for 34 bird species, 33 mammal species (including humans), and 19 reptile species. The ellipses illustrate the life-history strategy space occupied by these taxonomic groups (excluding humans), and are defined by the 95% bivariate confidence interval associated with the PC scores for each group. The arrows represent the principal component loadings for each of the demographic variables in the analysis: T = generation time; R_0 = basic reproduction number; e_0 = mean life expectancy; Ω = exceptional life span; H = distribution of mortality risk; S = degree of iteroparity — see text for details. See Table 2 for variable loading values. The outlier point for the non-human mammals is the blue monkey (*Cercopithecus mitis*), and the outlier for the birds is European honey buzzard (*Pernis apivorus*).

Principal Components Analysis: Humans Only

Examining the PCA for humans reveals a markedly different picture. Although again, the first two PC axes were sufficient to explain most 81.81% of the variation (61.26% and 20.55% for PC 1 and 2 respectively), these axes were less-readily identifiable as clear 'pace of life' and probability distribution of life events (or indeed "reproductive strategy") axes (Figure 5A). The loading for T is markedly larger than all others, and dominates PC2, but the relationship between T and other variables (as we will show below) is dependent on the time period analyzed. Interestingly, although the loadings for e_0 and H were orthogonal in the cross-species analysis, they were in opposition in the human analysis, indicating that increased average life span is associated with a decline in entropy (i.e., an increase in the equality of age at death). The closest association among loadings was between R_0 and S highlighting the close positive association between these traits.

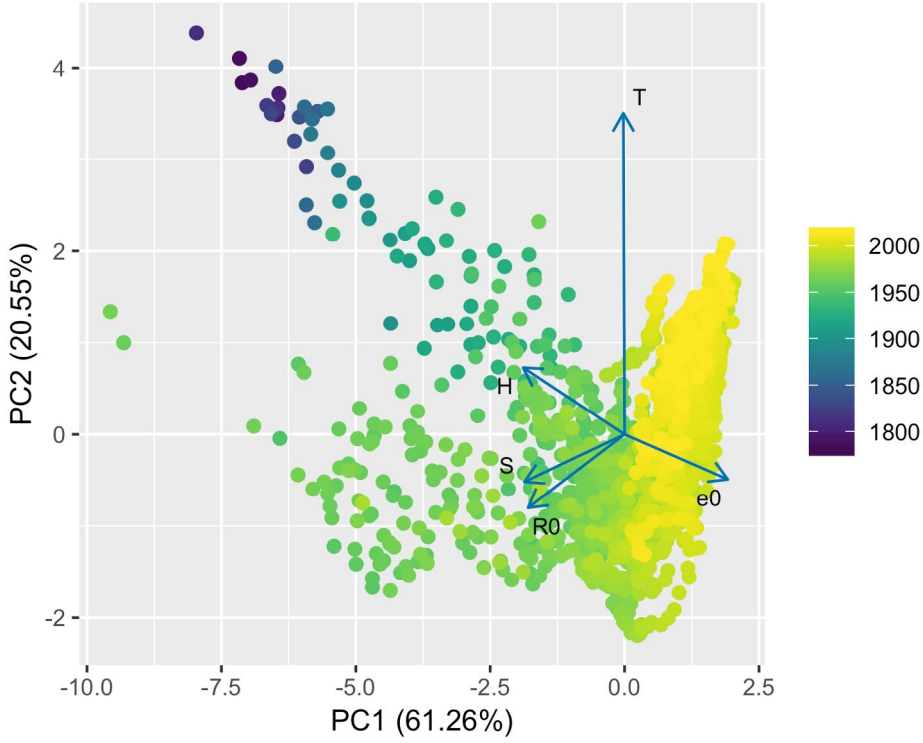


Figure 5: Principal components analysis (PCA) of demographic behaviour for 1657 MPMs for humans. There are 96 countries represented by between 1 and 90 matrices (mean = 17.26, median = 6), representing populations from years between 1780 and 2014. The points are colour coded, with older points being dark purple and more recent points being bright yellow. The principal component loadings for each of the demographic variables are represented by blue arrows: T = generation time; R_0 = basic reproduction number; e_0 = mean life expectancy; H = distribution of mortality risk; S = degree of iteroparity — see text for details. The key points to observe here are the strength and associations of the principal component loadings (i.e., which arrows sit together, and which are in opposition or orthogonal to each other). See Table 2 for variable loading values.

A particularly striking phenomenon clear from Figure 5 is the shift across demographic strategy space that has occurred through time that is apparent from the colour coding of the data points by year. This shift, from the top-left towards the bottom-right of the figure indicates, broadly speaking, that populations have trended towards increased life expectancy, decreased entropy (with mortality more concentrated towards the end of life), and with a shorter generation time. These trends can perhaps be seen more clearly by examining how some exemplar countries, Sweden, France, Japan, and Bulgaria have "moved" across PCA life history strategy space through time (Figure 6).

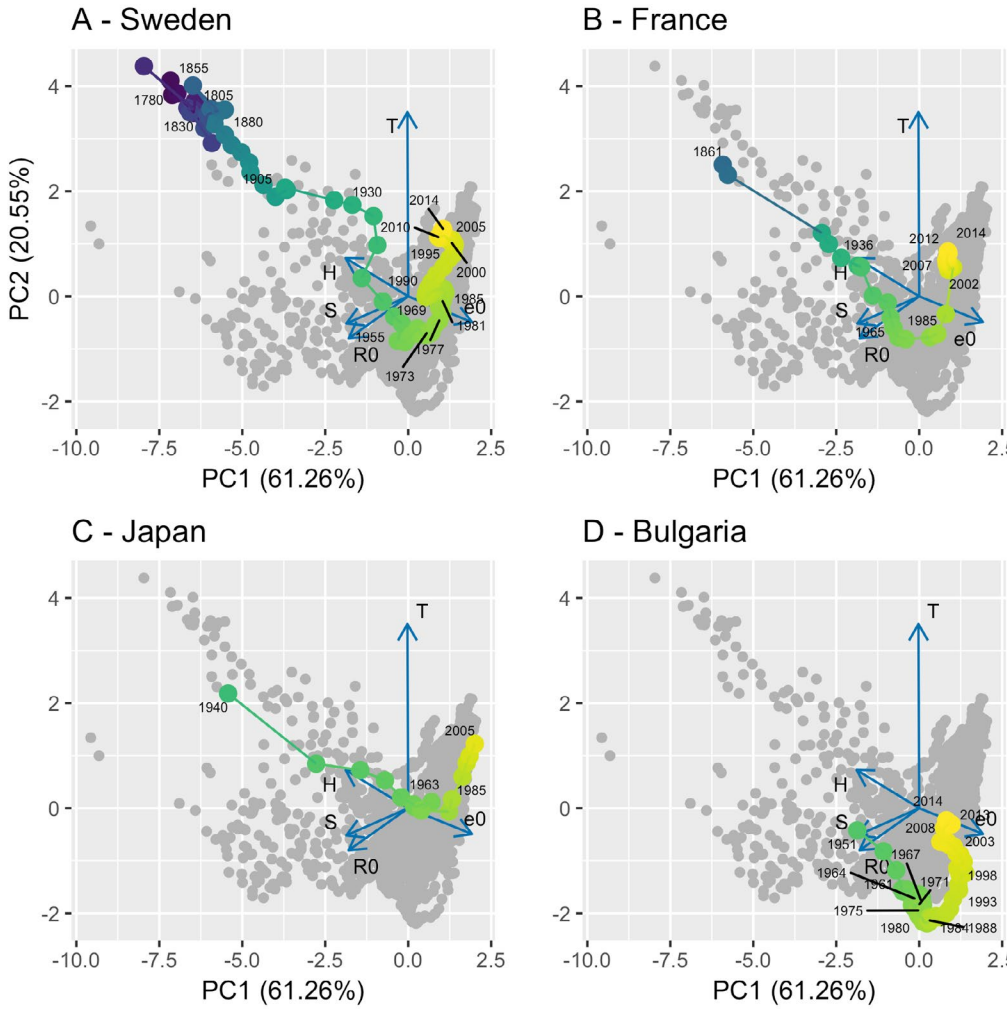


Figure 6: The traversal of demographic PCA-space by four example countries, (A) Sweden, (B) France, (C) Japan, and (D) Bulgaria. The grey points are the points for the non-focal countries while the coloured points represent the focal country. The points are colour coded to represent time, using the same scale as in Figure 5. The blue arrows represent the principal component loadings for each of the demographic variables in the overall analysis: T = generation time; R_0 = basic reproduction number; e_0 = mean life expectancy; H = distribution of mortality risk; S = degree of iteroparity — see text for details.

Discussion

We aimed to explore where the demography of humans fits in to the bigger evolutionary picture of other vertebrate species. The distribution of demographic behaviour among species is of profound interest to evolutionary biologists with researchers questioning whether, and how, species with certain combinations of demographic traits are restricted to particular habitats (Southwood, 1988). One of the dominant frameworks that has been used to study this topic is the fast-slow continuum, which focuses on the trade-off between survival (or somatic maintenance) and reproduction (Stearns, 1992). This trade-off proposes that the entire range of life histories can be described by position along a single axis with proportionally high allocation to reproduction at one end and low allocation at the other end. The existence of this fast-slow life history framework has received considerable support (Gaillard and others, 2005; Jones and others, 2008; Oli, 2004; Sæther, 1987), but the picture has been complicated by research revealing additional axes that structure observed life-history variation among species (Salguero-Gómez and Jones, 2016; Bielby and others, 2007; Gaillard and others, 1989).

Our examination of the principal components axes of humans, mammals (including humans), birds, and reptiles supports our hypothesis, confirming that the distillation of the great variety of demographic behaviour requires two major axes. These two axes explain a large proportion (~70%) of the variation life history across species. This figure is markedly larger than found across the plant kingdom (55%) by Salguero-Gómez et al. (2016b), but less than the 80% reported by Salguero-Gómez & Jones (2016) for an analysis across both plant and animal kingdoms. However, the life-history traits included differ among these three analyses making over-interpretation of these differences unwise. A more appropriate comparison is with the result of the human-only PCA conducted for this chapter, where we used the same approach and variables (with the exception of exceptional life span), and for which the PCA explained 82% of demographic variation. The result that a cross-species analysis explains less variation than a within species analysis is likely caused by the more constrained repertoire of available life history strategies within- rather than among-species.

Previous cross-species studies in plants and animals (Salguero-Gómez and Jones, 2016; Salguero-Gómez and others, 2016b) interpreted the two dominant principal component axes as (i) the pace of life and (ii) the reproductive strategy axes. The former axis relates to the timing of life events such as generation time (which is tightly linked to age at maturity), life expectancy, and life span, while the latter axis is related to the amount and distribution of reproductive output. As expected, we find good evidence for the existence of a dominant 'pace of life' axis in the cross-species analysis. However, we make a slightly broader interpretation of the second axis, which we interpret as indicative of the 'distribution of life events'. Our interpretation here is driven by the fact that both life table entropy (indicative of distribution of mortality) and degree of iteroparity (indicative of distribution of fertility) align well with this axis. The almost total overlap of the three taxonomic classes in life history strategy space occupied indicates that life history strategy is not structured taxonomically. It is striking that although humans may not be exceptional in terms of some individual demographic traits, when considered collectively in a PCA these traits reveal that our demographic behaviour falls some way outside of the norm for mammals. It is worth considering, however, that the data we are using in this study may not reflect the ancestral state of humans because they are dominated by contemporary populations with low mortality and fertility. In future work it will be interesting to explore how populations

that may be closer to the ancestral state fit into this schema (e.g., using data on contemporary hunter gatherers).

The direction and magnitude of the PCA loadings illustrate the 'life history architecture' — the complex of correlations among our chosen set of demographic traits. They show that across species the traits we included in our analysis can be divided into two orthogonal sets of highly-correlated traits: (i) life expectancy, exceptional life span, and generation time, and (ii) distribution of mortality risk, and degree of iteroparity. Net reproductive output fell in between these two major axes. Within these trait groups, as one trait has evolved, the others have 'hitched a ride' due to mathematical association or genetic correlation: e.g., as life expectancy has increased, so has exceptional life span and generation time. The orthogonality of these two groups suggests that although the within-group traits are tightly correlated, the traits in different groups are not, and have the capacity to evolve more-or-less independently. For example, it seems that species with short or long exceptional life spans (or life expectancies, or generation time) can have any type of mortality trajectory (indicated by the distribution of mortality risk, life table entropy). Since life span and life expectancy are measures of the pace of mortality, while entropy is a measure of the shape of mortality, this supports Baudisch's (2011) assertion that pace and shape are likely to be independent aspects of the mortality trajectory. We note, however, that exceptional life span and life expectancy are not perfectly aligned. This indicates that these measures do not scale together, but rather that as exceptional life span increases, life expectancy does not keep pace, which leads to a tendency for increased values of H (e.g., moving from a Type I towards a Type II or III survivorship curve, or towards greater inequality in age at death). This observation is supported by the result that the loading for H is not perfectly orthogonal to life expectancy.

An examination of the PCA for humans alone reveals that life history architecture within our species is rather different to the cross-species PCA. Our interpretation of the dominant axes as pace-of-life and distribution of life events are now far less clear cut. Furthermore, rather than the traits being divided fairly neatly into two approximately orthogonal groups, the situation of humans appears to be more complex. Nevertheless, there are some interesting observations: life expectancy is directly opposed to the distribution of mortality risk (H), indicating a close positive association between life span equality and life expectancy. This observation supports recent work on humans and our close primate relatives that shows a striking linear relationship between life expectancy and life span equality (Colchero and others, 2016). The marked shift in position in the life history space (PCA-space) begs the question: has life history architecture changed through time? Such changes in the demographic variable loadings might be expected given the well-known and dramatic reductions in infant and childhood mortality (Vaupel, 1986; Hill and others, 2012) and the occurrence of the demographic transition from high birth and death rates to low birth and death rates as societies industrialize (Kirk, 1996). However, this analysis is non-trivial given the within-country dependencies and the different geographic coverage of the data through time, and it would be difficult to disentangle temporal changes common across countries from compositional changes.

The clear differences between the cross-species and within-species analysis are enigmatic, but it is important to bear in mind that the processes leading to differences among data points in these two analyses are fundamentally distinct. Indeed, this distinction between, within-,and among species analyses have led some to question the wisdom of invoking life history theory,

and, in particular continua, such as the fast-slow continuum, to explain variation within species (Zietsch and Sidari, 2019). The relationships and differences among traits in the cross-species analysis are primarily the result of evolution. Selection pressures vary among species resulting in different optima for demographic traits depending on environmental conditions. Within humans (i.e., variation across human populations and through time), the observed demographic variation and structuring are likely the result of phenotypic plasticity (the capacity of a genotype to exhibit different phenotypes depending on the environment), rather than natural selection. This plasticity has resulted in some striking patterns in the within-human analysis.

The remarkable plasticity of demographic behaviour in modern humans is clearly seen with the passage through time of our four example countries across life history strategy space. The tendencies towards longer exceptional life spans and longer life expectancies are well-known among human demographers (Oeppen and Vaupel, 2002). So too is the trend that mortality distribution has shifted leading to greater equality in the age at death (Colchero and others, 2016; Gillespie and others, 2014). There is clearly a rather large difference among populations in the speed that life history strategy space is traversed. For example, it took Japan a fraction of the time to traverse this space than that taken by France and Sweden. Another striking feature is the non-linear U-shaped trajectory seen in all four of our example countries. It is possible that this may be driven by the advent of birth control methods in the 1960s-70s, but further investigation is needed to support this. Although the qualitative pattern shown in the different countries are similar, there are qualitative differences that will be fascinating to explore.

Our approach of distilling demographic strategy into principal component axes, which is derived from workers focusing on the life history evolution of non-human species (Gaillard and others, 1989), offers a useful tool for the exploration of human demography. First, it allows us to see how our life history strategy compares with other species; the results reveal we are a mammalian demographic outlier. Second, it offers a tool for tractably exploring how complex strategies are influenced by environmental drivers. For example, it would be interesting to explore the impact of technological and public health developments, wealth, and income equality on human life history *strategy* rather than single demographic variables. The comparably vast wealth of data available across diverse human populations represent a treasure trove to help us understand the development of life history strategies.

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Appendix: Species List

The species included in this analysis, in addition to humans (*Homo sapiens*) are as follows:

Mammals: *Alces alces*, *Brachyteles hypoxanthus*, *Canis lupus*, *Cebus capucinus*, *Cercopithecus mitis*, *Cervus elaphus*, *Eidolon helvum*, *Elephas maximus*, *Enhydra lutris*, *Eumetopias jubatus*, *Halichoerus grypus*, *Macaca mulatta*, *Macropus eugenii*, *Marmota flaviventris*, *Mirounga leonina*, *Mustela erminea*, *Odocoileus virginianus*, *Onychogalea fraenata*, *Ovis canadensis*, *Panthera pardus*, *Phocarctos hookeri*, *Propithecus edwardsi*, *Propithecus verreauxi*, *Rangifer tarandus*, *Tamiasciurus hudsonicus*, *Urocyon littoralis*, *Ursus americanus*, *Ursus maritimus*, *Zalophus californianus*.

Birds: *Agelaius phoeniceus*, *Amazona vittata*, *Ammodramus savannarum*, *Anas laysanensis*, *Anser anser*, *Aquila fasciata*, *Bonasa umbellus*, *Bostrychia hagedash*, *Buteo solitarius*, *Calidris temminckii*, *Campylorhynchus brunneicapillus*, *Centrocercus minimus*, *Certhia americana*, *Ciconia ciconia*, *Falco naumanni*, *Falco peregrinus*, *Fulmarus glacialis*, *Gavia immer*, *Gyps coprotheres*, *Haliaeetus albicilla*, *Himantopus novaezelandiae*, *Lagopus leucura*, *Lagopus muta*, *Milvus migrans*, *Pernis apivorus*, *Phalacrocorax auritus*, *Phoebastria immutabilis*, *Setophaga cerulea*, *Sterna hirundo*, *Sternula antillarum*, *Strix occidentalis*, *Thalassarche melanophris*, *Turdus torquatus*.

Reptiles: *Alligator mississippiensis*, *Apalone mutica*, *Apalone spinifera*, *Caiman crocodilus*, *Caretta caretta*, *Chelodina expansa*, *Chelonia mydas*, *Chelydra serpentina*, *Chrysemys picta*, *Clemmys guttata*, *Cryptophis nigrescens*, *Drymarchon couperi*, *Emydura macquarii*, *Kinosternon subrubrum*, *Malaclemys terrapin*, *Phrynosoma cornutum*, *Podocnemis expansa*, *Sceloporus arenicolus*, *Sceloporus grammicus*, *Vipera aspis*.

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10. The Role of Ontogeny in Understanding Human Demographic Behaviour

Paula Sheppard and David A. Coall

Ontogeny, the development of an organism from conception to maturity, is one of Tinbergen's two proximate explanations for understanding why we do the things we do. As well as genetic inheritance, the developmental environment, to which parents make a large contribution, is crucial for shaping a child's life. It not only shapes their physical and psychological development, but also influences the adult child's reproductive strategy and ultimately their life expectancy. Demographers care about fertility and mortality, which, when understood within an evolutionary framework, are two entwined processes that influence, and are in turn influenced by, the individual's developmental trajectory. Here, we provide a summary of how development operates throughout life: from the womb, through childhood, adolescence and puberty, the reproductive years through to menopause and death. We take a life history approach with a focus on how developmental influences during early life have long-reaching consequences for mortality and fertility. We illustrate each section with theoretical advances, empirical examples and evaluation of the current literature. We hope to demonstrate that thinking about human demographic behaviour can be revealing in light of ontogeny, and provide a useful theoretical basis for demographic research.

Introduction

Ethology is the study of animal behaviour, starting with the assumption that organisms behave in ways that are adaptive (i.e. that enhance reproductive success), given individual and environmental constraints. Human behaviour is complex and includes a much richer social environment than other animals, but the same ethological principals can be applied. Niko Tinbergen, a Nobel-prize-winning ethologist, conceptualized an insightful multifaceted approach to understanding animal behaviour (Tinbergen, 1963). He argued, following Ernst Mayr's (1961) teleology, that any ethological question could be answered at two levels — the ultimate (why a trait exists) and the proximate (how a trait operates), and each can be split in two components: phylogeny and function are ultimate explanations, and ontogeny and mechanism are proximate explanations. These four levels of explanation provide a framework for understanding behavioural traits.

Broadly, an ultimate explanation describes a phenomenon at its evolutionary, or adaptive, level — i.e. what is its function, and how did it evolve (phylogenetic). Proximate explanations are the more immediate or direct reasons which can be understood in terms of the mechanism(s) or triggers of the behaviour, as well as by the organism's development

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(ontogeny) as an individual. For example, if we were to wonder "why do bears hibernate?", four explanations can be given, and all would be correct. At the proximate level, bears hibernate because it is winter, food is scarce, and they feel cold (these are mechanisms). The propensity to hibernate is partly an innate trait, and partly learned by bear cubs denning with their mothers for the first few winters of their lives (this is the ontogenetic explanation). The ultimate explanations are that bears hibernate because it is adaptive for them to conserve energy and drop body temperature to survive harsh winters when food is scarce, improving their survival and reproductive chances — ancestral bear species who did hibernate were selected for — i.e. hibernation improves a bear's survival and reproductive chances. The phylogenetic explanation for bear hibernation is that bears are part of the Ursine family and hibernation is part of their evolved behavioural repertoire.[1] Tinbergen's framework remains a hugely useful paradigm for thinking about animal and human behaviour, and although a more modern usage might include supplementary questions to provide fuller explanations (Bateson and Laland, 2013), the proximate/ultimate distinction is the foundation of any evolutionary understanding of behavioural traits.

Here we focus on the role of ontogeny (or development[2]) in understanding human demographic behaviour. Development is partly about traits that develop in the womb through to puberty and reproductive maturation (at least in mammals), but also refers to learned behaviours, which are especially important during childhood. Demographers are interested in three main facets of human behaviour, namely fertility, migration, and mortality.

Following on from the previous example, we can apply Tinbergen's four levels of explanation to a demographic question. For instance, "why do humans live so long?". Phylogenetically, humans are primates, which all live relatively long for their body size, and, in fact, among primates humans have the longest lifespans and slowest life histories. Long lifespans are adaptive (functional) for cooperative breeding (sharing childrearing duties among kin and non-kin), which allowed for the human trait of raising numerous "stacked" dependent offspring at a time (Bogin, 1998). The mechanisms for longevity are complex but may include the neural and hormonal responses triggered by caregiving that reduce stress and accentuate physiological health, leading to longer, healthier lives (Hilbrand and others, 2017). The ontogenetic explanation is that we gestate our young for nine months but give birth to highly altricial neonates (underdeveloped newborns compared to other primates) requiring prolonged periods of infancy in which highly dependent suckling babies mature. Human childhoods are also long compared to other primates, who only have a short juvenile growth period between weaning and adulthood (Hawkes, 2003). Human children enjoy many childhood years in which they can indulge in important social learning and skill acquisition with very low costs as they are still fed and protected by their parents. These longer early life stages are part of a suite of life history characteristics particular to humans (Kuzawa and Bragg, 2012; Bogin, 1999).

1 Bears do not actually hibernate; they go into "torpor" which is a very deep sleep state, but not strictly hibernation. The four explanations as to why they do so still apply.

2 We use these two terms interchangeably in this chapter.

Theoretical Concepts

Life History Theory

We adopt a life history approach, focusing both on the human life stages prior to adulthood, as well as briefly addressing the ontogenetic process during later life, including old age, menopause and death. Ontogeny does not only refer to early development; it is relevant to the entire life course. The two demographic topics most relevant to ontogeny are fertility and mortality, and they are integral to understanding life history theory (although there is some evidence that migration can also affect human developmental trajectories (Núñez-De La Mora and others, 2007)).

Life history theory (LHT) is an evolutionary biology framework which can be used to explain the timing of human life events pertaining to growth, maintenance and reproduction at both the species and the individual level. The basic premise of LHT is that organisms make decisions about how best to allocate limited resources towards survival, growth and development, and reproduction in a bid to enhance their reproductive success, and thus their evolutionary "fitness" (genetic legacy) (Stearns, 1992; Kuzawa and Bragg, 2012). The fact that resources are limited leads to life history trade-offs; for instance, nutrition apportioned to growth cannot also be used for reproduction, which explains why organisms stop growing when they reach reproductive maturity. At the species level, the life histories of long-lived animals (including humans) are characterized by large body size, long gestation periods, production of relatively few offspring who remain dependent on the parent(s) for a long time (i.e. have extended childhoods), and relatively low early-life mortality. This suite of traits is typical of a slow life history species, and humans are among the slowest. This being the case, there is also plenty of variation among humans. Individuals who live in harsh environments with lower resource availability tend to live shorter lives than those from more affluent settings. Infant mortality is higher in harsh conditions, leading to higher rates of reproduction to offset this risk to lineage extinction (Chisholm, 1993). The relationship between mortality and fertility provides an ultimate, adaptive explanation for much of human reproductive behaviour.

Plasticity and prediction

Phenotypic plasticity is also important for understanding the basics of how development operates. The phenotype is the observable expression of the individual's genetic potential, as shaped by the environment; phenotypes are the various physical and behavioural features of the organism — each trait, such as height or hair colour, is a phenotype. There is a difference between what is genetically inherited, and what is produced by that heredity. On average an individual inherits half of his or her genes from each parent, and together these constitute the individual's unique genotype; all of our cells have the same genotype (except sex cells), but not all the genes are expressed in each phenotype, although they have the potential to be, and they can be passed on to the next generation. Phenotypic plasticity refers to the adaptive process that allows an organism to make flexible "choices" about how to behave or react to certain

environments[3] (Pigliucci, 2005; Fusco and Minelli, 2010). In other words, any genotype can express different phenotypes depending on the environment. This flexibility means we have the potential to express an array of behaviours around a given trait (i.e. adopt a strategy) and the environment we find ourselves in triggers one or more responses. Because this flexibility has been shaped through evolutionary history, and those individuals who could be flexible were more likely to survive and reproduce, plasticity is expected to be adaptive.

Insofar as developmental plasticity promotes Darwinian fitness, it also comes at a cost that can have negative health consequences in humans (Wells, 2019). There are conflicting views as to how the relationship between the environment and genotype works exactly. The concept of a predictive adaptive response (PAR) is that, given the environment in the organism's early development (usually in the womb for mammals), the organism programs its strategy and then sticks to it (developmental programming). The assumption is that early environments are highly indicative of future environments; therefore, a phenotype that aligns with an early environment that remains stable is assumed to be adaptive (Gluckman and others, 2005). For humans, however, this is an unlikely scenario as a great deal can change during a lifespan of fifty to eighty years.

Throughout hominid evolution, environmental instability has been predominant. Even ancestral humans would have encountered variability in environments due to climatic fluctuations and migration (Foley, 1995; Potts, 1998). A recent reconsideration of how plasticity operates has focused on internal prediction. This argument stresses that early environmental conditions can leave a mark on the physical or mental health of the individual (Nettle and others, 2013). If the environmental assault is serious enough, the mark it leaves can impact on the individual's lifespan by increasing the likelihood of premature death through a higher susceptibility to disease, or high levels of psychological stress. The individual's strategy is then calibrated accordingly. In this case, there is no need to predict the future environment; the impact of early environments is embodied, carried within the person (Rickard and others, 2014). In the context of life history theory, under harsh environments the priority is simply to survive, therefore, physiological changes during gestation in response to a challenging environment is "making the best of a bad start", not adapting for the future (Berghänel and others, 2017; Jones, 2005; Vitzthum, 2001; Rickard and others, 2014).

We next describe how ontogeny influences demographic traits (here, fertility and mortality) with the underlying assumption that humans react and behave flexibly in response to a combination of their genetic endowment and the environmental conditions within which they develop and mature.

Prenatal Environment and Development

Prenatal development focuses on the early stages of the life cycle, our most precarious time of life; it is the time of most rapid development, and thus the time of highest risk. Prenatal development, the development of a new life, is also where the parents' fecundity (the physiological ability to reproduce) is translated into fertility (the number of offspring) and ultimately reproductive success. The impact of this life stage, however, extends far beyond reproduction. The prenatal

3 In biology, "choices", "decisions", and "strategies" are generally not conscious (although in some cases they can be); they are responses to the interactions between environmental and genetic influences.

period is when many environmental and physiological factors, working through the mother, impact a foetus and have consequences for subsequent development. From the perspective of life history theory, foetal growth is a measure of the resource flow to the foetus at the expense of others (parental investment). Thus, foetal growth also represents the non-genetic intergenerational transmission of phenotypes. The prenatal environment has the potential to increase the fit between the offspring's phenotype and its environment, which can provide an evolutionary advantage by increasing the probability of survival and reproduction. It is therefore likely that natural selection would favour foetal sensitivity to maternal behaviour and physiology. In the broader ethology and biology literature, these are referred to as maternal effects (Maestripieri and Mateo, 2009).

Myriad factors influence pregnancy and the vast majority of non-genetic elements work through the mother's phenotype (Wells, 2010). These include but are not limited to maternal stress, prenatal nutrition, environmental toxins and teratogens, antenatal care, prenatal bonding and early psychosocial environments (Coall and others, 2019). Across mammalian species, due to internal fertilization, gestation and lactation, interactions between the mother and offspring are close and of extended duration. Thus, mothers have the most profound influence on their developing offspring's phenotype throughout pregnancy. The mechanisms by which the maternal phenotype can affect the foetus's phenotype include her behaviour and hormone levels, nutrition, mental and physical health and size. For example, across mammalian species, smaller mothers tend to produce smaller offspring, with the most influential processes beyond genetics being other constraints such as the first pregnancy, smaller pelvic outlet or reduced nutrient supply to the foetus (Gluckman and Hanson, 2004; Godfrey and others, 1999)

Life history theory examines the relationship between the environment and life cycles from an evolutionary perspective. Therefore, foetal growth is one phase of the life cycle that is heavily influenced by the mother's own life cycle. An example of this is the role of maternal constraints — there are limits on how large a baby can be — influencing foetal growth and potentially that of future generations. Indeed, in a study of 513 low-risk pregnancies, maternal birth weight was the *only* factor that consistently predicted children's foetal and placental growth, affecting outcomes including birth weight, placental weight, placental ratio, placental surface area and placental thickness (Coall and others, 2009). Within these constraints, the idea that maternal birth weight is among the strongest predictors of her offspring's birth weight and provides a "better" reflection of the likely nutrition environment over generations, rather than the nine months of pregnancy, is referred to as "intergenerational phenotypic inertia".

Kuzawa's (2005) intergenerational phenotypic inertia model provided an adaptationist rationale for expecting the effects of prenatal malnutrition or stress to last more than one generation: when environments are stochastic over time scales greater than a generation, nine months of gestation cannot provide the foetus with enough information upon which to "predict" its own within-generation optimal growth and development. Ultimately, possibly through the epigenetic regulation of growth factors (IGF2), intergenerational phenotypic inertia provides the foetus with information, not only about the environment into which it will be born, but also about the environment into which its mother was born, and perhaps even its mother's mother, and so on, back through an unknown number of generations. Such inertia reduces the impact of short-term variations in nutrition, allowing the broader nutritional environment to influence foetal growth. Thus, associations between foetal development and adult health risks seen in the

"Developmental Origins of Health and Disease" literature may reflect longer time frames than the nine months of gestation.

Within life history theory, because the amount of resources available is always limited (e.g. time, energy) there are trade-offs between the components of fitness, and the most all-encompassing trade-off is that between current and future reproduction (Stearns, 1992). At issue is whether it would be better for an organism's lifetime reproductive success to reproduce at a given time or to wait for another opportunity in the future. The major determinants of the optimal current-future trade-off are (1) the probability of death at a given age, and (2) the availability of energy and other resources that determine parents' capacity to invest in offspring (e.g. foetal growth). When environmental conditions are risky or uncertain, with high or unpredictable mortality rates and few or uncertain resources, organisms in general, including humans, tend to reproduce early and often, maximizing the probability of reproducing, but reducing the investment in each offspring (Coall and others, 2016). Conversely, when environmental conditions are safe and predictable with low and stable mortality rates and plentiful resources, organisms tend to reproduce later and less often, investing more resources in fewer offspring. Through their phenotypic plasticity, our ancestors were able to take advantage of good times by maximizing future reproduction (investing more in fewer offspring), and to cope with bad times by maximizing current reproduction (investing less in more offspring). None of this, of course, requires conscious awareness, and the world's most disadvantaged peoples still tend to reproduce early and often (Low and others, 2009). What becomes apparent though is that the trade-off between current and future reproduction means that foetal adaptations to the effects of environmental stress on the mother can have evolutionarily adaptive consequences for her (future reproduction), but developmentally disadvantageous effects on foetal growth and development and thus postnatal health (maternal effects).

Childhood Influences on Fertility

Human childhoods are unusually long compared to other primates (Bogin, 1998), allowing for an extended period of growth and learning, while still largely dependent on care-givers for nutrition, safety, and shelter. The developmental environment during childhood can have far-reaching consequences; family settings that are nurturing and facilitate child growth and development are associated with slower reproductive strategies, while difficult childhood settings might instead increase the pace of life and initiate earlier reproduction and possibly higher fertility. Empirical studies support this view, although only in so-called WEIRD[4] (Henrich and others, 2010) contexts where childhood psychosocial stressors often have a greater impact than nutrition (Sear and others, 2019). In more resource-stressed conditions, children whose early lives are nutritionally deprived will more likely delay reproduction in aid of growth.

It is arguable that early childhood (up to age 5–7 years) is a particularly critical period that sets the child's reproductive trajectory (Belsky and others, 1991; Ellis, 2004), at least in WEIRD contexts. The empirical evidence tends to focus on girls and on age at puberty (often only menarche) as an outcome.[5] Of these studies, those that separate early childhood from older

4 Western, Educated, Industrial, Rich, and Democratic.

5 Age at puberty is often taken to indicate if an individual is on a fast or slow life history track. This assumes that all life history traits (or at least puberty, reproduction and death) are correlated, which has not yet been empirically established.

childhood show a mixture of patterns. For instance, "harsh" parenting was associated with earlier age at puberty in a contemporary US sample, but this relationship was seen in both early and later childhood, and only for girls; boys appeared to be unaffected by harsh parenting, at least with regard to puberty (Belsky and others, 2007). Indeed, we might expect different effects for boys and girls; we discuss this in more detail in the next section. Also in the US, Quinlan (2003) found that parental separation during childhood was associated with earlier puberty, first voluntary sexual intercourse and first births for women. He tested for differences depending on which childhood stage the separation occurred, and although there were always earlier timed events compared to women who had had intact families since birth, the later the separation happened the weaker the association was.

In Malaysia, a country with a transitioning rate of fertility, and a lower economic setting than the US, Sheppard and others (2014), using father absence as a proxy for adverse childhood environments, report no association with timing of menarche in either early (before age 7) or later childhood. They did, however, find a statistically significant association between father absence and younger age at first birth, but only if the father became absent during *later* childhood (age 8–15). This might be explained by the different meanings "father absence" has in different cultural and economic contexts. These studies suggest that, while it is likely that childhood developmental ecologies have an impact on fertility-related outcomes, there might not be a critical early period in this respect. Generalizations like this should be made with caution as we cannot make true comparisons when childhood adversity is operationalized differently in each study, and they all use different methods. It might be more fruitful to think of developmental environments as sums of their parts where different types of adversity affect children differently and, depending on the cultural context, may not be indicative of adversity at all. For instance, the concept of "father absence" varies depending on the local norms regarding marriage and families (Sear and others, 2019). The absence of a father may be less of an adverse condition where extended families are more common and other alloparents can compensate, rather than the typical nuclear family found in WEIRD social settings.

Adolescence and Puberty

Adolescence bridges childhood and adulthood, and is mainly characterized by puberty. As with our relatively long childhoods, adolescence is purported to be the time when we hone our social skills further, and mentally and physiologically prepare for independence in adulthood (Sapolsky, 2017). Puberty is a key milestone in adolescence and is the physiological gateway to reproduction. Genetic, physiological (e.g. nutrition) and social (e.g. psychosocial stress) factors are known to influence pubertal timing. All children need to attain a certain body size (height and weight) and reach critical hormonal thresholds in order to successfully undergo this life transition (Ellison and others, 2012). There is usually a growth spurt that occurs during early adolescence which slows down as the pubertal stage commences. There is another growth spurt toward the end of adolescence and then continued growth to final adult height. All of these processes are fuelled by nutrition and exercise. However, they are also susceptible to psychosocial stressors, largely because stress hormones impact on growth and muscle mass, and negatively affect mental and emotional states (Ellis, 2004). Indeed, chronic psychosocial stress and high levels of cortisol resulting from disrupted environments may help to entrain adaptive life history strategies (Finch and Rose, 1995).

The timing of puberty is partially associated with the timing of first sex, the birth of the first child and reproductive lifespan, at least in women. Early maturers are more likely to have sex at a younger age, have earlier pregnancies and experience later cessation of reproduction (i.e. the start of menopause) compared with late bloomers (Coall and others, 2016). This suggests that the timing of menarche is a fair indicator of the pace of the reproductive strategy being pursued. Faster life history strategies, of which early reproduction is a component, are also associated with higher mortality (Nettle, 2010). For men, however, the story is not quite as clear, and the evidence much scanter than for women (Sheppard and Sear, 2012; Bogaert, 2005). Figure 1 illustrates how sex differences in human life histories might operate. Using data from the United States, Sheppard and others (2015) show two different life history trajectories between early childhood disruption, age at puberty and final adult height (an indicator of growth during childhood and puberty), for boys and girls. For girls, weight gain happens faster and puberty starts at a younger age, while for boys growth is slower and puberty is delayed — nonetheless, in both cases, childhood disruption ultimately leads to reduced growth and shorter adult stature.

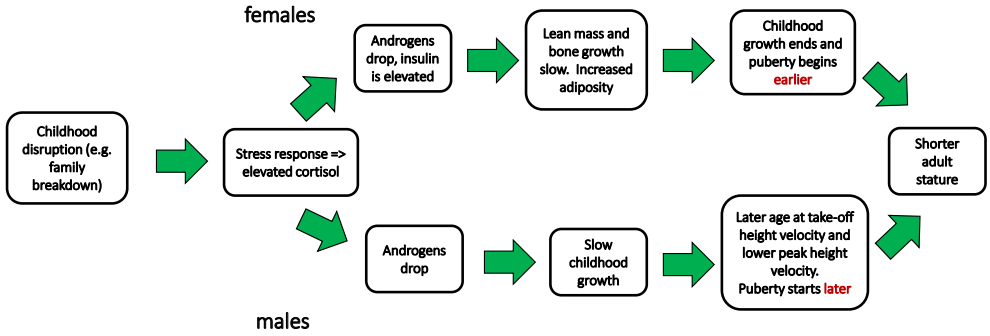

Fig. 1 Sex differences in stress responses to childhood disruption in high-income settings; different pubertal and growth pathways. All rights reserved.[6]

Demographers tend to focus on female fertility and largely ignore male fertility behaviour, but evolution predicts that men and women will not behave in the same way, and so a fuller understanding of demographic processes, especially around fertility, can only be attained by considering both sexes.

One associated adolescent trait that deserves a brief mention here is risk-taking. Risk-taking is most commonly found among adolescent boys until young adulthood (or even later). Although it is not only a male trait, it is much less frequently observed in girls (however, girls certainly can and do take risks too (Cross, 2010)). From an evolutionary standpoint, risk-taking is often thought to arise as an outcome of male-male competition (Chisholm, 1999). These are high-risk (e.g. failure, injury, death) high-payoff (reproductive and/or parenting opportunities) reproductive strategies. This behaviour is especially prominent during adolescence when young men are preparing for adulthood.

6 Reproduced from Sheppard et al. 2015. 'Childhood family disruption and adult height: is there a mediating role of puberty?', *Evolution, Medicine, and Public Health*, 1: pp. 332–42. By permission of Oxford University Press on behalf of the International Society for Evolution, Medicine & Public Health. For commercial reuse queries, please contact journals.permissions@oup.com.

After men become fathers their testosterone levels recede as they turn to parenting rather than mating behaviour (Gettler and others, 2011). Young men want to have sex and will compete to attract young nubile women. It is a dual-purpose strategy: there is competition with other males (such as outwitting each other or simply fighting), and also showing off prowess and skill to the sought-after females. There are, by definition, costs (sometimes very high) to risk-taking. The whole point of the risk is that if it goes wrong, it can cost the young man's life, result in injuries, or at the very least, cause embarrassment and the loss of status. Risk-taking requires the careful balance of risk; it needs to be risky enough to be impressive (ultimately increasing reproductive success), but not so risky that it results in no fitness legacy at all. Accidents happen when the risk is miscalculated, or unknowns are not factored in. Demographers who are interested in male-biased sex ratios and male mortality more generally would do well to understand male risk-taking strategies (Schacht and Borgerhoff Mulder, 2015). Seldom is the relationship between mortality and fertility more conspicuous than when witnessing male-male competition for females. Note that risk-taking is not the only male competition strategy available; not all young men fight and show off, at least not in life-threatening ways. Some men are more suited to other tactics such as displaying good parenting skills, being a good provider, exhibiting creative talents, etc. The strategy adopted will depend on the young man's resources, both physical and material (i.e. his environment).

Demographers are often interested in the differences in fertility and mortality rates between countries within the context of the demographic transition. In high-income, low-fertility countries, the constraints are different to those settings where nutrition and other basic needs for survival are prioritized. Where access to sufficient good-quality nutrition is scarce, as is often the case in low-income countries, the basic weight requirements to enable pubertal processes to begin are limited by food stress. As such, girls from these areas tend to have later pubertal development than girls from high-income settings where the opposite is true. In high-income environments, socioeconomically disadvantaged girls have more access to highly calorific nutrition (junk food) which might not be healthy, but leads to rapid weight gain and early onset of puberty (Coall and others, 2016).

The association between psychosocial stress and pubertal timing has been shown empirically from many studies in so-called Western cultures. Due to meagre evidence in other settings, it is much less clear how this association works in lower-income settings where nutrition is a more salient problem. Puberty data are difficult to collect and have not been the focus of many longitudinal studies in less affluent countries, which limits research in this area. Nevertheless, there are a few studies which provide some evidence for a different trend in low- and middle-income contexts (Sear and others, 2019). In Malaysia, young women from father-absent homes were found to commence puberty (measured as first menstruation) no earlier than those from dual parent families (although they did marry younger) (Sheppard and others, 2014). It is likely that the mechanism driving the association between family background and age at puberty in lower income contexts has more to do with paternal provisioning (household resources) than in higher-income settings, where psychosocial stress is more of a problem than resource stress. Along similar lines, no evidence to support the hypothesis was found for young women in South Africa who were raised in father-absent families, compared with those where the father was present until age six and age at menarche (Anderson, 2015). There was, however, a significant association between father absence and age at first sex and pregnancy. These

findings highlight the importance of understanding how contextual factors inform hypotheses derived from theories of human behaviour, such as life history theory.

Reproduction and Fertility

In demography, fertility is defined as the number of offspring an individual produces in a lifetime, and is usually measured for women. The total fertility rate (TFR) is a population-level measure of the average number of babies born to women of reproductive age (usually 15 to 49 years) in a given population. In addition to earlier declines in mortality and fertility, fertility in Europe has fallen dramatically over the last fifty years, along with decreasing mortality rates. This process is known as the "demographic transition to low fertility", and is thought to be driven by economic development, as there is a strong correlation between country-level economic development and reduced fertility worldwide. Worldwide fertility has dropped from around five babies per woman in 1960 to half of that in 2015 (The World Bank 2018). In OECD member states (mostly high- and middle-income countries), the TFR has shifted from 3.2 to 1.7 in the same period, i.e. below replacement level (for the population to remain stable the TFR would need to be 2.1).

Demographers spend much research effort trying to understand this transition. It is a very well-described phenomenon, but not fully understood in terms of why it occurs. Evolutionary theorists also try to make sense of the counterintuitive pattern that apparently higher wealth is associated with *lower* fertility, when Darwinian reasoning would predict more resources equated to *more* offspring, and thus increased genetic fitness. The story is complicated and somewhat beyond the scope of this chapter, but part of the problem is due to the conflation of population measures with individual level outcomes (Borgerhoff Mulder, 1998; Mattison and Shenk, 2019). In other words, not all people from rich countries are in fact rich, and when considering individual-level wealth, the relationship is actually non-linear, with the very wealthy and the very low-socioeconomic groups both exhibiting relatively high fertility. This is explained by the observation that mortality also varies by socioeconomic status. Poorer people, even in relatively wealthy settings, tend to have lower life expectancy than more advantaged groups — even in London there is a more than twenty-year gap in life expectancy across neighbourhoods (Cheshire and O'Brien, 2015). In such environments it pays, in fitness terms, to produce more offspring earlier to offset the higher risk of death.

How can applying Tinbergen's ontogenetic reasoning help us to understand fertility? A life history approach can help elucidate how early life conditions impact on the timing of first births, the timing of higher parity births, the decision to reproduce at all, and how many children to have in total. Despite the rather large body of evidence available on the developmental antecedents of puberty, there is relatively little on how development affects fertility (Coall and others, 2016). Furthermore, the evidence that we do have is not consistent across studies. Using data from the UK 1958 birth cohort, (Nettle and others, 2011) showed that women whose childhoods included: low paternal involvement; being breastfed for only a short while; frequent household moves; and being separated from their mothers had their first pregnancies at younger ages than women from non-disrupted backgrounds. Also, the more adverse conditions a woman had experienced, the younger the age at pregnancy (i.e. the effects are accumulative). Another study in the UK also found that poor childhood health was associated with earlier first births, even after accounting for the socioeconomic position of the family during childhood, and the

women's education (Waynforth, 2012). These studies looked at the timing of reproduction rather than total reproductive output or fecundity; other studies that do investigate markers of fertility have not been able to replicate these associations. Two studies from the US found that women who experienced adverse childhoods had irregular menstrual cycles, menstrual amenorrhea and difficulty conceiving (Jacobs and others, 2015; Allsworth and others, 2007).

While pubertal timing is certainly linked to reproductive timing, it is not the whole story. People's life history trajectories are not determined from early life, and our evolved ability to adjust our responses to the environment in flexible ways means that these relationships are only partly co-dependent, and partly independent processes (Bornstein, 1989). In other words, childhood environments may mediate the relationship between people's adult environments and their reproductive decisions. Indeed, the early environment does not "program" development, and only some who endure stressful childhoods go on to adopt an accelerated strategy of reproductive timing. Research using the 1958 UK birth cohort showed differences in reproductive timing between never- and ever-married women: early life adversity was associated with earlier pregnancy in never-married women, but delayed pregnancy in married women (Harville and Boynton-Jarret, 2013). Evolutionary demographers interested in fertility should consider the complex relationships throughout the life course, and be aware of likely mediating and moderating factors between early life, adult environments and childbearing (Coall and others, 2016).

What are the mechanisms? Early life and eventual reproduction are far apart in an individual's lifespan. If childhood experiences influence fertility decision-making, then how does this operate? One idea is that having more children is a "predictive adaptive response" (PAR) to early childhood stress. The main problem with this argument is that it assumes that childhood conditions are good predictors of future ones. In other words, that we live (and have always lived) in environments that change little over the life course. This is implausible. Another hypothesis is that early life adversity has a tangible impact on that person's health and that this is carried throughout life (e.g. stress reactivity, weight gain, growth), and is associated with higher mortality. Empirical evidence has so far revealed frustratingly little. In a longitudinal study of women born in Newcastle-upon-Tyne in 1947, low socioeconomic status (SES) and poor housing conditions at birth were both associated with younger age at first birth, and poor housing (though not low SES) was associated with higher completed fertility. Given the long duration between birth and the woman's reproduction, a number of potential mediating factors were tested, such as birth weight, childhood illness, age at puberty, etc., but these had little influence on either reproductive outcome. Further, consistent with the idea of a life history strategy, when both outcomes were included in the model, age at first birth completely mediated the relationship between poor housing and total fertility (Sheppard and others, 2016). This is a useful investigation; however, it was a small study from a non-representative population — larger studies of these processes will be more revealing. Similarly, early research looking at telomere length as a marker of health in humans also does not reveal much — a review of the literature finds only a weak correlation between early life adversity and telomere length (shortened telomeres are a sign of ageing) (Pepper and others, 2018).

Few empirical studies have examined the relationship between childhood conditions and fertility outcomes in pre-demographic transition societies, and those that do report mixed results. A commonly-used indicator of childhood insecurity is father absence, and is often the

focus of demographic studies among foraging peoples. For instance, the death of a father had no impact on fertility outcomes for sons or daughters among either the Venezuelan Ache or the Bolivian Tsimane foragers (Hill and Hurtado, 1996; Winking and others, 2011). Among the Belizean Maya and Paraguayan Ache males, however, father absence due to divorce was associated with delayed age at reproduction, possibly because father-absent sons were deprived of paternal investment leading to difficulty acquiring mates (Waynforth and others, 1998).

Menopause and Mortality

Menopause marks the end of a woman's reproductive years, and is of substantial interest to demographers because of the associated reduction in female mortality advantage after menopause, changes in disease risk profiles (Hill, 1996) and its inverse association with all-cause mortality (Jacobsen and others, 2003). At the same time, menopause is grounded in ontogeny. Menopause is defined as one year after a woman's final menstrual period and results from the gradual depletion of ovarian follicles that begins during foetal life and is complete at menopause. Therefore, events during development, such as age at sexual maturity, pregnancy, childbirth, contraceptive use and number of offspring influence the number of ovulatory menstrual cycles, the rate of ovarian follicle loss, and the length of reproductive lifespan (Bjelland and others, 2018; Gold and others, 2001). Several of these life history events have been associated with age at menopause.

Human life histories are characterized by a long lifespan and, particularly in women, a long post-reproductive lifespan after menopause. Originally, evolutionary biologists saw menopause and the long post-reproductive lifespan as unique human characteristics that may be usefully understood from an evolutionary perspective (Williams, 1957), although it is now known that menopause does exist in some long-lived species such as killer whales (Brent and others, 2015). Williams (1957) proposed that it may be beneficial for women to stop reproducing earlier, reducing their exposure to pregnancies of high risk to maternal and child survival. This would ensure mothers were more likely to survive to raise their existing family, in turn increasing survival. Data from natural fertility populations has not supported the proposed benefits to fitness of stopping reproduction early (Hill and Hurtado, 1991, 1996; Rogers, 1993). However, Williams' article inspired a new field of investigation examining the impact grandparents have on survival and reproduction in families that continues to grow today (Coall and Hertwig, 2010) and has been discussed in several of the preceding chapters in this volume. Indeed, in our ageing populations, understanding the roles that longevity, ageing and the post-reproductive lifespan play in the human life history may be particularly valuable to active ageing and positive engagement with family, community, leisure activities and personal well-being.

At an ultimate level, ageing, senescence and menopause have long posed a challenge for the evolutionary perspective. Why, if the unit of selection is the individual, should ageing and menopause exist? Clearly they would appear to be bad for genetic fitness (Medawar, 1952). Why haven't they been selected against? Moreover, as they occur after the reproductive lifespan is complete, there is little or no opportunity for natural selection to act upon them. These are valid points; however, they neglect the life-cycle focus of life history theory. As we have discussed, life history strategies are sensitive to resource availability and stress, creating trade-offs between the components of fitness (e.g. reproduction and maintenance). If post-reproductive adults, many of whom are likely to be grandparents, can invest in subsequent generations, thus influencing

reproduction, growth, development and survival, the post-reproductive lifespan can be seen and moulded by natural selection.

Theoretical perspectives including the Grandmother Hypothesis, Cooperative Breeding Hypothesis, and Embodied Capital Hypothesis and their variants, propose that older post-reproductive individuals contribute resources to children and grandchildren that change their life cycles (Voland and others, 2005). That is to say, help from alloparents, such as grandparents, provides the resources that influence development in subsequent generations. Empirical studies have investigated the impact grandparent presence has on fitness measures, such as the fertility of their children and grandchild survival. The vast majority of this research has been conducted in natural fertility and historical human populations, and shows that the presence of grandmothers and the help they provide is often associated with increased grandchild survival (Sear and Mace, 2008).

Perhaps surprisingly, much less research has focused on the association between interactions with grandparents and grandchild outcomes in contemporary post-demographic transition populations. In these populations with low mortality and fertility, the outcomes are likely to reflect the emotional, social and material resources necessary to compete in those environments. The correlational research does support an association between grandparental involvement and improved psychological adjustment, mental and physical development and educational outcomes (Sear and Coall, 2011). Thus, consistent with theoretical perspectives mentioned above, downward resource transfers, which may be particularly evident through grandparenting, may confer a selective advantage to subsequent generations that drive human longevity and ultimately extend the human life cycle for both males and females.

Changes in the life cycle of human ancestors also suggest that post-reproductive lifespans and childhood are linked. In examinations of the fossil evidence, the extended post-reproductive lifespan in women and the long, slow growth period of childhood development appeared at roughly the same time in human history (Bogin, 1997). Both of these unique features of the human life history — most likely linked by a general increase in longevity — are likely to have evolved together as a self-reinforcing unit (Carey and Judge, 2001). As long ago as *Homo erectus* (1.9 million to 143,000 years ago), longevity estimates suggest they were living beyond 60 years of age, some fifteen years after menopause. This suggests that human longevity has a much more distant foundation, well beyond the increases in lifespan evident from recent advances in medicine and technology.

From an evolutionary perspective it has been hypothesized that helping behaviour, which brings additional resources to an individual within and beyond the family, ultimately developed from ancestral parenting and grandparenting. In turn, this helping behaviour may have contributed to extending the human lifespan. Recent evidence suggests that among elderly people, helpful grandparents, parents, and unrelated community members experience increased survivorship compared to individuals who do not help. Using data from the 516 participants in the Berlin Ageing Study, it was found that helpful grandparents who looked after their grandchildren survived five years longer than non-caregiving grandparents or non-grandparents (Hilbrand and others, 2017). Likely mechanisms that may link grandparental investments and reduced mortality include various measures of improved grandparental health and well-being. To date, longitudinal studies with more power to conduct within-individual analyses that more closely approximate causal relationships have found mixed results. Generally, however, health

is unable to completely account for the relationship. With improved measures and statistical analyses, potential mediating pathways and causal relationships can be explored further in the association between grandparental involvement and longevity (Coall and others, 2018).

Conclusion

We hope we have demonstrated that thinking about human demographic behaviour in light of Tinbergen's four explanations, and, in particular, considering ontogeny, is useful to demographers. Evolutionary theory adds value to demographic studies by underpinning ideas, hypotheses and empirical studies with a prediction-generating theory. Ontogeny is one of Tinbergen's two proximate explanations for understanding how and why human demographic behaviour is adaptive. The genetic background and developmental environment are both crucial for shaping the individual's reproductive trajectories and mortality expectations. It is crucial to consider different aspects of the childhood environment (e.g. psychosocial stress and nutrition) and the potential interactions between them. We hope this chapter has provided some answers, but more importantly, new questions and research opportunities for demographers. Demographers take a keen interest in both fertility and mortality, and when these topics are understood within an evolutionary life-history framework, a deeper understanding of the biological processes around death and reproduction can provide a fuller explanation of human demographic behaviour.

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11. How It Works: The Biological Mechanisms that Generate Demographic Diversity

Virginia J. Vitzthum

Tinbergen (1963) proposed that a complete understanding of any behaviour requires knowledge of its function, evolutionary history, developmental history and mechanism of operation. This chapter is largely concerned with gaining some insight into the nature of the biological mechanisms generating variation in human fertility, and, consequently, demographic diversity within and across populations. My inquiry is informed by life history theory, an analytical framework within evolutionary theory for studying maturation, reproduction and aging and the associated behavioural and physiological mechanisms underlying the allocation of resources to these processes. Different allocation patterns are referred to as life history strategies (LHSs) and are subject to natural selection. Biological mechanisms can be usefully conceptualized as a set of suitably timed strategic responses to signals. I discuss this and other ideas about the mechanisms that underlie the implementation of LHSs, and introduce the concepts of "ecomarker" and "the physiological fallacy".

Drawing on empirical studies and theoretical models, I examine some intriguing features of human reproductive physiology that are directly relevant to demographic research in both low- and high-fertility populations. Several points, some contrary to common assumptions, emerge from this inquiry. For example: (1) the marked within- and between-population variation in many features of female reproductive functioning challenges the widespread assumption that there is a universal "normal" human biology. (2) The most likely outcome of a human conception is early loss. This unseen natural selection in the production of offspring may hamper investigations of hypothesized associations of post-natal reproductive success with resources or with offspring quality, even in low fertility populations. (3) Competition between incompatible but essential functions shapes the timing and operation of various mechanisms. Some biological, psychological and behavioural functions cannot readily co-occur. Of necessity, successful LHSs must juggle such incompatibilities regardless of the abundance of energy and other resources, therefore some reproductive mechanisms may not depend upon (or be responsive to) energy availability. (4) Biomedically, the absence of ovulation is typically considered a pathology (and in some cases it may be). However, from a life history perspective, each option of ovulating/not ovulating is a fork in the reproductive road at which there is a strategic decision to continue engaging in the possibility of reproduction or to forego the current opportunity. Not ovulating in a given cycle can be the best strategy for optimizing lifetime reproductive success.

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> It would be instructive to know [...] by what physiological mechanisms a just apportionment is made between the nutriment devoted to the gonads and that devoted to the rest of the parental organism ...
>
> — Fisher (1930)

> Evolution is a tinkerer.
>
> — Jacob (1977)

Introduction

What Must Be Known If We Are to Have a Thorough Understanding of Human Demographic Diversity?

Posed this way, the answer to the question would seem to be, "Everything!", and the goal appears unachievable. Recognizing this, we choose instead to focus on a single feature — perhaps age at menopause or marriage practices or hormonal concentrations. This specialization is necessary and productive but risks losing sight of the bigger questions that first piqued our curiosity.

Nearly sixty years ago, Tinbergen (1963) sought to mitigate this risk within the field of ethology[1] by proposing that a comprehensive and coherent understanding of behaviour arises from integrating the answers to four complementary questions. His framing proved to be an enduring guide for the thorough study of any feature of an organism (Bateson and Laland, 2013). Paraphrased for more general application, Tinbergen's four questions are:

- What is the function of the feature? (i.e. what is its current and/or former utility?)
- How has the feature evolved over time? (i.e. what is its phylogeny?)
- How does the feature develop in an organism? (i.e. what is its ontogeny?)
- What are the mechanisms that produce the feature? (i.e. how does it work?)

This chapter is largely concerned with the physiological mechanisms generating variation in human fertility and, consequently, demographic diversity across populations.[2] Like most organismal features, individual fertility is variable, but the mean and limits of this variation are characteristic of the species and subject to natural selection. Time, resources, competing demands and the physical constraints of biological processes all limit individual fertility even in the most successful members of a population and even in the most benign environment. In addition to physiological mechanisms, there are psychological, behavioural, social and cultural pathways that generate variation in human reproductive output.[3] Many of these pathways impact fertility via biological mechanisms.

Reproductive mechanisms can be delineated and studied without recourse to evolutionary theory or a consideration of Tinbergen's other questions. However, to do so would be to miss understanding *why* these mechanisms operate as they do. Neither is our understanding well-served by simply assuming that all features (or variants of a feature) are evolved adaptations; there is plenty of evidence to the contrary (Williams, 1966).

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1 A glossary of terms is provided at the end of this chapter.

2 For additional discussions of evolutionary demography using Tinbergen's framework see the following chapters in this volume: Mace on function, Jones et al. on phylogeny, and Sheppard and Coall on ontogeny.

3 For examples see the following chapters in this volume: Blurton-Jones, Borgerhoff Mulder, Dillion et al., Lee and Boe, and Tuljapurkar.

Fully understanding the causes of biological variation necessarily demands incorporating an evolutionary perspective (Tinbergen, 1963; Dobzhansky, 1973). Doing so, however, does not give us leave to accept any seemingly plausible story of the adaptive advantage of some trait or another (Williams, 1966; Gould and Lewontin, 1979; Caro and Borgerhoff Mulder, 1987). Without an understanding of mechanism, we risk spinning "just-so stories" (explanations relying more on our imaginations or preferences than on empirical evidence). Rather, our plausible conjectures are better seen as starting points for generating specific and testable hypotheses about how a thing works and what function it serves.

Mace (in this volume) discusses three ways to test hypotheses about function: experimentation, comparative studies of individuals within populations and comparative studies across populations or across species. These approaches are equally applicable to investigating mechanisms. Ideally, hypotheses about mechanism are addressed by demonstrating exactly how a purported causal agent is linked to an observed outcome. *How* questions about mechanism are not the only ones worth asking, and they are very rarely the first to be asked. But they must be answered if we are to thoroughly understand *why* human fertility, mortality and health vary, whether due to immediate circumstances or as a consequence of evolutionary processes or, most likely, the dynamic interaction of both.

Evolution through natural selection is often portrayed as a winnowing process that favours individuals with "the best" features for survival and reproduction, a description that gives the faulty impression that after many generations, most members of a species are nearly identical when it comes to basic functions such as the reproductive system. This faulty impression readily lends itself to the false assumption that there is not much variation in the physiological mechanisms enabling human reproduction (and thus, such mechanisms appear unlikely to be a significant cause of fertility differentials within and between populations).

To the contrary, there is substantial and compelling empirical evidence of within-species variation in biological mechanisms. Even identical genotypes can produce different phenotypes (variants in morphological, physiological, behavioural and psychological features), an outcome of developmental and epigenetic processes that facilitate individual adaptation to the environments encountered from conception through death.

Because environments change over time and space, individuals possessing a genotype that adjusts phenotype according to shifting conditions can have an evolutionary advantage over conspecifics who express only one phenotype, no matter the conditions encountered. The capacity for a genotype to express a variety of phenotypes is known as "phenotypic plasticity" and the range of possible phenotypes for a given genotype is referred to as the "norm of reaction" (Via and Lande, 1985; West-Eberhard, 1989, 2003; Stearns, 1989; Vitzthum, 1990; 2003; Scheiner, 1993). Such plasticity can also be disadvantageous (Dewitt et al., 1988), a reminder of the importance of testing specific hypotheses. Nonetheless, the evidence for phenotypic plasticity across a wide range of taxa and phenotypes supports its importance as an adaptive mechanism (West-Eberhard, 2003), and the analyses by Jones et al. (in this volume) suggests that phenotypic plasticity plays a significant role in generating human demographic diversity.

Individual adaptation is achieved through genetic, epigenetic and ontogenetic processes shaping the organism's phenotypes. Biological evolution is a consequence of natural selection acting on these phenotypes. Many biological mechanisms, including those associated with reproduction, are flexible and exhibit a dynamic response to external conditions. This capacity can cause variation

in fertility across the many physical and social environments in which humans live. Examples of this flexibility and the potential impact on demographic diversity are discussed in this chapter.

On a broader note, failing to understand the how and why of biological features can lead to pathologizing natural variation and reifying cultural constructs of what is normal or superior. This error sometimes takes the form of assuming that the average and distribution of some feature found in one's own population is universally true of, or an appropriate norm for, the entire human species (Mead, 1947; Vitzthum, 2020). But if, like me, your native population is WEIRD (Western, educated, industrialized, rich, democratic), then it represents only 12% of the world's current population (Henrich et al., 2010), and bears little resemblance to the conditions typical of human history during the many hundreds of thousands of years before humans invented agriculture. To help overcome this myopia, there is a fifth question worth adding to Tinbergen's four: How do the features of these mechanisms vary within and across human communities worldwide? Techniques developed over the past forty years have allowed us to begin to address this question; some of the answers are considered throughout this chapter.

Why Is the Study of Biological Mechanisms Useful to Demographers?

The answer, in brief, is that the identification and specification of biological mechanisms expands and enriches our understanding of how demographic variation is generated. As a case in point, an ever better grasp of mechanisms has been, and will continue to be, directly relevant to improving models of the proximate determinants of fertility.

Demographers' investigations of how fertility, mortality and migration impact population structure brought to light the marked variation in these processes across human populations. Not long ago, most explanations for this variation concerned the influence of sociocultural and economic factors, giving little attention to the biological processes involved in the production of offspring and the maintenance of a living body. This focus reflected demography's historical roots (Sear et al., 2016; Kreagar in this volume) and the assumption that there was little variation across human populations in such biological processes. As a consequence, much has been learned about the changes in fertility associated with varying sociocultural and economic factors (Balbo et al., 2013; Uggla in this volume), but relatively little about how these factors might play out through biological mechanisms. Even so, over time and for a variety of reasons, demographers' growing attention to biological processes and evolutionary biologists' keen appreciation of demographers' population data has prompted novel and productive collaborations to address this gap (Sear et al., 2016; Kreagar in this volume; Low in this volume).

Davis and Blake (1956) proposed the first formal demographic framework identifying a finite set of behavioural and biological mechanisms ("intermediate fertility variables") through which all other possible factors (sociocultural, economic, environmental, behavioural) must act in order to influence fertility. Of their eleven direct factors, only two ("foetal mortality from involuntary causes" and "fecundity or infecundity, as affected by involuntary causes") are biological variables, and the latter of these included the entire morphological and neuroendocrinological mechanisms of the ovarian cycle, conception and implantation. The authors lamented the absence of relevant data that would allow an assessment of the contribution to fertility of either of these two biological intermediate fertility variables.

In 1978, Bongaarts reformulated Davis and Blake's work as a set of eight "proximate determinants of fertility" and proposed a quantitative approach for estimating the contributions

of four determinants (#1–4 in Table 1) to a population's total fertility rate. Of these four, the only biological factor is lactational infecundability. Its inclusion in Bongaarts' analyses was a consequence of the by-then large body of data demonstrating that breastfeeding can suppress ovulation (Gioiosa, 1955; Perez, 1971; Vitzthum, 1994) and thereby contribute to inter-population variation in fertility (these data had not yet been collected at the time of Davis and Blake's work in 1956). Bongaarts also argued that the other four proximate determinants (#5–8 in Table 1) are *not* important contributors to differences in fertility between populations, but allowed that the three biological factors might be significant if venereal disease were present.

Through the years since, Bongaarts' model has been critiqued and revised (Reinis, 1992; Wood, 1994; Stover, 1998) including a recent "tune-up" (Bongaarts, 2015). His quantitative approach has demonstrable utility in addressing certain kinds of demographic questions, and his work continues to be the most widely applied demographic model of the proximate determinants of fertility. However, Bongaarts (1978, 2015) estimated the contribution to fertility of only one of the four biological proximate determinants, neglecting the others. This may explain why some analyses using this method could not adequately account for observed between-population variation in fertility (Wood, 1994). Also, at least some of the omitted biological determinants are likely to generate fertility differences *between individuals*, a possibility that can't be addressed using Bongaarts' approach.

TABLE 1. Proximate Determinants of Fertility

I. Exposure factors:
  1. Proportion married
II. Deliberate marital fertility control factors:
  2. Contraception
  3. Induced abortion
III. Natural marital fertility factors:
  4. Lactational infecundability
  5. Frequency of intercourse
  6. Sterility
  7. Spontaneous intrauterine mortality
  8. Duration of the fertile period

Source: Bongaarts (1978)

Beginning in 1988, Maxine Weinstein (a demographer), Kenneth Campbell (an endocrinologist) and James Wood (a bioanthropologist) proposed and refined a new model, "the proximate determinants of natural fertility" (Table 2) (Campbell and Wood, 1988; Wood and Weinstein, 1988; Weinstein et al., 1990; Wood, 1994). This framework can accommodate variation among populations, among individuals within a population and within particular individuals (e.g. over time). Moreover, their approach explicitly models the contributions of a comprehensive, but nonetheless small, set of behavioural *and* biological proximate determinants. This attention to biological mechanisms has revealed, for example, that one of the most important potential sources of inter-population variation in fertility is intra-uterine death. Although most pregnancy losses are undetected (except with a laboratory test), this needn't mean such loss is inconsequential for population structure (see discussion below in "Vote Early, Vote Often: Early Pregnancy Loss").

These theoretical advancements in demographic models of fertility are attributable, in part, to a burgeoning awareness, fuelled by empirical and theoretical studies alike, of the variability, complexity and flexibility of the underlying biological mechanisms that make reproduction possible.

TABLE 2. Proximate Determinants of Natural Fertility

I. Exposure factors:
1. Age at menarche
2. Age at menopause
3. Age at entry into sexual union
4. Age at onset of pathological sterility

II. Susceptibility factors:

Fecundability factors:
5. Length of ovarian cycles
6. Probability of ovulation
7. Duration of the fertile period
8. Frequency of insemination
9. Probability of conception from a single insemination in the fertile period
10. Probability of pregnancy loss
11. Length of the non-susceptible period following foetal loss
12. Length of gestation resulting in a live birth
13. Duration of post-partum infecundability

Source: Wood (1994)

The Take-Homes

Before delving into the details, these are the main arguments developed in this chapter regarding the biological mechanisms regulating human reproduction.

Core themes in demography (the causes of variation in fertility and mortality) map well with those of life history theory (LHT). LHT is an analytical framework within evolutionary theory for studying maturation, reproduction, ageing and the associated behavioural and physiological mechanisms underlying the allocation of resources to these processes (Promislow and Harvey, 1990; Stearns, 1992; Roff, 1992; Charnov, 1993; Vitzthum, 2008a; Hill in this volume; Low in this volume). Because there are unavoidable trade-offs in the allocation of finite resources (e.g. time, energy, nutrients) over a lifetime, different allocation patterns (referred to as 'life history strategies' [LHSs]) can produce variation in the quantity and quality of offspring, thus generating opportunities for natural selection and adaptation.

One useful approach for organizing an inquiry into the physiological mechanisms associated with LHSs is to conceptualize bodily functioning as a set of suitably timed strategic responses to signals. In subsequent sections, I will use empirical studies and theoretical models of human female reproductive functioning to discuss this and other ideas about these mechanisms. Rather than attempting to catalogue all of the mechanisms that contribute to the production of offspring, my aim is to gain some insight into the nature of these mechanisms — their shared properties — with an eye towards developing research questions and strategies that help us to

search for the physiological keys to human demographic diversity beyond the light from the nearest lamp post. The principle take-home points from this inquiry are summarized below.

(1) Some mechanisms rely more on the detection of change in a condition than on the assessment of a static condition. Because a life history strategy (LHS) is a series of allocation decisions, its success depends both on the relative amounts of resources distributed to competing demands and on strategic timing. Strategic timing necessarily relies on the detection/recognition of reliable signals of endogenous (internal, somatic) and exogenous (external, extra-somatic) current and changing conditions. In some instances, the *change* in conditions may be more readily detected and hence a more salient signal than the specific state of the condition. For example, regardless of the absolute concentration of progesterone at its peak during the menstrual cycle, it is the drop in progesterone that prompts a cascade of biological changes that characterize the ending of one cycle and the beginning of the next.

(2) LHSs are significantly constrained by factors other than energy availability, therefore some mechanisms may not depend upon (or be responsive to) energy availability. Trade-offs in the allocation of finite resources are unavoidable throughout the course of an organism's life. Because life demands energy, considerable research has rightly been devoted to ascertaining when and how energy is apportioned to somatic versus reproductive functions, and to the competing demands within each of these arenas. This emphasis on resource distribution has, however, overshadowed the limitations imposed on LHSs by constraints other than energy availability. For example, there are physical limits to the pace at which biological processes can proceed, and some essential biological, psychological and behavioural functions cannot readily co-occur. Of necessity, successful LHSs must juggle such incompatibilities regardless of the abundance of energy and other resources.

(3) There are often multiple mechanisms involved in the implementation of a given LHS; these mechanisms are connected and communicate ("cross-talk"). Of necessity we speak of "the ovary" or "the reproductive system" as if these were autonomous entities disembodied from the organism. But often an organism must execute compatible responses to a given signal across various bodily components. Such co-ordination is not necessarily reliant on identical responses to a given status of the signal (e.g. a specific hormone concentration), but may be accomplished, for example, through the presence of similar or different types and/or numbers of receptors in the target cells. These differences in signal recognition allow the same absolute concentration of a given hormone and/or the same change in that concentration to elicit distinct yet coordinated responses in these targets. A focus in human research on the absolute concentrations of hormones reflects what we are able to measure, but may not be all that should be measured to understand the mechanisms that generate demographic diversity.

(4) Physiological mechanisms may be conditioned on circumstances experienced during pre-natal and pre-adult development. Boas' measurements in the early twentieth century of the morphology of US immigrants and their children suggested the influence of early environments on subsequent adult biology, but the mechanisms were a mystery (Boas, 1911). Some fifty years later, physiological studies of humans native to harsh environments (high altitude, the Arctic) strengthened the arguments that adult functioning depended to some degree on conditions during development (Lasker, 1969; Leslie and Little, 2003). Subsequent epidemiological studies in industrialized countries provided further evidence of these links (Barker, 1990), and theoretical

and empirical advancements in epigenetics and developmental biology opened a window into the mechanisms by which the environment can alter gene expression (Kuzawa and Thayer, 2011). This current state of knowledge prompts testable hypotheses regarding the nature of the neurohormonal mechanisms that regulate physiology. In particular, a physiological response to a specific signal may be essentially constant for all members of a species or it may differ depending upon prior exposure. For example, the responsiveness of adult reproductive functioning to resource scarcity is highly variable between individuals and populations, perhaps as a consequence of differences in resource availability during development (Vitzthum, 1990, 1997, 2001).

(5) Ovulation is optional. Although necessary for conception, ovulation is not an automatic feature of a menstrual cycle. In several studies of healthy women, anywhere from 10%-40% of the sample did not ovulate in a given cycle (Vitzthum, 2009). Biomedically, absence of ovulation is typically considered a pathology (and in some cases it may be). But, from a life history perspective, each option of ovulating/not ovulating is a fork in the reproductive road at which there is a strategic decision to continue engaging in the possibility of reproduction or to forego the current opportunity. In some contexts, not ovulating in a given cycle can be the best strategy for optimizing lifetime reproductive success.

(6) Pre-natal selection of offspring may swamp post-natal differences in offspring quality and/or parental investment. The most likely outcome of a human conception is natural loss (Roberts and Lowe, 1975; Holman and Wood, 2001; Vitzthum, 2008b). At least half of these losses occur before implantation and another quarter in the subsequent five to seven weeks. It has long been assumed that the vast majority of these early pregnancy losses (EPL) are due to genetic errors in the conceptus. However, there is now evidence that a substantial portion may reflect a maternal LHS to delay reproduction if the current conditions are sufficiently inadequate for producing a live birth (Weinberg et al., 1994; Nepomnaschy et al., 2006; Vitzthum et al., 2009a). Thus, rather than being difficult for humans to conceive (as some have argued), it is now known that human fecundity (the capacity to conceive) is many times higher than human fertility (production of a live birth). This unseen pre-natal selection in the production of offspring may hamper investigations of hypothesized associations of post-natal reproductive success with resources or offspring quality.[4] Much of the selection has already occurred (i.e. the differential quality and/or subsequent survival among those concepti that have survived to birth is relatively small). In truth, everyone's children really *are* all above average.

Why Is Life History Theory Useful for Understanding the Mechanisms that Generate Demographic Variation?

Malthus (1798) envisioned an unflagging human reproductive system, excepting disease or damage. He was mistaken. For example, the absence of ovulation while intensively breastfeeding a young infant is not a failure of the reproductive system, but rather the adaptive response of a physiological mechanism shaped by natural selection to reduce the risk of premature investment in the next offspring (Short, 1976).

4 In an evolutionary context, higher quality offspring simply means those offspring having attributes (for example, larger size) that usually confer a relatively greater likelihood of survival.

Life history theory offers plausible and, ideally, testable evolutionary explanations for when and why reproductive effort varies. Not reproducing in some contexts may be a life history strategy (LHS) that could yield a higher lifetime reproductive success than would obligate reproduction at every opportunity.

Variation in LHSs within and across populations and generations suggests there are mechanisms for the flexible implementation of LHSs. This inference prompts a cascade of interesting questions about these biological, and perhaps adaptive, mechanisms that create variation in human birth, death and the experience in between. For example:

- What sorts of mechanisms are likely to underlie implementation of LHSs?
- What signals prompt allocating resources to one of several competing demands?
- Are all allocation decisions typically transient or might some be permanent?
- How are competing signals resolved?
- How does maturation stage interact with these signals?
- What are the constraints on LHSs?

Time is arguably the greatest constraint on LHSs. Because all individuals die, time is a scarce commodity. Time cannot be stored, created, foraged, harvested or shared. Social co-operation and/or multi-tasking may or may not mitigate against the scarcity of time, depending on the circumstances (e.g. nine women cannot make a baby in one month). Mortality schedules (the population-specific risk of death for each age or stage of life) express the length of time available for maturation (growth and development) and reproducing, and the pace at which these fundamental biological processes must be accomplished (Stearns, 1992; Charnov, 1993). Natural selection favours those organisms that respond to the scarcity of time with suitably strategic timing of their allocation decisions. For example, if mortality risk is low and the average life is long, one can afford to postpone the transition to reproductive investment until later, making use of the extended pre-reproductive period to grow larger, build knowledge, acquire skills and/or develop social capital. If the risk of death is high, natural selection tends to favour an early transition from maturation to reproduction (Promislaw and Harvey, 1990; Walker et al., 2006).

The myriad allocation decisions that constitute a LHS are not consciously cognitive but rather are executed via biological mechanisms responding to signals of endogenous (internal, somatic) and exogenous (external, extra-somatic) conditions. Strategic timing of allocation decisions requires an organism to process signals that are at least roughly reliable indicators of the current and/or changing status of these conditions.

The simplest conceptualization of a mechanism involves a reliable signal that is recognized by a transponder (receiver/transmitter), which then sends a different signal that elicits an appropriate response. For example, a signal of exogenous conditions (an "ecomarker") is processed through a sensory system and the brain, prompting a change in biology that elicits an investment of resources and time into one of perhaps several allocation options (e.g. a predator's growl prompts downstream responses that include rises in epinephrine and cortisol and will ultimately result in flight or freeze or fight).

At different points in a mechanism, signals may be molecular, electrochemical, morphological or psychosocial; behaviours of the individual, conspecifics or other organisms; social, economic

or cultural features of the group; and physical or biotic features of the environment. Signals of endogenous conditions (e.g. fat stores, rises in glucose) typically rely on molecular signalling and often involve neuroendocrine input and coordination (e.g. hypothalamic regulation of reproduction, sleep, hunger, thirst and body temperature).

An ecomarker may have a direct role in an organism's acquisition and/or distribution of resources, or it may act only as a reliable proxy that conveys information about factors external to the organism that have more direct roles in the organism's life history strategy. For example, in numerous organisms, the duration and intensity of daylight is an ecomarker of extra-somatic conditions that directly influences daily sleep/wake cycles, and that also influences longer-term physiological responses to seasonal variation in environmental conditions (e.g. reproductive functioning in response to predicted changes in food availability). These responses are endpoints in a mechanism that begins with absorption of light photons in the cells of the eye's retina, which cause molecular changes that eventually signal the pineal gland to produce and release melatonin. Receptors for melatonin are found in many brain regions, the pituitary, gut, ovaries and blood vessels. Neural receptors likely regulate circadian rhythms, and other receptors likely regulate reproductive function, cardiovascular function and body temperature (Brzezinski, 1997). It is not uncommon for molecular signals to be recognized by many different cells in an organism, each of which responds according to its own function.

Some of these conditions and the accompanying allocation decisions set the course for a lifetime (e.g. early maturation cannot be reversed). Other investment decisions are temporally limited and may incur few costs. For example, in healthy humans, pregnancy loss within a few weeks of conception does not appear to impair the probability of ovulation in the subsequent cycle (Donnet et al., 1990) or increase the subsequent mean waiting time to conception (Kaandorp et al., 2014).

Many allocation decisions re-occur throughout a lifetime as organisms navigate seasonal and circadian variations in environmental challenges and orchestrate the daily scheduling of physiological processes. For example, sleep is now recognized to be more than a means of conserving metabolic energy. Rather, it is an activity during which some necessary biological processes are better undertaken, either to avoid competing for resources with processes that must occur while awake, or because of incompatibility with such daytime processes. For instance, memory consolidation is optimized during sleep (Rasch and Born, 2013), night suckling has a greater impact on suppressing ovulation than day sucking (Elias et al., 1986; Vitzthum, 1994), and aspects of immune and reproductive functioning are modulated by melatonin, released in large measure only under cover of darkness (Nelson et al., 2002).

It merits reminding that one cannot assume which allocation response, if any, is adaptive (favoured by natural selection because it increases reproductive success) simply because of the mere fact that the organism displayed that response. Variations in such responses (i.e. better and worse allocation decisions over the course of a lifetime) are fodder for natural selection and hence the means by which LHSs can evolve in population-specific environments.

The Human Female Reproductive System — A Well-Tuned Machine or a Flexibly Responsive Behaviour?

The study of human fertility is biased towards female over male biology because pregnancy duration and other biological constraints limit the number of offspring a woman can produce and thus also, the rate of population growth. Men are not as unavoidably constrained, although, for a variety of reasons, neither is their reproductive capacity unbounded (Drea, 2005; Moya et al., 2016; Vitzthum et al., 2009b; Borgerhoff Mulder, in this volume).

The hypothalamic-pituitary-ovarian (HPO) axis, comprising three endocrine glands and the hormonal communications between these, is the primary pathway orchestrating physiological changes during the ovarian (menstrual) cycle and subsequent to fertilization (if it occurs). The hypothalamus, an almond-sized portion of the brain, links the nervous system to the endocrine system via the pituitary, a two-lobed pea-sized gland lying near the hypothalamus. The release of a neurohormone (gonadotrophin releasing hormone [GnRH]) from the hypothalamus prompts the anterior lobe of the pituitary to release other hormones into the blood that then circulate to and affect the functioning of the ovaries. The anterior pituitary's hormonal signals stimulate the development of ovarian follicles (the structure surrounding an immature egg cell) and prompt ovulation (the release of the mature egg from a single follicle). Ovulation is followed by transformation of the ruptured follicle into the corpus luteum, which produces the progesterone necessary for sustaining a pregnancy through the subsequent five or so weeks. In the absence of a conception, the corpus luteum regresses about a week or so after ovulation, progesterone begins to fall, and menstrual bleeding occurs (Figure 1).

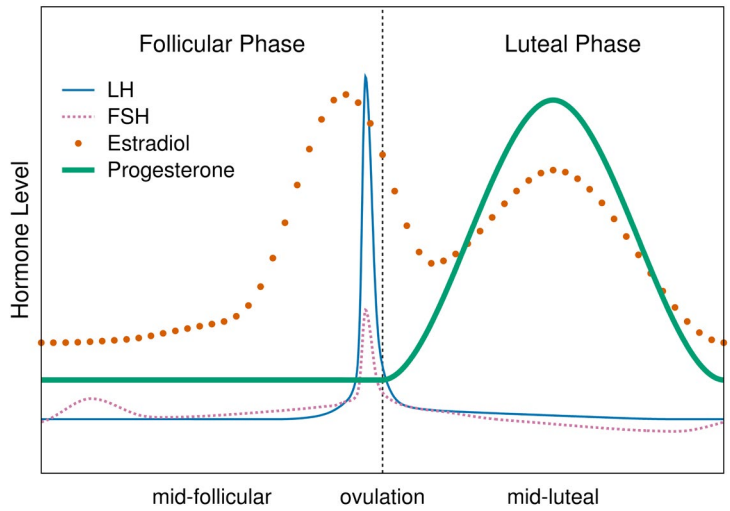

Fig. 1 Idealized depiction of hormonal changes (relative to day of ovulation) during the ovarian cycle. Follicle stimulating hormone (FSH) promotes follicle development. The estradiol peak prompts a surge in luteinizing hormone (LH), which binds to receptors on the follicle, thus initiating ovulation. The follicle transforms into the corpus luteum, which secretes progesterone. If a conception doesn't occur, progesterone declines, culminating in menses. In an ovulatory cycle, each phase lasts from about eight to twenty-two days (phase durations are not correlated); hormone levels in ovulatory cycles are highly variable between cycles, women and populations (Vitzthum, 2009).

The more-or-less monthly appearance of menstrual bleeding in most healthy pre-menopausal women (other than those who are pregnant or breastfeeding) tends to bolster the widespread belief that the female reproductive system is unflagging in its cyclical effort to conceive. This idealized view of ovarian regularity derives in part from Descartes' (1637) conceptualization of the body as a machine and is reflected in the work of Malthus (1798), who argued that moral restraint and early death were all that kept a population from outstripping its food supply in a few generations.

The powerful body-as-machine metaphor continues to subtly influence contemporary sciences. This impact is perhaps most evident in biomedicine. The image of a well-tuned machine, necessarily invariant in the form and coordination of its components, readily lends itself to a narrow definition of "normal" biology, and tends to perpetuate the classification of variants not meeting the criteria for "normal" as pathologies that require medical interventions. Such criteria for hormones and other biomarkers typically involve the designation of upper and/or lower thresholds outside of which the biomarker is considered abnormal. Given that medicine's mission is to identify illness and restore the patient to health, arguably such diagnostic practices are acceptable, even desirable, regardless of the underlying misconceptions. Better to recognize all who may be sick, and a few who are aren't, than to miss those needing treatment.

But what if the diagnostic threshold derived from a faulty assumption about normal variation has identified a treatment pool comprising more healthy people than ill: then what? If this seems far-fetched, consider the "impairment" referred to as luteal phase deficiency (LPD). First described by Jones in 1949, LPD is characterized as insufficient endogenous progesterone for the adequate development of the uterine lining, successful implantation and early pregnancy maintenance. Diagnostic criteria have included a short luteal phase (under the false belief that a normal luteal [post-ovulatory] phase is twelve to fourteen days) and low progesterone concentrations. But, in fact, the luteal phase varies considerably in cycles in healthy women (WHO, 1983), and the Practice Committee of the American Society for Reproductive Medicine (2015) has concluded, "no minimum serum progesterone concentration defines 'fertile' luteal function". Examination of the uterine lining (endometrial biopsy) was thought to be the diagnostic "gold standard" for luteal phase deficiency, however, rigorous clinical trials have concluded otherwise. For example, one large double-blinded study found that about half of the mid-luteal endometrial biopsies were considered "abnormal" according to LPD diagnostic criteria in *both* fertile and infertile women (Coutifaris et al., 2004). In other words, natural variation had been mistakenly perceived as pathology.

Although the body is obviously not a machine, sometimes this imagery can divert us from recognizing the inherently variable nature and flexible capacities of physiological mechanisms. In his essay laying out the four questions, Tinbergen (1963) praised Konrad Lorenz for having "made us look at behaviour through the eyes of biologists". In so far as metaphors can aid understanding, it may prove of use to flip the view and look at physiology as an ethologist might. Rather than analysing features of physiological systems (e.g hormone concentrations) as if they are species-specific morphological traits that are only modestly variable across populations, it may be more useful to think of physiological mechanisms as responsive behaviours whose range of expression reflects developmental conditions and is contingent on immediate circumstances.

This perspective is consistent with current knowledge of hormone-receptor signalling behaviours. A hormone exerts its effect by binding to a receptor and having a molecular

configuration suitable for that hormone (rather like a key in a lock). The hormone-receptor complex can then signal to the cell to perform some biological response. Receptors are a large class of proteins encoded in the cell's genes; receptors specific to a cell's function are manufactured by that cell. Regulation of receptor manufacture is affected by several endogenous and exogenous developmental and environmental factors, depending on the specific cell and intended action.

The relative number of receptors to hormone molecules is critical in regulating the cell's actions. Without receptors, hormones (no matter how high the concentration) cannot directly affect cell behaviour. The relationship between hormone concentration, receptor availability and biological response varies by receptor. There can be many different types of receptors for a given hormone, and a given receptor may be able to bind with different hormones.

High affinity receptors (those that form stronger molecular connections with a given hormone) can attract and bind hormones at low concentrations and trigger cell action. In other cases, hormones need to be present at high concentrations in order for enough receptors to be bound and thereby elicit a biological response. The rhythm of change may matter in some pathways (i.e. increases in the amplitude and/or frequency of pulsatile hormonal signalling, rather than a monotonic rise in hormone concentration, are necessary to prompt a response in a target cell). In some mechanisms, the presence of the hormone will prompt the cell to produce more receptors, and then, once enough hormone-receptor complexes are formed, the cell will perform its action ("upregulation" is the cell's creation of more receptors that make the cell more sensitive to the hormone). But at other times even high hormone concentrations will not trigger the production of more receptors (e.g. insulin resistance) and the biological response is not performed. In general, when available receptors become saturated (because of hormone binding and/or receptor degradation), the cell becomes less sensitive to the presence of the hormone (a process called "downregulation").

Physiological mechanisms are dynamic and variable, a consequence of evolution's tinkering, using the materials and tools bestowed by previous generations to deal with the task at hand (Jacob, 1977, 1994). While some pathways may be conserved (e.g. the link from hypothalamus to pituitary to ovary), features potentially shaped by developmental environments will likely vary (e.g. individuals' hormone concentrations and numbers of receptors). Such systems are rather like an orchestra — a composition (pathway) is typically followed, each instrument coming into play in a fairly predictable fashion, but the rhythm, volume and numbers of each kind of instrument (hormone signal) may vary as can the number of listeners (receptors) who come and go. Sometimes compositions from adjacent orchestras can be heard (cross-talk) and sometimes there's unexpected improvisation provoked by a novel situation (new foods, environmental toxins, glucose overload).

A Delicate Balance: Strategic Trade-Offs of Incompatible Essential Functions

The primary mission of a woman's immune system — to protect her body — is sometimes unavoidably at odds with the evolutionary imperative to reproduce (Abrams and Miller, 2011; Alvergne and Tabor, 2018). For example, because of sperm's genetic foreignness and the health risks posed by any pathogens in deposited semen, coitus might be expected to elicit a heightened immune defence in women. Yet such a response would potentially harm the sperm required for

a conception. Notably, however, sperm's reproductive value is limited to a few days (known as "the fertile window") leading up to and including the day of ovulation.

The key to balancing these incompatible functions is through the strategic timing of selected immune defences. Specifically, in sexually active healthy women, we would expect relatively high immune defences to protect against the risks associated with coitus, but also a transient dampening of some immune defences around the time of ovulation (the fertile window) in order to increase the chances for successful conception. In sexually abstinent healthy women, immune defences need not be as high as in sexually active women, and are not expected to change during the fertile window.

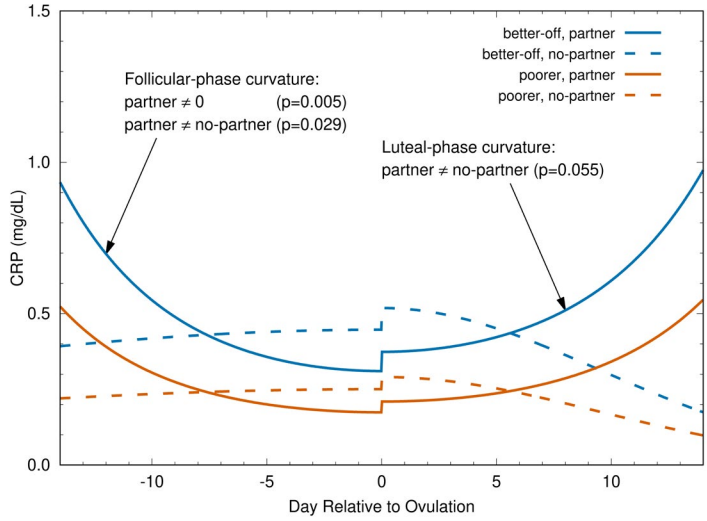

Fig. 2 Change in CRP during ovulatory menstrual cycle. Fitted models for the interaction of partnership status and socioeconomic status. CRP is significantly higher during the early follicular and late luteal phases (P = 0.029 and 0.055, respectively) in partnered (solid curves) than in unpartnered (dashed curves) women. In partnered women, CRP is lower around ovulation than at the cycle's beginning (fitted model curvature is significant at P = 0.005). In contrast to partnered women, CRP in the ovulatory cycles of unpartnered women is more stable over time (fitted model curvature is not significantly different from 0). The small increases in CRP at ovulation are not statistically significant in these models (Lorenz et al., 2015).

These predictions have been tested and supported in studies of Bolivian women and US women. In Figure 2, the predicted patterns are observed in two Bolivian samples, one of poor women and the other of economically better-off women (Lorenz et al., 2015). The selected immune biomarker (C-reactive protein, CRP) patterns during the menstrual cycle are the same for both samples. Likewise, the pattern is similar in a sample of US women, who are wealthier than both Bolivian samples (Lorenz et al., 2017). The comparability of this pattern across samples with different energetic resources suggests that greater energy availability does not modify the need to dampen some immune defences at ovulation in sexually active women. In other words, even though this dampening may come at significant health costs to women (e.g. autoimmune diseases, sexually transmitted infections) (Beer et al., 1996; Whitacre et al., 1999;

Wira and Fahey, 2008; Wira et al., 2010; Kaushic et al., 2011; Klein, 2012), it does not appear to be mitigated by greater access to energy resources.

Which Comes First — the Coitus or the Egg?

The differences in immune-reproduction co-ordination between sexually active and abstinent women are likely a consequence of seminal fluid components (e.g. cytokines) that provoke changes in the female reproductive system (Robertson and Sharkey, 2016). The presence of such components is a signal of the possibility of conception and the concomitant necessity of modifying immune responsiveness.

However, if ovulation does not occur, such shifts in immune function in response to seminal fluid are not needed and would be potentially risky for the woman's health. Therefore, one would expect sexually active women to have a higher probability of ovulating than sexually abstinent women (i.e. ovulation is worth the risk from a shift in immunity for sexually active women, but is an unnecessary risk in the absence of sex). Consistent with this prediction, Metcalf (1983; Metcalf and MacKensie, 1980) observed lower ovulation rates in unpartnered than partnered New Zealand women, and Wilcox et al. (2004) found concurrent increasing probabilities of ovulation and coitus in a sample of US women. However, neither study could specify the direction of the causal arrow (i.e. does coitus induce ovulation or vice versa?).

Now, a recent study that included daily documentation of coitus and serial hormonal biomarkers to detect ovulation has yielded strong evidence that coitus increases the probability of ovulation in humans (Prasad et al., 2014). It is plausible (but was not tested in this study) that seminal fluid components are an essential part of the mechanism linking coitus to the physiological decision to ovulate.

This finding flips the causal arrow on the hypothesis that hormonal changes accompanying ovulation in women prompt increases in sexual attraction, desire and/or activity. Most studies of this prediction have failed to demonstrate such an association. The large majority of these studies have assumed, without biomarker confirmation, the timing and occurrence of ovulation during the study cycle. Such assumptions are untenable in light of the evidence that ovulation is not inevitable, that its timing is not restricted to a narrow mid-cycle window (reviewed in Vitzthum, 2009), and that coitus itself promotes ovulation.

These observations and arguments, however, raise other intriguing questions. If a woman is not sexually active, why bother to ovulate at all? The fact that there is still an appreciable probability of ovulation in sexually abstinent women suggests that the cost of ovulation is low. One possibility is that once the wheels are set in motion, this low-cost process chugs along unless hindered (perhaps by signals that any risk of conceiving is a poor strategy at this time). Another possibility is that, outside of breastfeeding, there has been little selection against ovulation, but neither has there been strong selection for ovulation in the absence of coitus. Since sexual abstinence was likely uncommon during human's evolutionary history, opportunities for selecting against or for ovulation in the absence of coitus were relatively few. Even in those few instances, any resource savings in not ovulating may have been more than offset by the value of ovulating on the chance that coitus occurs.

This conjecture then raises the question, "If the cost of ovulation appears to be low, why not be an obligate ovulator regardless of coitus?" Perhaps tying ovulation to coitus is a selectively advantageous mechanism that helps to compensate for the short life of sperm, more closely

linking the availability of an egg to the deposition of seminal fluid than would otherwise occur. The timing of coitus may help to explain why the duration of the pre-ovulatory (follicular) phase of the cycle is more variable than the post-ovulatory (luteal) phase. The answers to these questions await further study of the physiological mechanisms regulating ovulation, especially of the links to coitus.

Vote Early, Vote Often: Early Pregnancy Loss

The collective evidence from several studies suggests that only one in five human conceptions are born. Thus, worldwide during an average day in 2019, there were 360,000 live births, 1.8 million new conceptions, and 1.44 million naturally lost human pregnancies (the vast majority of which were unrecognized by the woman or her clinician) (Vitzthum, 2008b).

This unexpected wastage was first brought to light in 1975 by two epidemiologists who estimated pregnancy loss in England and Wales to be 78% based on the marriage rate and reasonable assumptions regarding coital frequency and other relevant factors (Roberts and Lowe, 1975). Subsequent studies, though few, have reached comparable conclusions. Boklage (1990) combined the published results of several observational studies of pregnancy loss in industrialized populations and developed a parametric model from which he estimated a total pregnancy loss of about 76%. Holman and his colleagues (Holman and Wood, 2001) mounted an impressive study that monitored nearly 500 non-contracepting married Bangladeshi women and collected urine samples, later assayed for a biomarker of implantation, from 1,561 menstrual cycles. With these data, they detected post-implantation pregnancies and losses, and estimated the total pregnancy loss from conception to birth to be about 80%.

Modelled estimates suggest that pregnancy loss is greatest between conception and implantation (about 50–60% of all concepti). But there is little direct evidence because during this early stage women are unaware that they are pregnant, and there is not yet an easy-to-collect reliable biomarker for detecting conceptions prior to implantation. Implantation, which occurs 9 ± 3 days after conception, is recognized by a rise in human chorionic gonadotropin (hCG). Over-the-counter early pregnancy tests are designed to react to the presence of this hormone in urine samples, and several studies have made use of this biomarker to estimate pregnancy loss rates. Estimates of loss occurring from implantation through the subsequent month were about 25% to 30% of implanted concepti in several studies in industrialized populations (Wilcox et al., 1999; Ellish et al., 1996; Zinaman et al., 1996; Wang et al., 2003; van Montfrans et al., 2004). In Bolivian women, 31% of implanted concepti were lost within five weeks of conception (Vitzthum et al., 2006). Based on hazard models (which produce higher and more accurate estimates), Holman and Wood (2001) estimated loss within five weeks of conception to be about 65% in 28-year-old Bangladeshi women. Among settled Turkana agriculturalists in Africa, about 70% of implanted concepti were lost by ten weeks after conception (Leslie et al., 1993). Several studies have shown that by the end of the second month of pregnancy, the risk of subsequent loss has dropped to only 10–15% (Vitzthum, 2008b). In many cultures, women decline to mention to others that they are pregnant until this stage has been reached and the risk of not going to term (i.e. not giving birth) is low.

Roughly speaking, based on the collective evidence, of one hundred conceptions, fifty-five would not successfully implant, twenty-two would be lost during the month after implantation, and three more would be lost in the subsequent months, yielding twenty live births.

Evaluating Offspring Quality

Such apparently wasted effort naturally prompts questions about the causes underlying these losses. The canonical response is that concepti are lost early in pregnancy either because their genetic defects preclude normal development or because maternal mechanisms cull poor-quality offspring unlikely to mature and contribute genes to subsequent generations. Such weeding allows a woman to redirect investments to current or future offspring (Temme, 1986; Kozlowski and Stearns, 1989; Haig, 1990, 1993, 1999). Even given abundant resources, a low-quality conceptus should be rejected quickly to avoid wasting maternal time that could be given to attempting another conception.

Perhaps the most important maternal mechanism for evaluating offspring quality depends on the embryo's own ability to produce sufficient hCG as it begins implantation. Production of this hormone is proof of the embryo's ability to carry out protein synthesis, the most minimal requirement of viability (Haig, 1993). In a process referred to as corpus luteum rescue, the embryo signals its presence through hCG binding to receptors on the corpus luteum, which responds by continuing progesterone production to sustain the pregnancy (recall that falling progesterone concentration results in menstruation) (Jabbour et al., 2006).

Timing as well as the volume of conceptus-produced hCG is critical in this mechanism. The rise in hCG that accompanies implantation must occur between six to twelve days after ovulation if the conception is to be sustained. The later that implantation begins, and the later that the rescued corpus luteum subsequently produces more progesterone, the more likely it is that the conceptus will be lost during the subsequent month. Failure to produce sufficient progesterone quickly enough is a consequence of the embryo's inability to produce enough hCG, rather than any defect in the corpus luteum (Baird et al., 1991; Vitzthum et al., 2006).

In effect, through the mechanism of corpus luteum rescue, the embryo will trigger its own rescue if it can produce, at the right time, enough hCG to assure continually rising progesterone production by the corpus luteum. An unknown proportion of concepti fail this first test and menstruation renews the cycle. The maternal opportunity cost for having conceived and lost this early is very low. Early pregnancy loss does not appreciably lengthen the time to the next cycle (Vitzthum et al., 2000b), lower the probability of ovulation in the subsequent cycle (Donnet et al., 1990), or increase the subsequent mean waiting time to conception (Kaandorp et al., 2014). Furthermore, menstrual flow is not appreciably greater, which suggests that energy expenditure may not be much higher (Vitzthum et al., 2001).

For embryos that do make it past this first gateway, at least 30% and as many as 50% will be terminated before the end of the subsequent month, by which time another gatekeeping mechanism has come into play. The luteo-placental-progesterone-transition (LPPT), occurring by about five weeks since ovulation/conception and seven weeks since the first day of the last menstrual period, is a developmental period during which the production of progesterone from the placenta (an offspring structure) begins to be greater than that from the corpus luteum (a maternal structure). Because progesterone is essential for the maintenance of the pregnancy, if this shift does not occur, the pregnancy will not continue (i.e. insufficient production of placental progesterone is indicative of a poor-quality offspring).

In conceptions that do continue, the LPPT has shifted the locus of physiological control of the pregnancy from the mother to the offspring. It is in an offspring's own interests to sustain the pregnancy and, in large measure, during the LPPT the embryo is becoming the master of its own

fate. Once it has the ability to produce enough progesterone without maternal contribution, any maternal interests contrary to those of the offspring may not prevail. Consistent with this prediction, only 10–15% of those pregnancies that survive through the LPPT are subsequently lost before birth.

The LPPT is a well-documented physiological change that occurs during early pregnancy. The evolutionary explanations for the LPPT are predicated on the fact that a mother and her offspring are not genetically identical and hence the optimal degree of parental investment to give and receive are likely not to be identical (Trivers, 1974; Haig, 1993). At first consideration, the idea of parent-offspring conflict would appear to be at odds with an expectation that it is in the evolutionary interests of a mother to invest in her offspring. Life history theory, however, recognizes that there are trade-offs — what is invested in one offspring cannot be invested in another. For example, in some environments (e.g. those with high infant mortality) it may be selectively advantageous for a woman to have two smaller children rather than one larger child. Therefore, natural selection is expected to favour the maternal life history strategy that produces the number and quality of offspring that will result in the greatest lifetime reproductive success for her under the environmental conditions in which she lives, even if her LHS is not the optimal investment from the perspective of each offspring (see Strassman and Gillespie, 2002 for a notable example).

Evaluating Maternal Quality

Life history theory also predicts that maternal somatic conditions and/or external environmental circumstances that are inadequate for sustaining a pregnancy through to term may prompt rejection of a conceptus, even if that offspring is not defective (Wasser and Barash, 1983; Peacock, 1990; Vitzthum, 1990). The LPPT imposes a timing constraint on maternal decisions to terminate investment in the current conception. If it is in the mother's evolutionary interests to do so, then termination is best effected while her own physiological mechanisms still regulate the bulk of progesterone production (i.e. before the LPPT). The timing of the LPPT reflects opposing interests. Selection on the offspring favours an early LPPT and the accompanying physiological control of pregnancy continuance. Selection on the woman favours a later LPPT so as to keep her investment options open.

Although it may initially appear paradoxical that a parent would terminate investment in a non-defective offspring, this option may be evolutionarily advantageous given the costs and risks associated with continuing a pregnancy, giving birth and breastfeeding an infant. A mother's investment in the production of a live offspring is sufficiently high that it can, and sometimes does, cost her life (Vitzthum and Spielvogel, 2003). Before the advent of antibacterial sulfonamides in the mid-1930s, an estimated 300–900 women per 100,000 pregnancies died from pregnancy-related causes (Loudon, 2000). Currently, about 300,000 women die each year (UNFPA, 2019). Mortality is only the tip of the iceberg, with maternal morbidity affecting millions of women each year. With such high costs, natural selection on offspring is expected to be especially high early in gestation (when maternal investments, opportunity costs and risks are low) and relatively lower after birth (by which point considerable resource and opportunity costs, and much of the cumulative risk to the mother, have already been incurred).

Under the assumption that early pregnancy loss (EPL) is due almost entirely to genetic defects in the conceptus, there have not been many empirical studies of the hypothesis that maternal somatic or environmental conditions are significant contributors to EPL. Three studies have addressed these questions. The North Carolina Early Pregnancy Study (NCEPS) recruited

221 women who self-collected daily urine samples (subsequently assayed for progesterone and estrogen metabolites and hCG) while attempting to become pregnant naturally (Wilcox et al., 1999). Project REPA (Reproduction and Ecology in Provincía Aroma) collected thrice-weekly saliva (assayed for progesterone) and urine samples (tested for hCG) from 191 menstruating rural Bolivian women in a stable sexual partnership (Vitzthum et al., 2004). Twenty-four Guatemalan women self-collected thrice-weekly urine samples later assayed for several hormones (Nepomnaschy et al., 2006). Data from these studies on variability in the risk of EPL, and the environmental and hormonal mechanisms associated with these patterns, suggest life history strategies reflective of maternal factors are also at play in EPL.

In general, genetic defects are expected to occur randomly over time. Thus, if EPL were due almost entirely to genetic defects, EPL would also be expected to be randomly distributed over the course of a year. It was therefore surprising to find seasonal peaks and valleys in the distribution of EPL detected during the NCEPS (Weinberg et al., 1994). Although the pattern was clear — a peak at least four times greater than the trough occurring some time from September through December in three consecutive years — the authors were unable to explain it.

Project REPA also observed seasonal differences in EPL (Figure 3). The arduous planting and harvesting seasons had a 3.7 times greater risk of loss than the other seasons, and agropastoralists were nine times more likely to experience EPL than those engaged in some other livelihood. The authors attributed the seasonal increase in EPL to the demanding physical labour of farming, but also noted that inadequate food reserves and greater psychological or immunological stress could also be contributing.

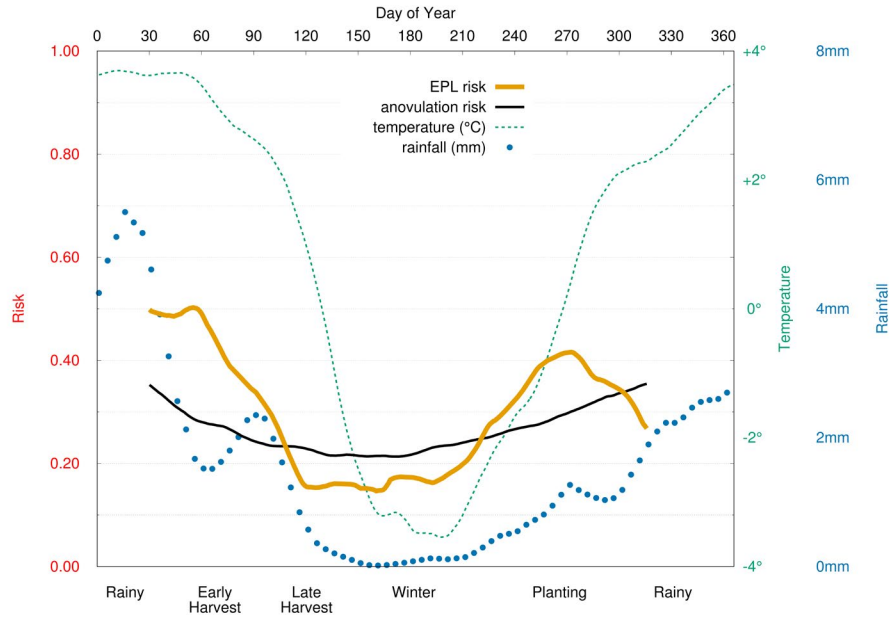

Fig. 3 Seasonal variation in anovulation and early pregnancy loss (EPL). Risk (left-hand scale) of anovulation and EPL, and daily rainfall (far right-hand scale), and minimum-temperature (near right-hand scale) as functions of time (top scale, day of year). Agricultural activities (bottom scale) are positioned relative to day of year. Risk of EPL and anovulation are elevated during the most energetically demanding periods (Vitzthum et al., 2009a).

Two proposed physiological mechanisms that might link poor maternal conditions to EPL have been tested. Reflecting the important role of ovarian steroids in preparing the uterine lining for implantation and sustaining a pregnancy, one hypothesis predicted that ovarian steroid concentrations would be lower in conception cycles that end in EPL than in conception cycles that are not lost. Data from NCEPS and Project REPA failed to support this prediction. In both studies, the ovarian steroid profiles of the successful and lost conceptions did not differ from ovulation through early pregnancy (Baird et al., 1991; Vitzthum et al., 2006) (Figure 4).

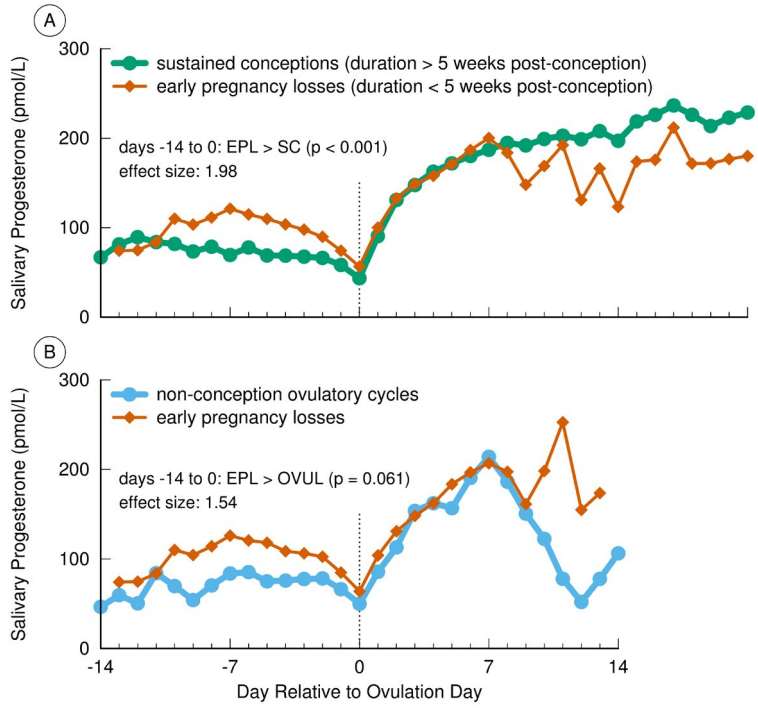

Fig. 4 Progesterone (P4) concentrations in sustained conceptions, early pregnancy losses (EPL), and non-conception cycles in Bolivian women. Post-ovulation P4 did not significantly differ between EPL and conceptions persisting for at least 5 weeks after conception (Panel A). In contrast, preovulatory P4 was significantly higher in EPL compared with sustained conceptions (Panel A) and compared to non-conception ovulatory cycles (Panel B) (Vitzthum et al., 2006).

A second plausible mechanism for maternal evaluation of conditions involves cross-talk between the HPO-axis and the hypothalamic-pituitary-adrenal (HPA) axis. During the pre-ovulatory phase of the ovarian cycle, the adrenal cortex is the main source of progesterone, typically produced at levels much lower than those of the progesterone produced by the ovaries following ovulation. However, under stressful conditions (e.g. increased physical activity, food restriction, psychosocial stress), the adrenal glands increase production of cortisol and adrenal progesterone. Elevations of these hormones early in the ovarian cycle may disrupt normal ovarian functioning including development of the follicle, ovulation, implantation and/or sustaining an implanted conceptus (Vitzthum et al., 2006).

Evidence from the Guatemalan study and from Project REPA suggests this mechanism underlies at least some EPL. In the conception cycles of the Guatemalan women, those with high cortisol concentration were 2.7 times more likely to end in EPL than those with normal cortisol concentrations. In other words, 90% of those conceptions with elevated cortisol were lost as compared to only 30% of those with normal cortisol concentrations (Nepomnaschy et al., 2006). In the conception cycles from the Bolivian women (Figure 4, Panel A), adrenal progesterone during the follicular phase was significantly higher in those pregnancies that terminated prior to the LPPT compared to those that persisted beyond this transition.

The findings from these three studies are consistent with the life history prediction that, in addition to the quality of the offspring, maternal somatic status and environmental conditions are potentially important determinants of whether or not to continue investment in a new conceptus. Although energy stores (adipose tissue) and seasonal energy availability can be major factors in maternal reproductive decisions, psychosocial, micronutrient and immunological/disease conditions may also trigger termination of reproductive investment, regardless of maternal energy adequacy.

A Pair of Paradoxes and the Physiological Fallacy

> Honourable errors do not count as failures in science, but as seeds of progress …
>
> — Gould (1998)

Beginning in the late 1970s, an intellectual dispute arose between demographers and biologists (bioanthropologists, physiologists, medical scientists, evolutionary biologists) regarding the role of energetics (caloric intake and expenditure) in human reproduction.

Biologists, on the one hand, had both good theoretical arguments and considerable data in favour of the position that energetics is a major determinant of fertility. In particular, studies of US and European women who were following calorie-restricted diets and/or regular strenuous exercise regimes (either in or outside a laboratory setting) were observed to experience disruptions in their menstrual cycles, including reductions in reproductive hormone concentrations. Furthermore, with increasing energetic severity, the disruptions could become so pronounced that ovulation and menses ceased altogether (dubbed "exercise-associated amenorrhea"). Although some observers considered these changes to be pathologies, Jerilyn Prior (1985a, 1985b, 1987) and a few others argued that these were adaptations to spare women from conception when energetically stressed, a condition that could increase maternal and offspring risks for morbidity and mortality.

Demographers, on the other hand, had a world's worth of compelling population-level demographic data that supported the position that energetic stress (other than starvation) has only a trivial impact on human fertility. Bongaarts (1980) laid out the data and arguments in a widely influential paper in *Science*. Perhaps his most convincing point was that many of the very populations experiencing the most significant energetic stress were also those with the highest fertility.

Faced with a seemingly unresolvable paradox — physiological data demonstrating energetic impacts on individuals in industrialized countries yet no apparent impact on population-level fertility parameters in energetically stressed populations — the two sides, for the most part, retreated to their respective domains.

A technological development and more data, however, revived the discussion in the late 1980s. The first studies of possible differences between populations in the concentrations of reproductive steroid hormones (progesterone, estrogens) were largely motivated by an interest in finding the causes of marked population differences in the risks for breast and other cancers. In general, these studies found lower concentrations of these hormones in Asian compared to US and UK "white" populations (Dickinson et al., 1974; MacMahon et al., 1974; Trichopoulos et al., 1984; Bernstein et al., 1990; Key et al., 1990; Shimizu et al., 1990 Wang et al., 1991). There was little, if any, suggestion in the published literature from these epidemiological studies that the observed hormone differences might also cause population differences in fertility.

The first study by bioanthropologists of reproductive hormones in African populations also observed lower concentrations compared to those observed in European and US samples (van der Walt et al., 1978). However, these investigators explicitly argued that the lower concentrations were indicative of lower fecundity and were perhaps evolutionary adaptations to energetic stress. The development of salivary hormone assays (an alternative to blood-based assays) allowed other anthropologists to collect data from several energetically stressed rural populations (Democratic Republic of the Congo, Nepal, Bolivia and Poland), all of which proved to have average salivary progesterone concentrations significantly lower than the average observed in a sample of US women (Ellison et al., 1989; Panter-Brick et al., 1993; Jasienska and Ellison, 1998; Vitzthum et al., 2000a).

These additional observations generated a second paradox. Although these energetically stressed populations had relatively lower progesterone concentrations, they did not necessarily have low fertility. For example, women in the rural Bolivian population, with an average progesterone concentration only 70% that of US women, had an average of seven live births each, with some women reporting as many as thirteen offspring (Vitzthum et al., 2004).

The resolution of each of these two paradoxes was not simply a matter of figuring out who was right and who was wrong (the various studies had, in fact, been well executed by competent scientists). Rather, we needed to re-think our assumptions and interpretations of the available data with fresh eyes. This re-assessment involved taking the empirical data at face value — specifically, (1) women in industrialized populations had relatively high progesterone concentrations and experienced ovarian cycle disruption, including lower progesterone, when energetically stressed, and (2) women living in energetically stressful conditions had relatively low progesterone concentrations, but nonetheless were having lots of babies — and examining these biological patterns within an evolutionary framework. The following assessments arose from this approach (Vitzthum, 1990, 1997, 2001, 2009, 2020).

First, there is no scientific justification for assuming that hormonal data from US/European women are a normal or desirable standard against which to compare all other populations. The fact that interpopulation hormonal variation does not correspond to interpopulation differences in fertility strongly suggests that there is no species-specific "normal" progesterone concentration necessary for reproducing. Comparable to the misdirection taken in medicine by assuming that statistically defined thresholds are genuine markers of normalcy, so efforts in reproductive ecology have been led astray by assuming that higher concentrations of reproductive hormones necessarily equate with higher fecundity and fertility.

Second, acute and chronic energetic stressors are not necessarily biologically equivalent. The timing (whether pre-natal, pre-adult, or during adulthood), duration and magnitude of a

stressor can all impact how an organism responds to the challenge. The disruption in ovarian function that accompanies an acute energetic demand is a temporary cessation of reproductive investment in favour of temporarily increased somatic demands. If the organism never resumes reproductive investment before dying, it is likely to be at a selective disadvantage compared to individuals who do reproduce. Therefore, if an acute temporary demand persists, the organism may become less sensitive to this demand so as to resume the normal array of bodily functions (a physiological state called "homeostasis"). Unlike acute demands, chronic energetic demands must be managed differently because these stressors are the very nature of the environment in which the organism lives and must reproduce. The organism must have a life history strategy that results in successful reproduction in these tougher conditions.

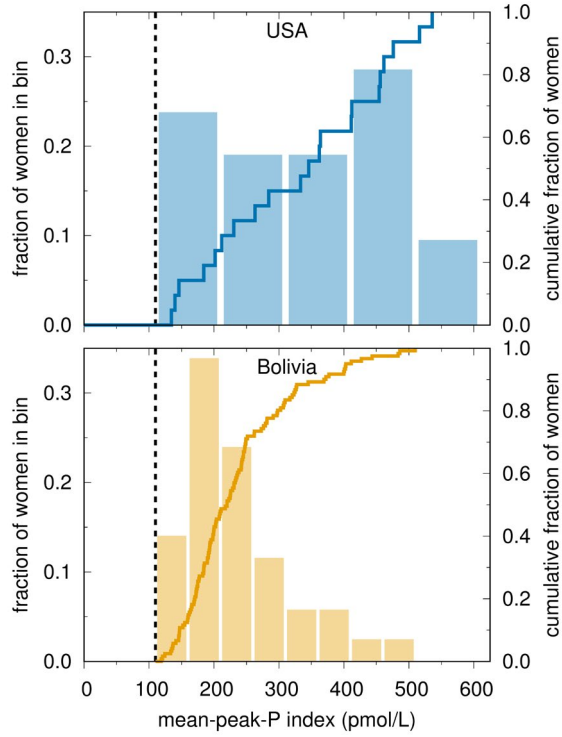

Fig. 5 Progesterone variation in ovulatory cycles. Histogram (left scale) and cumulative distribution (right scale) of a mid-luteal index of hormone concentration (mean-peak-progesterone). Progesterone concentrations differ substantially between the two populations and between women within each population (Vitzthum et al., 2004).

Third, between-population differences in reproductive hormone concentrations are not necessarily equivalent to the within-person reductions in hormone concentrations associated with an acute energetic stressor. This mistaken equivalency — a "physiological fallacy" somewhat analogous to the "ecological fallacy" (Robinson, 1950; Selvin, 1958) — ignores both the processes that generate a given hormone concentration and the units of analysis, and hence misidentifies the absolute level of the hormone as the necessarily salient signal in biological mechanisms. Rather, at least as regards the role of ovarian steroids in mechanisms that

implement LHSs, the current evidence suggests that it is the temporal *change* in the hormone's level that transmits information about changes in the factors that influence reproductive investments. If change is the (more) salient signal, then it is likely that there is not strong selection for specific hormone levels. Rather, there is the potential for high, yet nonetheless normal, variability in absolute hormone concentrations within and between populations (Figures 5, 6).

The prediction that marked hormonal variability is normal is supported by empirical studies demonstrating that different reproductive hormone concentrations are functionally equivalent across individuals and populations. Although progesterone concentration is significantly lower in Bolivian than in Chicago women, Bolivians successfully conceive at these lower concentrations (Figure 6) (Vitzthum et al., 2004).

In the search for mechanisms that regulate LHSs and generate demographic diversity, the largely unexamined assumption that there are necessarily species-specific "normal" concentrations of a given hormone has led us down blind alleys and obscured our understanding of how the HPO axis works. Dropping this assumption has both resolved previously inexplicable paradoxes and suggested novel models that better reflect how physiological mechanisms transform signals and implement LHSs.

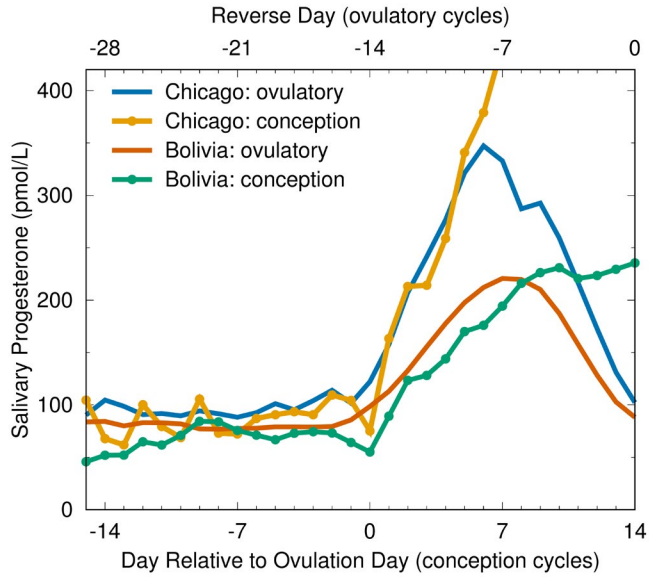

Fig. 6 Salivary progesterone profiles in conception and ovulatory non-conception cycles. Progesterone concentrations in ovulatory cycles are significantly lower in women from Bolivia than in women from Chicago throughout the ovarian cycle, and also lower during and subsequent to ovulation in conception cycles (Vitzthum et al., 2004).

Evolving Research Directions for the Study of Mechanisms

It has been nearly a century since Fisher reflected on what might be gained by knowing something of the physiological mechanisms underlying resource allocation strategies and more than half a century since Tinbergen re-emphasized the centrality of determining mechanism

in our efforts to thoroughly understand a behaviour. Yet we've just begun to delve deeply into the unexpectedly complex details of exactly how an organism achieves successful trade-offs between survival and reproduction. This recent progress is possible because of advancements in biomarker measurements and field-friendly methods for collecting longitudinal as well as cross-sectional data in community-based studies. The expansion of complex statistical models and greater computational capacities have also improved analyses of this wealth of data. Although the investment can be high, the pay-off is often impressive.

Theoretical developments are as necessary as better technologies for discovering the origins and functioning of a specific mechanism underlying a life history strategy (LHS). For example, recognition of parent-offspring conflict regarding optimal parental investment explains *how* pregnancy loss can be a successful LHS in some circumstances and *why* there are maternal mechanisms to test offspring quality. The evolutionary insight that pregnancy is more akin to a Greek play laden with conflict than a pas-de-deux laced by harmony deepens our understanding of human biological variability and potentially prompts re-evaluations of explanatory models in related fields (e.g. medicine, reproductive technologies, demography).

Below I briefly describe two of the many research areas concerning life history mechanisms that deserve attention as we move forward.

Adaptations, Cross-talk, and Trade-offs: More Is Needed on the Mechanics of Mechanism

Whether short-term (a day, a season) or long-term, resource allocations are potentially subject to natural selection (i.e. they may be adaptive responses that have evolved to increase lifetime reproductive success in a given set of population-specific conditions). Testing whether or not a life history strategy or some specific investment decision is adaptive is a daunting task, especially in the long-lived human species. Knowledge of the specific mechanism by which a given resource allocation is accomplished facilitates the testing of adaptation hypotheses, and can enlighten efforts to address the other three of Tinbergen's four questions.

Although it is obvious that there must be considerable coordination of actions among the various systems of an organism, for the most part investigators have understandably ignored those interactions in favour of tackling (relatively) manageable questions. It is now evident that we must begin to venture out beyond these imagined borders, and grapple with how various systems inter-communicate and thereby effect responses that are potentially adaptive for the organism.

To accomplish this goal requires figuring out the details of how a mechanism is engaged and operates. For example, at a particular point in a specific pathway, is the salient feature of a signal its absolute status (e.g. hormone concentration) and/or a change in its status (e.g. a rise or fall regardless of the signal's baseline concentration)? What are the specific contexts and factors (nutrient or time limits? mechanical or physiological incompatibility?) that tend to prompt one response over another? By what signals and pathways is this response recognized and responded to by other organismal components?

In addition, although this chapter has focused on the physiological mechanisms by which life history strategies are implemented, resource allocation strategies may also involve somatic, behavioural or extra-somatic mechanisms. Maintaining body-fat stores, food caching (whether buried nuts or dried agricultural surplus) and building reciprocal social networks all demand

time and resources diverted from immediate reproductive investment, but each of these strategies can mitigate the risk inherent in variable environments and thereby potentially increase lifetime reproductive success (Lee and Boe, in this volume). How, then, do such non-physiological mechanisms become an integral component of the organism's LHS and thus influence the workings of physiological mechanisms and reproductive output? At least part of the answer to these questions involves learning more about the precise mechanics of the pertinent mechanisms.

Linking (Adult) Mechanisms and (Pre-adult) Ontogeny

In their tribute to Tinbergen, Bateson and Laland (2013) emphasized that consideration of "mechanism always requires specification of a point in development". Ontogeny rightly comprises the entire developmental history of the organism up to the time at which a behaviour is occurring, and the causes of that behaviour may trace back to the organism's conception (Tinbergen, 1963).

It has been recognized for some time that early conditions can shape an organism's functioning during its subsequent life. This calibration of individual physiology to local environments is necessary for an organism to mature and to execute a successful LHS. We have only recently begun to identify the epigenetic processes involved in this ontogenetic preparation for the future. The conditions experienced early in life are the best predictors of those likely to be experienced throughout life, and hence the LHS shaped by that early environment is likely to be the most advantageous LHS in later life (Vitzthum, 1990, 1997, 2001, 2009).[5] This process does not require any conscious decisions by an organism. Rather, among the ways in which an organism's biology might respond to environmental signals, natural selection will favour those responses that result in a relatively greater reproductive advantage.

Thus population-specific environmental conditions experienced during an individual's pre-adult development are likely to affect her reproductive functioning throughout adulthood. Women who developed in energetically demanding environments may be biologically acclimated to such conditions and hence are less likely to experience ovarian cycle disruption under a given energetic stressor than would a woman who developed in a more benign environment (Vitzthum, 1990, 1997, 2001, 2009). Metaphorically, women in industrialized populations are akin to hothouse flowers, cultivated under ideal conditions — small perturbations can have large negative effects until the ideal conditions are restored. In contrast, wildflowers must successfully reproduce in the typical environments in which they have grown, even if those conditions are more demanding compared to a hothouse. From the perspective of life history theory, there is nothing necessarily paradoxical about high fecundity and fertility under conditions that are energetically demanding (compared to some other environment) if these are the conditions in which the individual developed, and which are likely to persist for the rest of the individual's reproductive life. (This argument acknowledges that there are minimum energetic requirements for somatic maintenance and pregnancy below which successful reproduction does not occur.)

5 Similar arguments have been made independently regarding the psychological mechanisms underpinning animal behaviors that are flexibly responsive to environmental cues (Gigerenzer et al., 1999; Hutchinson and Gigerenzer, 2005; McNamara and Houston, 2009; Fawcett et al., 2012).

The centrality of age-specific mortality risk in the evolution and execution of population-appropriate LHSs suggests there are mechanisms that convey information reflective of mortality schedules. These mechanisms likely involve epigenetic and other physiological processes that influence reproductive physiology. However, the specific endogenous or exogenous signals that generate suitable epigenetic modifications of the genetic regulation of reproductive maturation and functioning are unclear. How does reproductive physiology become attuned to the local population's mortality schedule, or, put another way, how do mortality schedules become embodied within an individual?

For example, the timing of puberty, marking a shift of resource allocation from growth to reproduction, varies by several years within and between human populations and can change markedly in a single generation. What signal(s) prompts this transition at an age that is likely to be advantageous, given the current population-specific mortality risks? Whatever these signal(s) may prove to be, they likely have the following attributes:

1) Because of the evolutionary history of extant taxa, the same signalling mechanism is likely shared across multiple phylogenetic lineages (e.g. all primates or all mammals).
2) The signals must act early in life (before maturation is complete) so that the organism can grow and mature at the strategically "best" pace and age.
3) The signals are proxies for mortality schedules rather than the organism having experienced death itself (by which point the signal would be of no use to the organism).
4) Most (perhaps all) such signals are likely to be non-specific as to the causes of death because (given a specific time of death) the cause of death is irrelevant to one's lifetime reproductive success.

In 1993, Chisholm proposed a life history model linking childhood experiences of stress (as reasonable proxies for mortality schedules) and later reproductive strategies. Subsequent papers (Chisholm et al., 2005; Coall and Chisholm, 2010; Sheppard and Coall, in this volume) provided additional support for the model including evidence for mediation of the physiological pathways through the HPA axis. Working from different data and premises, Geronimus (1992) proposed the "weathering hypothesis" to explain ethnic differences in the US population in the timing of fertility. She attributed this and ethnic health disparities to the negative impacts of various stressors mediated by the HPA-axis (Geronimus et al., 2006). That these different models point to the same physiological mechanism linking mortality, health and reproduction to present and past conditions suggests that more study of the HPA-axis (in particular, its cross-talk with other physiological systems and epigenetic processes) could shed light on the links between ontogeny and the responsiveness of life history strategies to mortality schedules.

Concluding Remarks

The 150th anniversary of Darwin's publication in 1859 of *On the Origin of Species by Means of Natural Selection* was marked by worldwide celebrations of his far-reaching contribution. Mostly forgotten was that by 1900, for want of evidence, natural selection had relatively few supporters (Bowler, 1983). Rather, mainstream biology favoured several alternative explanations for evolution.

It is now a century since the beginnings of "The Modern Synthesis": the integration of Darwin's ideas about evolution and Mendel's work on heredity. This merger of theory and mechanism by Fisher (1918), Haldane (1924), Wright (1932) and others is rightly considered the foundation of contemporary evolutionary biology (Huxley, 1942), having spawned innumerable theoretical and empirical advancements in biology and other fields. Of late there have been calls for an "extended evolutionary synthesis" and other elaborations (Pigliucci and Müller, 2010; Jablonka and Lamb, 2014; Laland et al., 2015) of the Darwinian-Mendelian model that had been catapulted by the modern synthesis. In large part, these newest developments are an outcome of investigations into the mechanisms that build an organism and manage its functioning from conception through growth and reproduction to death.

Like the modern synthesis, these extensions and the empirical evidence supporting them are worthy of demographers' attention. Collectively they provide a roadmap to understanding the variability and plasticity of human biology and behaviour within an evolutionary framework without resorting to gene-centric reductionism or genetic determinism (which often yield unsatisfactory explanations for complex bio-behavioural phenotypes). While it is evident that humans, like any biological entity, are subject to and the result of evolutionary processes, behaviour within and between human populations is typically more than the simple expression of "a gene for" this or that phenotype (the same can be said for non-human species).

Obviously, the behaviour of an organism is never fully independent of its body. Behavioural and biological mechanisms share a fuzzy boundary across which a signal from one side of the border can prompt a cascade of responses on the other. Unpacking the interactions of biological and behavioural mechanisms is one particularly promising strategy for better understanding how extra-somatic as well as somatic factors influence reproductive functioning and generate demographic diversity.[6]

Consider, for example, lactational suppression of ovulation, a flexibly responsive bio-behavioural mechanism that is the very essence of being a mammal and one of the most influential determinants of variation in fertility. Breastfeeding in humans (WHO, 2009) is a coordinated behavioural repertoire of two persons: mum holds and guides the baby, who must latch on to and suckle the nipple. Mothers typically learn their part through repeated observation or active teaching from relatives and midwives. Reflexes in the infant's central nervous system prompt suckling, itself the physical stimulus that initiates the cascade of neuroendocrinological sequelae in mum's body that inhibits ovulation. But only to a point — mum could decide to breastfeed for years but nonetheless ovulation would return much sooner. This is not a consequence of her behaviour but of the mechanism itself, which has been shaped by natural selection to return her to a fecund state despite nursing an older infant. Mum could also decide not to breastfeed at all, but this can have negative health consequences for mum and baby alike, and for mum's reproductive success. Leaving aside additional points regarding the somatic and extra-somatic factors that influence a mother's decisions regarding nursing (Tully and Ball, 2011), it is evident that lactational suppression of ovulation is neither a strictly biological nor strictly behavioural mechanism, and thus its effect on fertility cannot be accurately assessed without considering both sides of the coin.

6 Recall that *extra-somatic* refers to social, cultural, economic, physical and biotic factors, and the behaviours of others.

At the beginning of this chapter, I proposed adding a fifth question to Tinbergen's four: How do the features of mechanisms vary within and across human communities worldwide? Quite a bit, as it turns out. Ages at menarche and at menopause vary by many years. Ovarian steroid concentrations in ovulatory cycles of healthy women differ severalfold without accompanying differences in fecundity or fertility. Rates of ovulation and early pregnancy loss vary seasonally and between populations.

This phenotypic variability reveals the flexibility of individual responses to local ecologies and the workings of life history strategies. Optional ovulation is a life history mechanism that prevents premature diversion of resources from a nursing infant or risky shifts in immune defences in a sexually abstinent woman, or allows foregoing the current opportunity for conception until conditions improve. Conception is not an irrevocable maternal commitment to reproduction. Gate-keeping mechanisms operating during implantation and the subsequent weeks prior to the LPPT afford opportunities for maternal termination of investment (early pregnancy loss) if the conceptus is flawed, or maternal status or extra-somatic conditions are not favourable. At these early stages, human reproduction is a "rent-to-own" contract with low exit costs.

There is practical as well as theoretical value in recognizing that physiological mechanisms have context-dependent outcomes, exemplified by the attempts to develop breastfeeding behaviour into a natural contraceptive. Much effort was invested in determining the best nursing pattern (suckling frequency and/or duration) for suppressing ovulation for the longest time post-partum (NFP, 1991). This goal proved unattainable. Researchers found that the same breastfeeding pattern did not have comparable impacts on fecundity in different women and populations, and that very different nursing patterns had similar suppressive impact (in mathematics, this is known as a "many-to-many mapping" from nursing pattern to ovulation suppression). This complex variation in signal and outcome precluded proposing a specific regime as a contraceptive. Nonetheless, if a baby's only food was from being breastfed whenever hungry, ovulation was suppressed for an extended period, though exactly how long was not predictable with the available data. Although frustrating for health care providers, these findings are consistent with life history theory (Vitzthum, 1994).

Numerous examples of phenotypic plasticity within and across populations challenge notions of a universal "normal" human biology. While there are barriers to good health in all human populations, it should not be assumed that all deviations from such norms (especially those established with data from WEIRD populations (Heinrich et al., 2010)) are necessarily pathologies requiring fixing. Furthermore, we should not necessarily expect there to be a universal "best" adaptation in all populations to an identical challenge to reproduction or survival. For example, Beall (2006) has documented three quantitatively different phenotypes in response to low barometric pressure among indigenous high-altitude populations in the Andes, Tibet and Ethiopia. These phenotypic variants likely reflect the different biocultural histories of the populations and may also be moderated by other features of the local ecologies.

Cross-population studies have also prompted interesting questions regarding ecomarkers (signals of extra-somatic conditions). In some instances, the change in an ecomarker may be more readily detected and hence a more salient signal than the specific state of the condition. In either case, the utility of the ecomarker will likely differ across contexts (for example,

directional changes in photoperiod would be a reliable ecomarker of changing seasons at high latitudes but not near the Equator).

The physiological mechanisms that underlie LHSs operate in ways that vary both within and between populations, and this variation can have profound demographic consequences. These mechanisms have evolved to respond to the changing availability of resources and the finite time during which an organism must develop and reproduce. Some essential biological, psychological and behavioural functions cannot co-occur; successful LHSs must juggle such incompatibilities regardless of the abundance of energy and/or other resources.

It turns out that evolutionary processes are more numerous and nuanced than a mere culling of the less fit and, as a consequence, reproductive functioning is more like a flexibly responsive behaviour than a well-tuned machine. The details of "how it works" are, as yet, only partially understood. Clearly there is much work left to be done and exciting revelations await.

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Glossary

biomarker: "A characteristic that is objectively measured and evaluated as an indicator of normal biological processes, pathogenic processes, or pharmacologic responses to a therapeutic intervention" (Biomarkers Definitions Working Group 2001); examples include hormone concentrations, birth weight, blood pressure and many others; biomarker choice and interpretation depends on many factors (Worthman and Costello 2009)

C-reactive protein (CRP): an acute-phase protein produced by the liver in response to signals from immune agents. Low circulating concentrations of CRP reflect a broad array of processes related to ongoing baseline somatic maintenance, but rise abruptly within about two hours of an acute insult

conspecific: members of the same species

corpus luteum: the transformed follicle that had enclosed the egg cell prior to ovulation

cross-talk: biomolecular communication between two signalling pathways

downregulation: in the context of hormones and their targets, the cell becomes less sensitive to the presence of a hormone because available receptors have become saturated due to hormone binding and/or receptor degradation (also see upregulation)

early pregnancy loss (EPL): natural termination within the first few weeks of conception

ecomarker: a reliable signal of extra-somatic conditions

endocrine system: comprises the internal organs that release hormones directly into the circulatory system in order to affect the functioning of more distant target organs

epigenetic: refers to structural changes of the chromosome that alter the expression of the genotype (and thus modify phenotype) without changing the DNA sequence

ethology: the study of behaviour (usually refers to non-human animal behaviour)

extra-somatic: external to the body (e.g. social, cultural, economic, physical and biotic factors and the behaviour of others)

fecundity: refers to the capacity to conceive

fertility: refers to the production of a live birth

follicle: the structure surrounding an egg cell prior to ovulation

follicular phase: the pre-ovulatory segment of the ovarian cycle

genotype: the genetic sequences in an individual that contribute to a phenotype

gonadotrophin releasing hormone (GnRH): is synthesized and released by the hypothalamus; stimulates the synthesis and release of gonadotrophins (LH and FSH) from the anterior pituitary

human chorionic gonadotropin (hCG): produced by the conceptus; binds to receptors on the corpus luteum which, as a consequence, maintains its production of progesterone essential to sustaining the pregnancy

hypothalamic-pituitary-adrenal (HPA) axis: the physiological system comprising the hypothalamus, pituitary and adrenal gland; plays a central role in responding to stressors

hypothalamic-pituitary-ovarian (HPO) axis: the physiological system comprising the hypothalamus, pituitary and ovary; plays a central role in the regulation of reproduction

life history strategy (LHS): a pattern of maturation, reproduction and resource allocation that is subject to natural selection

life history theory (LHT): an evolutionary framework for studying maturation, reproduction and aging, and the associated mechanisms underlying resource allocation of to these processes

luteal phase: the post-ovulatory segment of the ovarian cycle

luteal phase deficiency (LPD): a disorder that is said to be caused by insufficient progesterone during the luteal phase but that lacks definitive diagnostic criteria

luteo-placental-progesterone-transition (LPPT): a shift in progesterone synthesis during pregnancy from the corpus luteum to the placenta

norm of reaction: the range of possible phenotypes for a given genotype

ontogeny: the development of an organism from conception until death

ovulation: the release of the mature egg from a single follicle

phenotype: a morphological, physiological, behavioural or psychological feature of an organism; a phenotype may be determined only by genes, or solely by environmental factors, or by the interaction of genes and environmental factors

phenotypic plasticity: capacity for a genotype to express a variety of phenotypes

phylogeny: the evolutionary relationships among current and extinct species

somatic: referring to the body (also see extra-somatic)

upregulation: in the context of hormones and their targets, the cell's creation of more receptors so that the cell is more sensitive to the hormone (also see downregulation)

SECTION 4:
GENETIC EVOLUTIONARY DEMOGRAPHY

The following two chapters on genetic evolutionary demography provide an ideal one-two punch for bringing new readers up to speed on how genetics influence, and are influenced by, demographic patterns. Wachter, a key figure in evolutionary demography whose career has encompassed formal demography, biodemography, statistical methods and many other topics, covers genetic processes that have deep historical roots in the field: mutation accumulation, antagonistic pleiotropy and genetic load; each is explained in clear detail and related to current thinking in demography and genetics. One of the things Wachter does so well in this chapter is to give the reader a non-mathematical overview of the core concepts behind mutation accumulation, or the accumulation of genetic mutations with age. The immediate application of this is the theory of ageing, but the general surrounding discussion pertains to how natural selection operates on gene variants with "good", "bad", or neutral effects on fitness. These mechanisms are at the root of theoretical explanations for the origins and drivers of ageing patterns among humans and across species.

Mills and Tropf, sociologists who have broken ground with the quantitative analysis of genetic data, give us an excellent complement to Wachter's chapter, by providing an overview of their seminal research on how variations in the actual human genetic code are linked to observed patterns in fertility behaviour. This brings the reader up to date on this large and rapidly expanding body of literature, but also provides succinct methodological summaries behind some of the concepts we know as acronyms that occur in papers about genetic influences on behaviour, like SNP or GWAS (SNP is a single nucleotide polymorphism or a small unit of genetic difference among individuals with the potential to cause differences in observed phenotypes; GWAS is genome-wide association study, which is the needle-in-a-haystack statistical procedure for searching through vast amounts of genomic data to find the SNPs that are most likely to be consistently associated with observed behaviours or physical characteristics). They present us with the genetic evidence for a question commonly asked by the general public: are humans still evolving by natural selection? To which the answer is clearly "yes", because there are unique genetic variants that are positively associated with higher fertility traits.

However, we still understand little about exactly how genetic variants are associated with complex outcomes such as fertility. This means that any such research analysing links between genetic architecture and complex behaviours, such as fertility, needs to be very carefully interpreted, in order to guard against its misuse in eugenic ideology – a warning clearly stated by Mills and Tropf in their chapter. There are, therefore, many remaining puzzles to solve before we fully understand the links among fertility, genetics and fitness, in part because of the historical and ongoing process whereby human societies rapidly transition from high mortality and fertility to low mortality and fertility.

12. Genetic Evolutionary Demography

Kenneth W. Wachter

Since the 1990s, biodemographers comparing demographic schedules across divergent species have highlighted features in common, plausibly reflecting evolutionary influences in common. Optimal life history models and stochastic vitality models garner inspiration from Darwinian theory. Models for genetic load go further, explicitly incorporating the three fundamental processes of evolution — natural selection, mutation and recombination — and their consequences for genomes. These models draw age-specific demographic implications from assumptions about mutation accumulation. The genetic variants posited by the theory are now coming into observation in genomic data. A search is underway for contemporary effects of genetic load on measures of health, ageing and survival. It may be possible to tell how far an evolutionary heritage from deep in the past persists amid the altered environments of the present, shaping demographic regularities.

Evolutionary Ideas in Demography

With the rise of biodemography, evolutionary ideas have come to play leading roles in demographic thinking. The discovery of tapering mortality rates at extreme ages in Mediterranean fruit flies by James Carey and collaborators (Carey et al., 1992), in Drosophila by James Curtsinger and collaborators (Curtsinger et al., 1992), and in a coordinated set of research projects led by James Vaupel initiated three decades of empirical demographic studies of species with widely ranging body plans and life histories, and uncovered striking commonalities in the shapes of age-specific demographic schedules. It is natural to seek a source of commonalities in what all organisms have in common: Darwinian evolution.

In succeeding years, demographers came into contact with Darwinian thinking and especially with classical evolutionary theories of senescence. In tandem with empirical studies, three main strands of mathematical modelling began to flourish — optimal life history theory, stochastic vitality theory and mutation accumulation theory. These strands draw, respectively, on techniques from economic optimization, reliability statistics and population genetics, and they also contribute to these fields. Initially, the strands took somewhat separate paths. Today, they should be understood in combination.

A description of the three kinds of modelling is given in a paper by Kenneth Wachter, David Steinsaltz and Steve Evans (Wachter et al., 2014). That paper contains an account of many of the ideas in this chapter from a more formal point of view. In this volume, the first strand, optimal life histories, is featured in other chapters. The second strand, stochastic vitality models, enters at various points in other chapters. The third strand, mutation accumulation, is the principal subject of this chapter.

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Mutation accumulation theory differs in one major respect from the other strands: it explicitly models the genetic mechanisms that power evolution, including mutation, recombination and natural selection. It works with a mathematical representation of the genome and a set of formulas that connect genetic determinants with demographic rates. The other strands draw inspiration from Darwinian principles and implement criteria motivated by processes of natural selection, but, in most cases, they do not bring the nuts and bolts of genetics into their formulations. In those strands, arguments do not necessarily depend on genotypic determinants but may refer broadly to strategies and adaptations playing out in daily life.

Mutation accumulation, by contrast, is about genotypic determinants. As large samples of genotypic data become available, the elements of mutation accumulation theory can be confronted with those data and guide hypotheses. Rich empirical opportunities are opening up.

From the early days, biodemographers benefitted from the authoritative 1990 volume *Longevity, Senescence, and the Genome* by Caleb Finch, joined in 2000 by a comprehensive mathematical treatment by Reinhard Buerger, *The Mathematical Theory of Selection, Recombination, and Mutation*. They also built on the extensive works of Brian Charlesworth, (e.g. 1994), leading up to his influential paper (2001). In 1997, under the auspices of the Committee on Population (CPOP) of the U.S. National Research Council, *Between Zeus and the Salmon* (Wachter and Finch, 1997) crystallized biodemography as a field.

In parallel with the assimilation and extension of theory by biodemographers, and under the stimulus and guidance of Richard Suzman at the National Institute on Aging (NIA) groundwork was laid for the collection of genetic markers in social and demographic surveys. CPOP volumes *Cells and Surveys* (Finch et al., 2001) and *Biosocial Surveys* (Weinstein et al., 2008) sponsored by the NIA helped bring this goal to fruition, while *Offspring* (Wachter and Bulatao, 2003) sponsored by the National Institute of Child Health and Human Development expanded the purview from aging to fertility. The current breadth is shown by the latest CPOP-NIA volume *Sociality, Hierarchy, Health* (Weinstein and Lane, 2014) In happy confluence, as the theoretical reach of evolutionary demography has been extended, datasets pairing genomic measurements with social and behavioural variables, so-called "sociogenomic data", have become available in large quantities and high quality to empower empirical research.

Mutation Accumulation in Brief

Genetic variants fall into three categories with respect to natural selection: beneficial, neutral and deleterious. An allele is any one of the forms taken by a variant at a site in the genome. Many though not all variants are Single Nucleotide Polymorphisms (SNPs), differences in what can be pictured as a single letter in the genetic code. Mutations change the variant.

As for beneficial alleles, once introduced through mutation they tend to spread through a population and reach fixation, eventually no longer showing up as genetic variation but helping to determine design features of the organism. Once natural selection has done its job of driving a beneficial mutant allele to fixation, it bows out of the picture. As for neutral alleles, they increase or decrease in frequency at random in a population through the process of genetic drift, more slowly the larger the effective population size. Neutral alleles account for most observed genetic variation. As for deleterious alleles, they systematically decrease in frequency in a population. Mildly deleterious alleles decrease slowly under continual pressure from

natural selection, but their numbers are also slowly renewed by new mutations. They account for much, though not all, of the rest of observed genetic variation.

Mutation accumulation theory is a description of the representation and consequences of mildly deleterious alleles.

Deleterious alleles, always prominent, have recently been brought into the spotlight with the book *Crumbling Genome* by Alexey S. Kondrashev (2017), with its good background treatment of relevant genetics and recent results. For the study of deleterious mutations, a rich repertory of population genetic models exists, but mainly without detailed elements of demography. The model that Steve Evans, David Steinsaltz and Kenneth Wachter developed in an American Mathematical Society monograph (Evans et al., 2013), adds to the repertory by concentrating on and building in age-specific demographic structure. It is the model featured in this chapter. The mildly deleterious alleles it describes are genetic variants changing organisms in small ways that entail slightly less favourable age-specific rates of survival and fertility when their effects are averaged out over varieties of environments and over numbers of generations.

In mutation accumulation, alleles enter the population at slow rates, generation by generation, through new mutations. Alleles carried by parents are shuffled together and dealt out to offspring at each generation by the process of genetic recombination. Alleles are passed to descendants less frequently the lower the rates of survival and fertility they imply, enforcing natural selection. Alleles weeded out by natural selection are replenished by new mutations, and their representation typically reaches an equilibrium, "muation-selection equilibrium".

Sir Peter Medawar (1952) had the insight that bad alleles can be passed on more often if their bad effects are only felt later in the lifespan, after parents have borne and nurtured more of their potential offspring. Natural selection removes late-acting alleles more slowly from the population and leaves more of them around to accumulate at equilibrium. Here is one reason why evolution should favour mortality rates rising with age and fertility rates falling with age: keeping fewer early-acting alleles means being subject to lower mortality and less impaired fertility at early ages. Keeping greater numbers of late-acting alleles means being subject to higher mortality and more impaired fertility at older ages.

The power of this idea is two-fold. Firstly, it implicates something that all species have in common, namely natural selection. Thus, it is a plausible option for explaining cross-species commonalities in demographic outcomes.

Secondly, the mathematics of natural selection plays a central role in the predictions that emerge, to some extent overriding details in particular specifications. The mathematics comes from general principles of population genetics and does not much depend on *ad hoc* assumptions of constraints.

Each individual carries his or her own collection of mildly deleterious mutant alleles. That legacy is called "genetic load". Mutation accumulation theory envisions age-specific genetic load helping shape age-specific risk at the individual level.

Individual-level effects of genetic load are not to be confused with aggregate population-level effects of heterogeneity. Differences in genetic load within a population do constitute a form of heterogeneity. They do contribute to the heterogeneity in frailty and in observable risk factors familiar to demographers (e.g. Wachter, 2014a, pp. 185–97), and the effects of demographic selection within cohorts across the life course are not absent from the model. But they are a sideline, not the main story. Demographic selection occurs within each generation,

leaving its mark not on individuals but only on aggregate rates, whereas natural selection acts generation after generation on variability in genetic load, moulding an age trajectory for senescent mortality that reflects physiology at the individual level.

Mutation and natural selection act slowly, and loads we see today were honed long in the past. In the past, health impairments that are now survivable and even quite tolerable till late in life may then have had lethal consequences earlier in life. In humans, most senescent mortality is found at ages now well beyond the years of childbearing and childrearing. But genetic evolutionary theory proposes that we are seeing patterns in survival at late ages today that were imprinted at earlier ages over evolutionary time.

Concepts and Model

The model for mutation accumulation by Evans, Steinsaltz and Wachter (Evans et al., 2013) requires a fair bit of mathematics for a full description, but the concepts behind it can be explained without resort to formulas, which is the goal of this chapter. Attention is restricted to the setting most studied so far: the application to adult age-specific hazard functions. Each application depends on specification of a "selective cost function", a function that quantifies the difference that carrying a specific load of alleles makes to the chance that carried alleles are passed on to the next generation. Marginal selective cost is the difference that one extra copy of that allele makes to the chance. For the application here, the selective cost function is calculated in terms of decrements to the Net Reproduction Ratio, the "NRR".

Among demographers, the NRR is the most popular measure of population growth from generation to generation. It is also called the Generational Replacement Ratio. Properties are described e.g. in *Essential Demographic Methods* (Wachter, 2014, pp. 79 ff.). The NRR is preferred to the other popular measure, Lotka's intrinsic rate of natural increase, for reasons explained by Charlesworth (2000, p. 930) and by Wachter, Evans and Steinsaltz (Wachter et al. 2013, p. 10146).

Nurture as well as procreation affects the successful formation of each next generation. The NRR can be easily modified to incorporate effects of parental survival on the survival of their offspring. With more effort, selective costs can be defined to take account of grandparental nurturing of grandchildren — individuals who can carry their grandparents' alleles.

In its general form, the model applies widely to fertility and to infant and child mortality as well as to adult mortality. Mating success is as much, or more of a contributor to realized fertility as is fecundity; so complexities abound. The model further applies to alleles with stochastic effects, to sophisticated selective cost functions, and beyond. Such broader applications largely await future development.

How does the model for mutation accumulation work? Each allele is associated with an action profile, a non-negative function of age which is to be added to the hazard function for each individual who carries the allele in his or her genome. Alleles are labelled, not by their sites in the genome, but by their action profiles. They are gathered into teams. The alleles in each team share the same action profile. An individual's genetic load is specified by the number of alleles from each team that the individual carries. The individual's hazard function is calculated by adding up the increments to the hazard from each carried allele, added to a common, population-wide baseline hazard. The state of the population is represented by a probability distribution on the counts of alleles from each of the teams.

As a default option, pending future progress toward better options, the baseline mortality schedule may be chosen to be constant over age, representing extrinsic background risks of death. When alleles affecting fertility are not included in the model, the baseline fertility schedule may be chosen to be a fixed schedule compatible with empirical estimates from present-day hunter-gatherers. In this way, the shape of adult mortality is studied under provisional assumptions about the shape of age-specific fertility and about the levels of fertility and infant survival; that is, about the pace of recruitment in the population.

It may be reasonable to suppose that, over evolutionary time, homeostatic regulation responding to population density and resource availability maintained near-zero long-term population growth. In applications, the level of recruitment is often reset to be consistent with long-term zero growth.

In future research, considerations from other strands of evolutionary demography, from optimal life histories and stochastic vitality may help supply more realistic baseline schedules. These strands surely hold promise for understanding the physiological and adaptive contexts within which the age-specific action profiles of alleles come into being across the life-course.

Different timescales are involved. Stochastic vitality models largely speak to processes within single lifespans. Life history optimization involves trade-offs that may be consequential within one generation or a few generations. The trade-offs may be implemented by short-term phenotypic adjustments and adaptations, channelled by genetically determined pathways of influence, but not necessarily tied down to observable genetic variation. Mutation accumulation plays out over dozens or hundreds or even thousands of generations — long timescales over which natural selection leaves its mark on genetic variation.

It is essential to bear in mind that each new mutation occurs in the genome of an individual. Early humans lived in bands, but a new mutation does not occur simultaneously in the genomes of all members of a band. It occurs in an individual. On average, about half of the individual's children and a quarter of the grandchildren carry the new mutant allele, a little more if beneficial, a little fewer if deleterious, but a small portion of the band.

Beneficial alleles differ from deleterious alleles in their age-specific demographic relevance. Beneficial mutations are much less common than deleterious ones. Striking at random, it is easier to break than to improve. Low numbers mean there is less chance for small beneficial effects to cumulate into noticeable total impacts. Those who study beneficial mutations mainly focus, not on mildly beneficial ones, but on strongly beneficial ones. Strongly beneficial alleles spread quickly toward fixation and leave their mark as what are called selective sweeps. It is true that a mildly beneficial allele at any site runs a risk of extinction before fixation, and the risk does depend on the age-specific action profile. But recurrent mutations at the site blur this dependence. Beneficial alleles now fixed in the genome may derive from mutations so far back in time as to allow for multiple tries before success at fixation. Thus, Medawar's story connecting deleterious mutations to demographic schedules does not have a clear counterpart for beneficial mutations.

Recombination is essential to the demographic dynamics. While one-sex models are useful in some areas of population genetics, they are useless for understanding age-specific consequences of natural selection. Recombination makes it possible for some offspring to inherit lower loads of alleles than their parents, thus keeping a modicum of low-load genotypes in the population. The low-load genotypes anchor equilibria. Without

recombination, counts of deleterious alleles would have to trend upwards. In a finite population, this unhappy process is known as Muller's Ratchet and leads to collapse (Buerger, 2000, pp. 303--305). In infinite population models, a kind of renormalization of loads can let equilibria exist (Steinsaltz et al., 2005), but their properties are no guide to the realistic outcomes from two-sex models with recombination.

Technically speaking, the model for mutation accumulation being described here is an infinite-population model in continuous time. A long proof (Evans et al., 2013, pp. 51–110) shows that it is the limiting form of standard discrete-generation models from population genetics in a limit in which mutation and selection act more slowly than genetic recombination. Recombination is intrinsically a rapid process, with at least one and typically several recombination events per chromosome per generation. Mutations occur in every generation, but most are neutral. Those that act detrimentally, mildly and age-specifically on outcomes like adult survival are only a small subset of all mutations and enter the population at correspondingly modest rates. As for natural selection, mild action denotes, by definition, a slow response to natural selection. Thus, the assumption that recombination is rapid compared to mutation and selection is realistic in this context. The model is meant to apply over the substantial numbers of generations in which loads affecting demographic schedules are being shaped. Genetic drift, genetic dominance and back mutation are not treated in the formulation in the monograph (Evans et al., 2013) but extensions including back mutation are under study by others.

Although mildly deleterious alleles are being found in substantial total numbers in genomes, most alleles are neutral. The alleles relevant to mutation accumulation are sparse (Wachter et al., 2014b, p. 10850). They are well scattered across sites and across chromosomes. Genomic associations between nearby sites due to the process known as linkage disequilibrium can safely be ignored for this demographic application.

Among deleterious mutations, the model of Evans, Steinsaltz and Wachter treats those that mainly matter to the demography: those with effects that are mild, that is, not too strong and not too nearly neutral. Strongly deleterious mutant alleles head toward extinction possibly faster than recombination can thoroughly shuffle them, and possibly faster than the model predicts. Very nearly neutral mutant alleles have very small effects on demographic schedules. For them, the finite sizes over time of real populations matter. Genetic drift, not included in the model, gives extra help in removing the alleles or very occasionally lets them edge upward in frequency toward fixation.

According to the model, a randomly sampled individual carries a randomly sampled, Poisson-distributed load of alleles. The alleles are drawn randomly from each team, teams being labelled by their shared action profile. In fact, the alleles in each team are located at sites in the genome, and individuals can only carry zero, one or two copies at any site. The model envisions the count for a team being summed up over draws from many sites at which the deleterious alleles have low population frequencies. If some sites display high frequencies, differences between Binomial and Poisson sampling could introduce distortions. At each site, intrinsic randomness in family size and survival from generation to generation mean that a new mutant allele may quickly become extinct or may wander randomly upward in frequency for a while. The overall load from a team averages out over these random outcomes, site by site, and takes a smooth path through time predicted by the model.

Implications

Thanks to the simplifications achieved by passing to the limiting-form, continuous-time model, predictions of the demographic implications of mutation accumulation (Evans et al., 2013) are easy to compute. The inputs to such a computation are threefold: (a) a collection of functions of age that serve as profiles of age-specific action for each team of alleles; (b) overall rates of mutation per unit time from wild-type (predominant form) to deleterious form for each team of alleles; (c) baseline age-specific schedules for mortality and fertility. In the background is the assumption that the recombination mechanism satisfies some straightforward conditions, and does outpace mutation and selection.

Implementation requires code that computes hazard functions for the individuals in each subgroup that share the same counts of alleles from each of the teams, along with the implied Net Reproduction Ratios for each such subgroup. Formulas from the model then specify time derivatives of the proportional representation of each team of alleles in the population. In practice, efficient algorithms step through time in discrete intervals which should be seen as covering multiple generations — intervals long enough to show long-term average effects from alleles, but short enough to trace a smooth path of accumulation. A starting state without deleterious alleles under most specifications progresses toward an equilibrium. The population hazard function at equilibrium is the most informative output from the model. Examples of such predictions are presented in a 2009 paper (Wachter et al.).

In 2001, Brian Charlesworth recognized that simple specifications for mutation accumulation led to adult hazard functions tending to rise exponentially with age (Charlesworth, 2001). Such hazards are called Gompertz hazards, harking back to an 1825 paper by Benjamin Gompertz. In 1867, Makeham added a constant, age-independent extra term (Smith and Keyfitz, 2013, pp. 231–40). Gompertz and Makeham hazards for adults are ubiquitous. They are observed in countless species including our own. The question of how to account for them, and for their modification at extreme ages, is a central problem in formal demography. In Charlesworth's picture, the assumed action profiles can be highly stylized. The mathematics of natural selection does the work of turning featureless profiles into exponential hazards. Teams have to differ in ages of onset of the main deleterious effects, providing a mix of early-acting and late-acting alleles to fit into Medawar's paradigm.

Charlesworth made selective cost depend linearly on counts of alleles. Mutation accumulation, however, is an inherently non-linear process. When survival is depressed by the effects of some alleles, the reproductive potential left to be affected by additional alleles is smaller. This non-linearity makes the mathematics more complicated, calling for the extensive machinery and long proofs found in the monograph (Evans et al., 2013).

Happily, however, the most noteworthy implication from Charlesworth's treatment holds up in the full non-linear model: adult hazards mimicking Gompertz and Makeham fits arise naturally from stylized allelic action profiles. Furthermore, something intriguing occurs when action profiles are made just a little less stylized. Instead of assuming no effect at all up to some age of onset, one can assume small effects up to such an age, with the main effects coming afterwards. Such an assumption is generally enough to make hazard function trajectories start to level out at extreme ages. Such levelling out is also predicted in some of Charlesworth's own variants, but for different and less fundamental reasons. Hazard functions that level out are said to reach plateaus. Plateaus are commonly seen in

large populations of model organisms, and evidence for plateaus at extreme ages in carefully validated human datasets is growing (Barbi et al., 2018).

Mutation accumulation is a story about small effects — many and various — which only become visible when they accumulate in large numbers. There is little prospect for measuring the actual action profiles for specific alleles. But for the most part, within limits, the actual functional forms for the action profiles do not matter very much. The dynamics of the Darwinian process reward us with a degree of robustness to details of specification. Alleles with bigger negative effects are weeded out more quickly and are present at smaller frequencies at mutation-selection equilibrium. To a first approximation (before taking non-linear interactions into account), doubling the effect of each allele in a team halves the equilibrium frequency for alleles in the team, and the contribution to the hazard function remains nearly the same. This mechanism of compensation controls the cumulative impact. As a result, the predicted shapes of demographic schedules are being driven less by assumptions about action profiles and more by properties of the mechanism of natural selection itself.

This compensation mechanism does not adjust away any age-specific structure in the mutation rates themselves. Those rates are generally taken to be relatively unstructured, but that remains a hypothesis.

Demographers and statisticians have proposed a number of different explanations for Gompertz and Makeham hazards, along with a number of explanations for plateaus. These should be seen as contributing, rather than competing, explanations. Multiple kinds of processes plausibly play mutually supporting roles. But the striking feature of the explanation offered by mutation accumulation, not shared by most other approaches, is that the same process that predicts exponential rise also predicts tapering at extreme ages. Here is a unified explanation on the table.

There are other implications of the model with empirical importance (Wachter et al., 2014, pp. 10849–10851). They go beyond what can be described in detail here. In one direction, the model allows proof of a generalization to the non-linear setting of the identity known as Haldane's Principle. Haldane's Principle is an equilibrium relationship between the totalled-up selective cost of mutations and the overall mutation rate (Buerger, 2000, pp. 105 ff., 143 ff.). The former represents outflow of deleterious alleles, the latter, inflow, and they come into balance at equilibrium. When selective costs are calculated from demographic schedules via changes in Net Reproduction Ratios, the presence of other alleles alters the cost of any new allele. Outflow is no longer a linear, summed-up function of the separate costs of each allele. However, the non-linear interactions obey a more sophisticated version of Haldane's Principle, allowing inflow at equilibrium to be predicted from evidence bearing on selective costs.

Probabilities of survival cannot exceed one. Mortality rates cannot be less than zero. Consider any given set of age-specific rates of fertility and mortality. We can start with what the Net Reproduction Ratio would be in the absence of all adult mortality. The portion of adult mortality contributed by genetic effects of deleterious alleles brings down the Net Reproduction Ratio by some unknown amount. That is the selective cost of the alleles, when selective cost is being measured by decrements to the NRR. We can then notionally include all the other contributions to adult mortality, external and internal. They further reduce the NRR down to its value in the presence of adult mortality calculated from the age-specific rate schedules. The

reduction to the NRR from some portion of adult mortality has to be less than the reduction to the NRR from all adult mortality, so we have a way of putting an upper bound on the selective cost of those deleterious alleles that affect adult mortality.

Measurements of survivorship and fertility from anthropological field studies of hunter-gatherer populations give some exemplars of age-specific human schedules that could have prevailed over evolutionary time (Gurven and Kaplan, 2007). Combined with an assumption of near-zero rates of long-term population growth, this evidence allows us to implement upper bounds on selective costs, and so, via Haldane's Principle, to obtain bounds on mutation rates for that subset of deleterious alleles affecting adult age-specific hazard rates.

Such calculations show that mutation accumulation theory passes a rough consistency check, since bounds on mutation rates for this subset of deleterious alleles come in below estimates (Kondrashev, 2017, p. 109) for total mutations contributing to genetic load. These estimates of flow can also be compared with estimates of stock: estimates of numbers of mildly deleterious mutant alleles present in the human genome, discussed in the final section of this chapter. Together, estimates of flow and stock can be combined into estimates of average antiquity for alleles observed today.

Common Misunderstandings

In appreciating the place of mutation accumulation in evolutionary demography, it is essential to avoid four common misunderstandings.

Sometimes it is imagined that demographic models for mutation accumulation posit age-specific triggers for action from alleles, requiring an implausible age-based clock. By no means! The age-specific action profiles should rather be pictured as net outcomes that emerge gradually from slight differences in physiological processes. As processes work themselves out over the life-course, small genetic differences leave their mark on the eventual mix of ages at death. Any effect of genes on fitness necessarily has some age-specific signature on fertility and mortality.

A leading framework for understanding senescence is the "disposable soma theory" developed by Thomas Kirkwood (1977) and extended by many others. Sometimes it is suggested that mutation accumulation theory is at odds with disposable soma theory. By no means! The one builds on the other. Prime examples for mutation accumulation are mutant alleles that slightly reduce the efficiency of investments in maintenance and repair, in growth and in reproduction just as described within the disposable soma framework.

Antagonistic pleiotropy is a term that applies where the same genetic variant has multiple (pleiotropic) effects working in opposite (antagonistic) directions at different ages; for instance, trading off advantages at younger ages against debilitation at older ages. Sometimes mutation accumulation and antagonistic pleiotropy are regarded as mutually exclusive alternative explanations of senescence. By no means! The two complement each other. Mutation accumulation accommodates any alleles with a pleiotropic mix of negative and positive effects on fitness, so long as the net effect in relevant environments is negative. Antagonistic pleiotropy takes centre stage when the net effect is positive. In that case, mutant alleles typically head toward fixation. Mutation accumulation emphasizes persisting genetic variation in mutation-selection equilibria. Antagonistic pleiotropy takes over for understanding systemic properties at fixation or headed toward fixation.

Sometimes an impression lingers that mutation accumulation typically brings a threat of population collapse in which hazards diverge over time toward infinity. By no means! Typical predictions are for well-behaved equilibrium states with adult hazards exponentially rising with age and levelling off into plateaus. They mirror patterns familiar for humans and many organisms.

Misleading impressions about the salience of collapse arose when proofs of collapse under contrived conditions were offered in mathematical papers (e.g. Wachter et al., 2013). The proofs were offered for the purpose of dispelling any notion that the new nonlinear models were just fancy ways to obtain the same qualitative predictions as the linear models of Charlesworth that inspired them. The proofs remain of theoretical interest but should not draw attention away from realistic cases. Collapse is easily avoided, and the ease with which it is avoided is itself illuminating.

Genetic Load and Socio-Genomics

The existence of the process of mutation accumulation is well-established. With the burgeoning of genomic data, geneticists routinely observe mildly deleterious alleles, numerous in total, although sparse among all variants. Some are present at sufficient frequencies to qualify as Single Nucleotide Polymorphisms (SNPs) while others at lower frequencies qualify as Single Nucleotide Variants (SNVs). In line with expectations, the loads carried by individuals are heterogeneous. An early comprehensive report is found in a 2012 paper in *Science* (Tennessen et al., 2012).

Any deleterious allele acting over the long term necessarily has some age-specific profile of action, either uniform or structured. It stands to reason that some genetic imperfections manifest themselves earlier in life than others. It is readily imagined that it may be easier for physiological adjustments to postpone rather than eliminate ill effects. Something like ages of onset may show up often in action profiles. As mentioned already, however, the kinds of effects at issue are too small to be observed directly, allele by allele.

Some handfuls of single nucleotide polymorphisms have effects on contemporary measured traits that achieve statistical significance in Genome-Wide Association Studies (GWAS). These are not the kinds of SNPs or SNVs involved in mutation accumulation. Mutation accumulation makes its mark with much larger numbers of much more nearly neutral SNPs and SNVs. They show up in aggregate in the background, likely accounting for the lion's share of so-called "hidden heritability". Hidden heritability is the difference between the heritable portion of variance in a trait inferred from twin studies or parallel methods, and the portion visibly accounted for by genetic variants with effects large enough to be detected. The SNPs and SNVs at stake in mutation accumulation would not achieve genome-wide statistical significance, but they would contribute to the weak pervasive correlations that make constructs called polygenic scores useful predictors of traits like height, educational attainment and cognitive status.

There are arguments (Wachter, 2014b) for expecting that GWAS-significant SNPs reflect interactions between specific features of modern environments and genetic propensities. Alleles with large negative consequences for fitness over hundreds of generations would mostly have been weeded away by natural selection. Any allele now affecting health in detectable ways can mainly only be on the scene today if it is affecting health in new ways.

Newspapers feature speculations about alleles inherited from Neanderthals that were good for surviving Ice Ages and are now bad for patrons of fast-food malls. Varying environments across space, and fluctuating environments across time complicate any attempt at a general account, but, by and large, contemporary conditions may be expected to have a prominent role in the large deleterious genetic effects visible today.

Small imperfections are another matter. Genetic programs for basic biochemical and cellular processes, for systems of resource deployment and for systems for repair have roots far back in evolutionary time. Variants that induce slightly less efficient or resilient versions of these processes could have been affecting health and viability over many generations and still be doing so today.

It therefore seems worthwhile to interrogate genomic data for evidence of associations between genetic load and health and demographic outcomes. For such an investigation, two kinds of measures are needed: firstly, measures that identify sets of mildly deleterious alleles in the genome, and, secondly, measures of present-day traits and conditions that can serve as proxies for components of health and survival that could have been relevant to Darwinian fitness over evolutionary time.

Geneticists have been developing a repertory of methods for distinguishing effectively neutral alleles from deleterious or favourable alleles subject to selective pressure. Seven variants of such methods are used as criteria in the paper already mentioned by Jacob Tennessen and twenty-two co-authors, (Tennessen et al., 2012). The distinction between deleterious and neutral depends on effective population sizes over appropriately early spans of time. Generally speaking, there are two kinds of information to exploit: the first based on the phenomenon of sequence conservation across species, the second on inferred impacts of mutations on protein structure and function. An example of the first is Genomic Evolutionary Rate Profiling (GERP) (Davydov et al., 2010). An example of the second is the Polyphen family of indices (Adzhubei et al., 2010).

The GERP approach takes advantage of the occurrence of stretches of genetic code that are highly similar across a number of species, in this case thirty-four species of mammals. These "conserved sequences" can be aligned with each other. For many sites in the genome, it becomes possible to say which species today share the same allele at that site, either entirely or mainly. These readings can then be combined with reconstructions of the phylogenetic tree of life, detailing how species of today have descended and split off from ancestral species. Statistical methods allow us to work back to guesses at the alleles found in the ancestral species, and so to count how many times across the tree one form has substituted for the other. If the site is one with neutral alleles, then the substitutions are expected to be due to the slow process of genetic drift, and the expected number of substitutions can be predicted. If the site is one with a mildly deleterious allele, natural selection will make it harder for that allele to have drifted to fixation as many times as for neutral alleles. The upshot is a criterion for distinguishing neutral from deleterious alleles.

The Polyphen approach supplements information on conserved sequences with knowledge about the effects of DNA mutations on amino acids and functional properties of proteins. The upshot is an alternative score with a mix of advantages and limitations.

These approaches give demographers analysing genomic data a basis for marking and counting up numbers of mildly deleterious alleles carried by each individual. Different criteria

yield different indices. All indices are subject to wide margins of uncertainty, but geneticists are continually developing new and improved strategies.

The other part of the demographic endeavour is the collection and compilation of measurements of traits. The traits are intended to supply sensible proxies for health conditions that could bear on survival and fitness in the evolutionary settings of long ago. However, sample sizes for single detailed surveys, like the Health and Retirement Study and its counterparts, are too small to allow for estimates of genetic effects that are not underpowered. The only practical option at the present time is to combine measurements of the same trait across a number of large surveys which also collect comparable genomic data. The drawback is that the range of traits measured in comparable ways across surveys is narrow, although rapidly expanding. Educational attainment and cognitive assessments were among the first. Only a few studies have usable data on adult mortality, since respondents who have been recently genotyped had to be alive at genotyping. Present-day longevity would also not necessarily match up with traits crucial for survival under the conditions of long ago.

Counterbalancing these limitations is a piece of technical good luck which can stand demographers in good stead. The outputs produced by the consortia carrying out studies with combined samples typically report coefficients and standard errors for constructs called polygenic scores. As long as these coefficients have been computed in their original simple form, without various complicating refinements, the reported outputs are sufficient for demographers independently to compute regressions and other analyses relating their indices of genetic load to the measured traits.

Pilot studies exploring this research program have been conducted by the present author in collaboration with Iain Mathieson, now at the University of Pennsylvania, and Amal Harrati, now at Stanford Medical School. For the most part, associations of indices of individual genetic load with available traits, educational attainment, and an index of cognitive age have not been statistically significantly different from zero. Those null results remain unpublished. However, the traits so far examined are hardly good proxies for components of evolutionary fitness. Richer and more appropriate data are likely to become available in the future. The line of investigation remains promising.

All the strands of evolutionary demography are rich in theoretical insights and engaged with data of many kinds. Mutation accumulation plays a special role, because the elements explicitly modelled — mutation, recombination and natural selection — are the elements directly reflected in the genomes of members of populations now subject to study. This chapter has described a way in which evolutionary demographic thinking makes contact with today's empirical genetics. Much is to be learned.

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13. Genetics and Reproductive Behaviour: A Review

Melinda C. Mills and Felix C. Tropf

Fertility and reproduction have been core topics across multiple disciplines, including the study of reproductive behaviour outcomes such as tempo (timing) and quantum (number) of fertility, but also fecundity, infertility and reproductive development. The aim of this chapter is to provide a comprehensive and introductory overview of the central theoretical and empirical approaches to the study of the genetics of human reproductive behaviour and review key findings. We start with a brief definition of fertility and reproduction, followed by an overview of interdisciplinary approaches and findings. We then explore why it may be useful to adopt a biodemographic and genetic approach to reproduction, the central empirical methods that have been used, core findings to date, and conclude with a discussion and reflection on future directions of research.

Introduction

Fertility and reproduction have been central topics in the disciplines of (evolutionary) demography, sociology, anthropology, biology, medical sciences, and genetics. Broad interest likewise stretches across human, plant and animal studies. This chapter focuses on human reproductive choice, which includes the study of outcomes such as the timing and number of births. These are often also related to development traits such as the onset of menarche or menopause, the onset of sexual behaviour, and infertility related diseases. Although the majority of research on this topic within the social sciences has focused on social science and environmental explanations (Balbo and others, 2013), there is a growing body of research that adopts a biodemographic or sociogenomic approach (Mills and Tropf, 2016). Although reproductive behaviour has been largely linked to choice and decision-making — thus regarded as highly socially-determined — a growing amount of evidence highlights the importance of biological and genetic factors, which have been shown to be intertwined with social determinants and behavioural aspects of reproduction.

The aim of this chapter is to provide an interdisciplinary overview of the burgeoning genetics of reproductive behaviour literature, take stock of the central findings, and suggest promising areas of research in the future. We review work primarily in the areas of demography, sociology, and genetics, but with some attention to related disciplines and research in evolutionary biology and anthropology, reproductive medicine, psychology, and behavioural and molecular genetics. This is an introductory chapter aimed at providing an overview. For more specific reviews about research on the genetic association between fertility and psychological traits or on leveraging

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results from genetic discovery studies for evolutionary research (both topics we touch upon in this chapter), see also Kim and Lee (2019) and Guo and others (2018).

The current chapter provides an overview of this research to date starting with a brief definition of fertility and reproduction, the link with natural selection, stark differentiation of this research from historical eugenics and a brief overview of socio-environmental explanations. We then turn to a summary of the central behavioural and molecular empirical approaches, together with core findings. This is followed by a reflection regarding the differences of genetic effects in relation to certain country or birth cohort contexts and between the sexes. We then conclude with a discussion and reflection.

Defining Fertility and Reproduction

The terms fertility and reproduction take on different meanings in demography and sociology, reproductive medicine, and genetics. In demography, fertility refers to the actual bearing of live births. Demographers and sociologists often discuss two interrelated aspects of fertility, namely the "quantum" or actual number of children individuals have over a certain period, and the "tempo" or timing of when they have these children (Bongaarts and Feeney, 2000). Tempo is obviously highly related to quantum since the delaying of first births may result in a lower quantum or number of children. For this reason, we often use fertility and reproductive behaviour interchangeably throughout the chapter. Especially when reviewing the literature, we focus on the number of children ever born (NEB) as a measure of fertility quantum and on age at first birth (AFB) as a measure of fertility tempo.

In reproductive medicine, "fertility" is used in a different manner and related to the ability of individuals and couples to conceive. Infertility denotes the ability/inability of couples, women or men, to conceive and have children given unprotected intercourse (Joffe, 2010), while in demography this is signified by the terms (in)fecundity or sterility. In *biological research*, the focus is often on lifetime reproductive success (LRS) (Byars and others, 2010) or the number of offspring (Zietsch and others, 2014), which is what demographers refer to as "quantum" or the number of children ever born. In *evolutionary research*, fertility quantum is often used as a surrogate measure of "fitness". If the number of surviving (and reproducing) children of an individual is computed relative to those of their peers of the same birth cohort, this might indicate *relative* reproductive (dis-)advantages for individuals and has been use as a proximate measure for *relative* fitness (Kirk and others, 2001; Stearns and others, 2010). This in turn is used to measure how far the fertility quantum leads to relatively higher chances of successfully transmitting genes to the next generation. This link to fitness means that fertility has vital consequences for the study of natural selection and evolution, while fertility quantum remains a largely imperfect proxy for fitness as discussed in more detail elsewhere (Mcgraw and Caswell, 1996; Jones and Bird, 2014).

Fertility, Natural Selection, and Evolution

Improvements in hygiene and the reduction in prenatal, infant and child mortality in industrialized societies means that the number of children ever born (i.e. quantum) has emerged as a readily available proximate measure for lifetime reproductive success (LRS) relating (imperfectly) — see also Mcgraw and Caswell, 1996; Jones and Bird, 2014) — to fitness (Stearns and others, 2010). This refers to Fisher's (1930) fundamental theorem of natural selection (Fisher, 1930), which

states that because fertility is highly correlated with fitness that its heritability at equilibrium should be, in theory, close to zero. As we demonstrate shortly, however, a series of studies have produced evidence that this is not the case.

Non-zero heritability of fertility indicates ongoing natural selection. If specific genetic variants are associated with higher reproductive success, they are passed on to the next generation more often than others and we expect them to become more frequent in future generations. A couple of studies therefore explored whether genes, which are associated with number of children ever born, are also associated with other traits. If genes that increase height, for example, are also associated with having more children, we expect future generations to be (genetically) taller than current ones (Stulp and others, 2015). A number of studies therefore used both twin data and, more recently, molecular genetic data to "live-track" ongoing human evolution (Milot and others, 2011; Kirk and others, 2001; F C Tropf and others, 2015b; N. Barban and others, 2016; Sanjak and others, 2017).

As we have argued previously, there are several reasons to remain cautious about predictions of the actual evolutionary change that we can expect from previous findings of ongoing natural selection in humans (Courtiol and others, 2016). Firstly, the relationships between genotypes and phenotypes remain poorly understood. Secondly, comparable phenotypic information is often not present across multiple generations. Thirdly, much of natural selection on contemporary human populations is driven by cultural and environmental factors that themselves change very rapidly. Only selection sustained in one direction over many generations produces significant genetic change. Fourthly, while it is imperative to measure physiological changes across generations, their relevance for population characteristics such as average number of children, education, body-size or heart rate are (most likely) negligible in the short term compared to cultural changes.

As discussed more extensively in other chapters in this volume, natural selection in humans is often studied using several key traits or phenotypes, such as height, which can be measured with or without the use of genetics. As reviewed elsewhere (Courtiol and others, 2016), this ranges from the simplest design of a twin or family model that measures how much variation is attributed to genetic differences between relatives to the use of actual whole-genome data.

Is Adopting a Genetic Approach to Fertility Related to Eugenics?

There has been a reticence to adopt a genetic or biological approach to fertility, particularly in some disciplines and quarters, due to the assumption that it may be linked to eugenics. It is essential to clarify that the research described in this chapter and conducted by these researchers is not related to eugenics and we actively oppose this link. As we have previously noted elsewhere (Mills and Tropf, 2016), there is a dark history of eugenic policies that emerged in the 1880s and that were linked to atrocities in recent history. Eugenics focused on so-called "improvements" that could be made to humanity via supposedly scientific methods that were misguided and incorrect and involved selective breeding. The aim of the eugenics movement was "to affect reproductive practice through the application of theories of heredity" (Levine and Bashford, 2010, p. 3). The aim was to prevent life (sterilization, contraception, abortion), make life "fitter" (training, rearing of children, public health) and promote pronatalist goals, but also, at its most extreme, to end life (so-called euthanasia

of the disabled) (Levine and Bashford, 2010). The eugenic approach has been widely, and rightly, condemned by all serious scientific audiences. It is essential to note that the type of research described in this chapter and within the mainstream of contemporary peer-reviewed research in behavioural and molecular genetics has no eugenic goals or ties. Considering this grave history of linking eugenics with fertility, however, we continue to find it essential to explicitly acknowledge this point and to be vigilant in order to prevent similar abuses in the future.

Socio-environmental Predictors of Reproductive Behaviour

Fertility and reproduction, as discussed in this chapter, remain largely behavioural outcomes, related not only to genetics and biology, but also influenced by individual and partner-level choices and preferences, and institutional environments. For this reason, although we focus on the "genetics" of fertility, we acknowledge that it is one piece of the puzzle and that socio-environmental predictors will be the strongest predictors in many cases. Various reviews have examined the core factors that predict fertility outcomes (Balbo and others, 2013; Mills and others, 2011), which we briefly summarize here. Factors influencing fertility are generally divided into three theoretical levels of micro- or individual factors, meso-level, which includes the family level, for example, and macro- or societal-level factors.

The core micro-level factors that impact fertility have been identified as *partnership formation*, including instability and quality of partnerships, multiple partnering and re-partnering and the emergence of different types of partnerships such as cohabitation (Billari and Kohler, 2002; Mills and Blossfeld, 2005). Partnering often impacts the timing, postponement and ultimately the number of children. *Education* is also a prominent predictor, usually based on Becker's classic theory of human capital (Becker and Becker, 2009). Education levels, particularly for women, are likewise seen as key in fertility decision-making, linked to opportunity costs, impact of enrolment and role conflict, as well as the field of education chosen, with most studies examining how higher education results in fertility postponement (Tropf and Mandemakers, 2017). *Economic and employment uncertainty* are also key, building on Easterlin's theory of economic deprivation (Easterlin, 1976), which posits that in historical periods of general economic uncertainty and rising unemployment, individuals will forgo partnering and fertility. It relates to the "affordability clause" to have children (Rindfuss, Ronald R., Vandenheuvel, 1990), with multiple empirical studies demonstrating how economic, employment and temporal uncertainty results in family formation postponement (Mills and others, 2005).

Perhaps the most relevant for this current review is the body of literature on the *intergenerational transmission of demographic behaviour* and, in particular, fertility. Empirically speaking, this work often compares the similarities of particular fertility-related events (menopause, age at first child, number of children) across successive generations. They then mostly observe a moderately positive correlation between parents and their offspring. The bulk of this research has focused on number of children (Murphy and Wang, 2001) and the tempo of fertility — mainly the intergenerational transmission of teenage motherhood (Kahn and Anderson, 1992). Others have examined how parents transmit value, preferences, attitudes and contraceptive knowledge (Rijken and Liefbroer, 2009). This relates literature that examines the socioeconomic status of the family of origin, often

finding a negative relationship between the father's education or the mother's levels of employment (Balbo and others, 2013).

Meso-level factors have also been shown as important predictors, including social networks and interaction which involves *social learning* (to gain knowledge, for example about contraceptives or what it is like to be a parent) and *social influence* (how peer groups impact attitudes and behaviour) (Balbo and Barban, 2014). *Social capital* and access to resources such as goods, money, ability to help or power have also been shown as important predictors (Balbo and Mills, 2011). The *gendered division of labour at the household level* has likewise been shown as an important factor regulating fertility (Mills and others, 2008).

Macro-level societal factors are also core predictors of reproductive trends. This includes the focus on *economic period effects* such as the commonly observed pro-cyclical relationship between economic growth, recessions and fertility (Sobotka, Tomáš, Skirbekk, V., Philipov, 2011). This is often strongly related to research on *employment trends and the impact of employment*. *Social policy measures* and *welfare regimes*, including labour-market, family and market constellations, tax and housing have also been extensively evaluated, but with mixed results in relation to their direct or causal impact on postponement and number of children (Mills and others, 2011). Larger *value and attitude changes* in addition to the *widespread* "*contraceptive revolution*" characterized by the second demographic transition has also been touched upon as an important explanation for the postponement and foregoing of children (Lesthaeghe, 1995).

Biodemographic and Genetic Approaches to Fertility

Although we continue to acknowledge the strong impact of socio-environmental factors on reproductive behaviour, a growing number of researchers argued for some time that fertility may be influenced by an individual's genetic architecture and beyond, such as proteins, hormones, neurons, gametes and other factors (Udry, 1996; Wachter and Bulatao, 2003; Wachter, 2008; Freese, 2008). The importance of biological factors underlying fertility was recognized by early demographers in their recognition of key "proximate determinants" of fertility, including fecundability, contraceptive use, exposure to intercourse or sterility (Bongaarts, 1978). Some of the earliest calls to integrate genetic considerations into demographic fertility research were by Udry (Udry, 1996), who was able to think beyond the existing data constraints of the period to hypothesize plausible relationships between reproductive biology, social environment and fertility. He acknowledged that not only fertility outcomes, but also behavioural choices and motivations to have children were likely guided by genetics and hormones. This was followed by a larger biodemographic focus on this topic at the start of the 2000s (Wachter and Bulatao, 2003; Rodgers and others, 1999; Rodgers and Kohler, 2012). Biodemographic and genetic approaches to fertility can be roughly divided into two types of research: behavioural genetics, which adopts a twin and family design, and molecular genetics, which uses whole-genome data, often from unrelated individuals using various methods.

Behavioural Genetics: Twin and Family Studies

A series of early twin and family studies, mostly in demography, have linked biological and genetic components to fertility behaviour (Kohler and others, 1999; Kohler and Rodgers, 2003; Rodgers and others, 1999; Kirk and others, 2001). Adopting a "twin design", they separate the genetic (i.e. examining monozygotic twins) and shared (i.e. growing up in the same household) or non-shared environment. Monozygotic twins are genetically identical, sharing 100% of the same genetic material while dizygotic twins — just as any brother and sister — share on average around 50%. If monozygotic pairs are more similar in their fertility behaviour in comparison to dizygotic twins, this is interpreted as a reflection of genetic effects. The extent to which genes influence a certain behaviour or disease — the "heritability" — is quantified as the proportion of variance in that trait within a population, which is explained by genetic variance (Visscher and others, 2008). The simplest way to estimate heritability is to subtract the correlation in a trait between dizygotic twins from the correlation between monozygotic twins and multiply the result by two (see also Snieder and others (2010) for a very short but quite comprehensive introduction to simple twin modelling). The central premise is that genetic and biological dispositions of individuals influence fertility either directly via genetically mediated variations, or, since many aspects regulating fertility possess considerable volitional control (e.g., decision of age at first birth, fertility preferences), via underlying temperament or personality influences on fertility decisions.

Figure 1 provides an overview of key studies to date that have examined the heritability mainly of the number of children ever born (NEB) and age at first birth (AFB) across different countries and birth cohorts. We see that there is more information on women as opposed to men, which is typical in this area of research and often related to data-gathering customs. We likewise observe that the heritability for AFB women ranges between just over 0% to 35% of the observed variance within these birth cohorts (i.e. 0.002 Denmark 1931–52 to 0.35 UK 1930–39). For the NEB for women, the range is 24–43% and for men between 24–28%. A recent meta-analysis of all twin studies conducted until 2015 suggests that across all fertility traits studied, on average around 30% of the variance is associated with genetic differences in a population (Polderman and others, 2015).

Key studies include early work in Denmark (Rodgers and others, 2001) that found around a 30% heritability of number of children ever born, which was later replicated in Sweden (Zietsch and others, 2014). Others have estimated a heritability of around 26% for women in Finland (Nisén and others, 2013), the UK (Tropf and others, 2015a), and Australia (Kirk and others, 2001), whereas others have found no effect in the US (Neiss and others, 2002) nor in some birth cohorts in Denmark (Rodgers and others, 2008). The twin studies also show considerable variation between the sexes, across countries and birth cohorts, which we return to in our discussion of GWA studies (genome-wide association studies) later in this chapter.

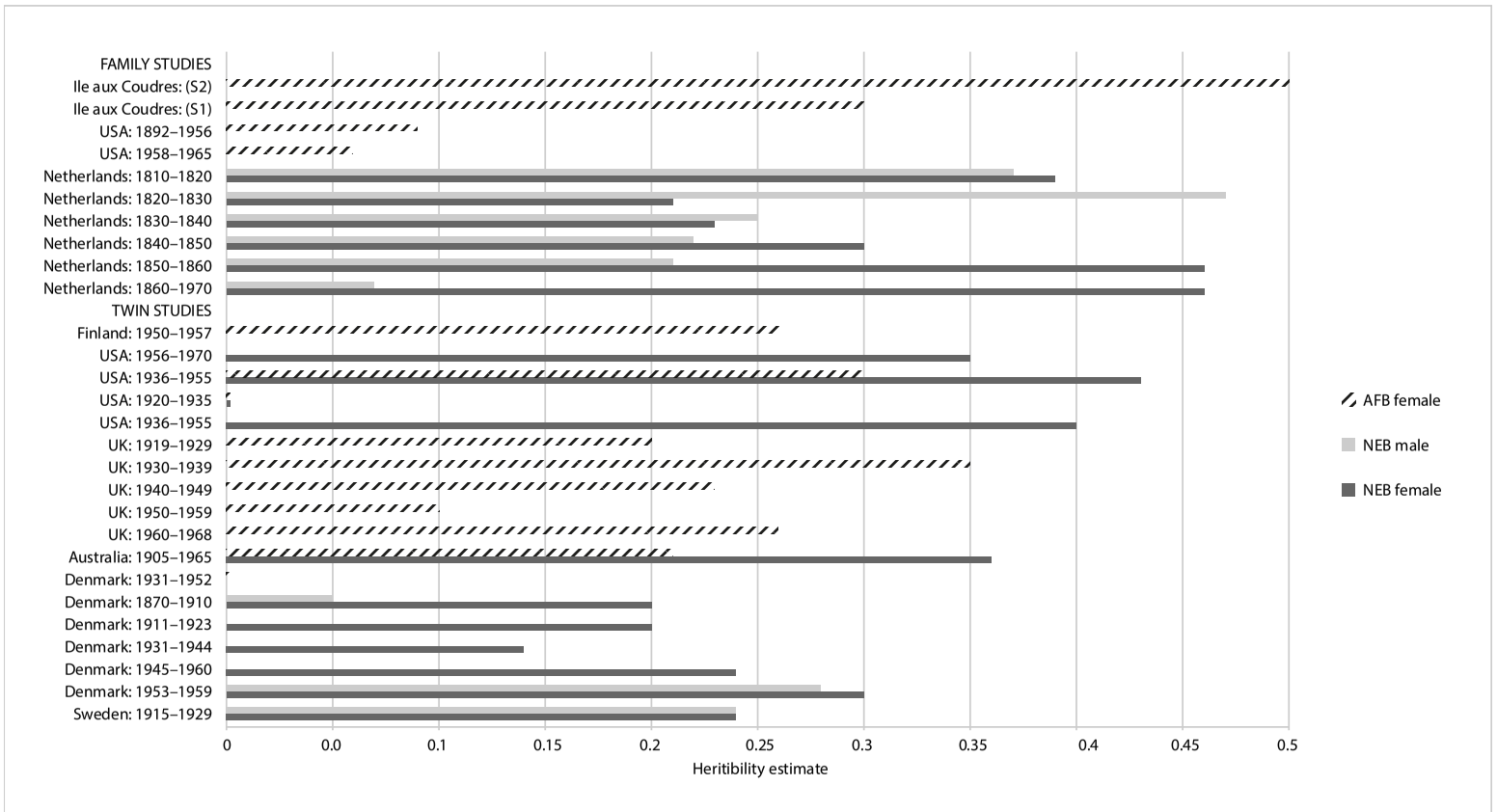

Figure 1 Summary of fertility heritability estimates by birth cohort and country by fertility trait: (AFB) age at first birth, (NEB) number of children ever born. Source: Adapted from Mills & Tropf (2015) and Barban et al. (2016), (produced by authors).

Molecular Genetic Approaches

Recent advances in methods and the widespread availability of large sociogenomic datasets has resulted in a rise of studies adopting a genetic approach. Whereas behavioural genetics focused on whether fertility has a genetic basis and if so, to what extent it is heritable — and suffer from several strong assumptions and practical limitations (Tropf and others, 2015; Nolte and others, 2019) — molecular genetics attempts to isolate where the genetic variants are located, in addition to a focus on the structure and function of DNA, and the generation of individual based genetic scores predicting fertility behaviour (Mills and others, 2018). These genetic variants are called single nucleotide polymorphisms (SNPs) and allow us to explore the data in new ways by applying novel statistical tools.

Candidate Gene Studies

An initial and early approach applying molecular genetic data was the candidate gene approach, which has an *a priori* hypothesis about the underlying biological pathway of a trait and directly focuses on a gene or set of markers. This was often due to the fact that certain datasets only genotyped smaller areas on the genome, offering only limited genetic markers also for small sample sizes. Although this technique is still used when the results are derived from a large GWAS (genome-wide association study), discussed shortly, previous work has been heavily criticized for producing false positive results. In this type of candidate-gene approach, genetic variants were compared with a sample of individuals (treatment group) that had the genetic marker with those who did not (control group). Although there are no direct candidate-gene studies on core fertility traits, several early studies examined sexual behaviour (Guo and others, 2008; Halpern, 2000) and contraceptive use (Daw and Guo, 2011), generally in relation to hypotheses related to risky behaviour and sensation seeking and linking it to the dopamine receptor or serotonin transporter. These studies have now been criticized for small sample sizes and lack of statistical power, false positives and biased positive results (Ioannidis, 2005).

GREML Studies

The increased availability of genome-wide molecular genetic data across the whole genotype for a larger number of individuals was coupled with new analytical techniques. A core advance is the Genomic-Relationship-Matrix based restricted maximum likelihood (GREML) method, which produces a more direct estimate of heritability using single genes across the whole genome for unrelated individuals. GREML analysis is a feature of the statistical program, which provides different types of Genome-Wide Complex Trait Analyses (Yang and others, 2011). GREML allows researchers to quantify the extent to which common genetic variants influence certain traits, such as the age at first birth and total number of children (Tropf and others, 2015b). Simply put, the GREML method calculates the genetic similarity between unrelated individuals based on their genetic material (i.e., their SNPs). This genetic similarity matrix is then related to the similarity in an outcome amongst individuals — which in our case is fertility. For example, if you share what we call your "segregating genetic material" (i.e., what makes *you* genetically *you*) at a level of 0.05% with one group and 2.5% with another, we would say that you have a higher similarity in your fertility behaviour with the second group. Parallel to twin

studies, we expect closer related pairs of individuals to be more similar in their phenotypes if the phenotype has a genetic basis. Since the genetic overlap between pairs of individuals from different families is very small, large numbers of people are required for this type of analysis.

The first study to examine fertility in the form of age at first birth and number of children ever born was published in 2015 (Tropf and others, 2015), with the main results shown in Figure 2. Using Dutch and UK-based data, the main finding was that for the first time we were able to quantify the extent to which common genetic variants (SNPs) influence fertility. This study found that the differences in women's age at first birth (AFB) and the number of children ever born (NEB) were associated with genetic differences. For the age at first birth, 15% of the observed variance was explained by genetic variation in common genes; for the number of children, it was 10% (see Figure 2).In demography and sociology, it is well established that the AFB and NEB are strongly correlated. In other words, if you have your first child later, you will have fewer children (Sobotka, 2004; Tropf and others, 2015b). The aforementioned study (Tropf and others, 2015b) also shows that the genetic effects for both outcomes overlap, which is partly explained by the association between AFB and NEB. In other words, it appears that the genes related to the time that women have their first child appear also to influence the number of children they ultimately have. The study thus partly explains why women who have children earlier also have a higher number of children.

This study, and similar ones that followed (Beauchamp, 2016; Kong, 2017), also contribute to the controversial debate about whether humans still evolve via natural selection. If particular genes are related to higher reproductive success (i.e. having more children), these genes will be passed on with a higher frequency to future generations. As discussed previously, NEB is seen as a proxy for "fitness", and additive genetic variance in NEB therefore indicates ongoing natural selection within modern populations under study. The study examined women from the UK and the Netherlands born in the twentieth century, showing that those who had a genetic predisposition for an earlier age at first birth have had a reproductive advantage across the generations. Genes associated with an earlier AFB have been passed on more frequently to the next generation, allowing the authors to conclude that natural selection acts not only in historical, but also contemporary populations (Tropf and others, 2015b).

Studies on contemporary evolution, however, raise some perplexing findings (Tropf and others, 2015; Beauchamp, 2016). If genes associated with an earlier AFB, for example, are more likely to be passed down to the next generation, why is it that younger generations in the contexts that were studied were not having their children at an even earlier age? What we see in fact, is that women are doing exactly the opposite in most industrialized nations. Since the 1970s, women are having their first child around 4–5 years later, which is now on average at age 28–29 years (Mills and others, 2011). This massive postponement in the age at first birth suggests that the socio-environmental influences considered as important by social scientists and discussed previously, such as women's educational expansion and entry into the labour market and the widespread use of effective contraception, has had a much stronger influence on fertility trends than natural selection. However, physiological changes should not be ignored and given the fact that both genes and the socio-environment can be shown to empirically matter for fertility, there is still a need for an integrative "sociogenomic" research design that draws from both genetics and the social sciences to better understand and predict human fertility.

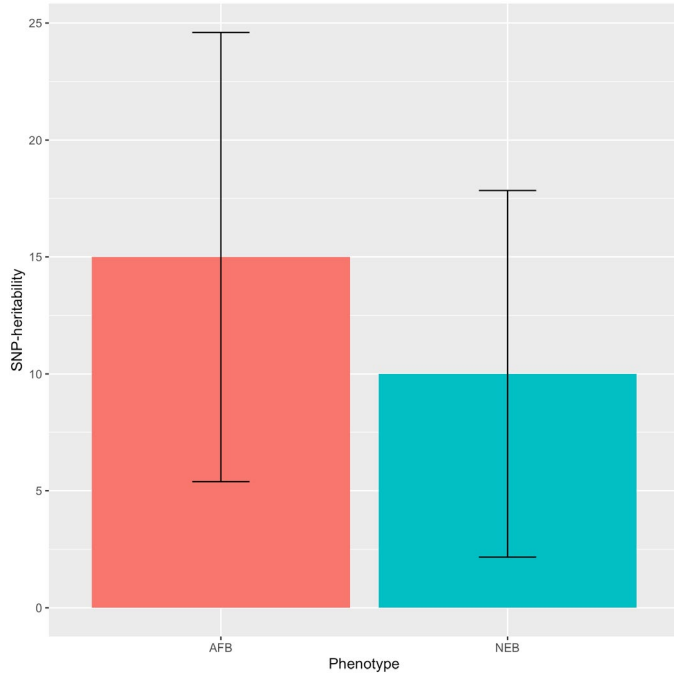

Figure 2 Estimates of the genetic variance explanation from common genes for the age at first birth (AFB) and the number of children ever born (NEB). (The genetic variance component is called heritability). Note: Adapted from Tropf et al. (2015)

GWAS (Genome-Wide Association Studies) and Polygenic Scores

GWAS

Since around 2006, Genome-Wide Association Studies (GWAS) emerged as a promising new approach to connect genetic variants to a phenotypical outcome of interest (Visscher and others, 2017). GWAS refers to hypothesis-free testing of genetic associations with outcomes of interest without any a priori assumptions about either the biological pathway or a particular location. It likewise embraces the fact that there are multiple genes (polygenic) and pathways associated with fertility that are difficult to specify in advance with our current state of knowledge. In GWA studies, we rapidly scan markers across the whole genome of many people (>100,000) to find genetic variations associated with a particular trait. GWAS are possible due to the completion of the Human Genome Project in 2003 and the International HapMap Project in 2005, which enable us to detect and measure genetic polymorphisms. As with other genetic data available until now, it is necessary to have the DNA from each participant in the study, often via a blood or saliva sample. Each person's DNA is then placed on tiny microarray chips and scanned on automated laboratory machines. These machines quickly overview each person's genome for strategically selected markers of genetic variation, referred to as SNPs (single nucleotide polymorphisms).

A GWAS therefore runs millions of separate regressions on the phenotype (outcome) of interest across the genome. Due to the large number of SNPs that are tested in GWASs, an association must

achieve a stringent threshold of statistical significance ($P < 5x10\text{-}8$) in order to be considered as validated. A positive association refers to the case where there is a greater frequency of a genetic variant in individuals with that trait compared to those in the control group (i.e. absence of trait). The association identifies a genomic region and not a specific causative mutation that might be involved in the development of the trait or behaviour.

The computational GWAS approach remains promising for social science research due to the fact that it overcomes some of the mistakes inherent in candidate-gene studies in the past. But also, since complex fertility traits often evade the specification of *a priori* biological pathways, it remains a useful exploratory technique. It is also the only technique currently available that has the potential to discover novel genotype-phenotype associations, which could then be used in further, more reliable follow-up studies and test for indications of where researchers need to search and pursue potential biological pathways. It also allows population stratification to be controlled — to some extent — which, however, remains a key issue in avoiding bias and misinterpretation of results in this type of research (Wray and others, 2013 and Mills and others, 2022).

Previous GWAS discoveries successfully detected SNPs that are associated with reproduction. Over seventy GWASs have been published for thirty-two traits and diseases associated with reproduction (Montgomery and Zondervan, 2014). This includes identification of genes such as those related to age at menarche (Sulem and others, 2009; He and others, 2009; Elks and others, 2010), menopause (He and others, 2009; Snieder and others, 1998; Perry and others, 2013), and endometriosis (Painter and others, 2011). The first GWAS on reproductive behaviour isolates 12 loci for age at first birth (AFB) and number of children ever born (NEB) (Nicola Barban and others, 2016). It engages in an analysis of sixty-two datasets with information from 238,064 men and women for age at first birth, and almost 330,000 men and women for the number of children. The study showed that DNA variants linked with the age at which people have their firstborn are also associated with other characteristics reflecting reproduction and sexual development, such as the age at menarche, voice-breaking in boys, and the age at which women experience menopause. Some of these genes were already known to influence infertility, while others had not yet been studied. The genes that were isolated also pointed to pathways and tissue types that were involved in human development, infertility, and sperm differentiation in men.

This was extended recently by two considerably larger studies on AFB (~540,000 individuals) and age at first sexual intercourse (~389,000) (Mills and others, 2021), as well as NEB (~717,000) and childlessness (~450,000) (Mathieson and others, Forthcomoing). Due to heavily increased sample sizes, these studies isolated almost one hundred and three hundred loci for AFB and AFS, and forty-three new loci for NEB and childlessness. A stunning finding was that — linking the contemporary findings to ancient genome data — that the FADS1/2 locus has been under natural selection for over 10,000 years and appears still to be so today.

Polygenic Scores (PGS)

Since reproduction is a complex behavioural outcome, it is not simply one candidate gene that can be used to predict outcomes. Rather, it is often a myriad of genetic loci compiled into a comprehensive polygenic score (PGS), which has been explored in detail in relation to the previous AFB and NEB GWAS (Nicola Barban and others, 2016) in another paper (Mills and others, 2018). We now summarize these results here. A polygenic score is a linear combination of the effects of

genetic variants present across the whole genome and can be interpreted as a single quantitative measure of genetic predisposition. Just as a battery of multiple questions on personality types or attitudes towards immigration can make up a scale that is measured by one index, a PGS assumes that individuals fall somewhere on a continuum of genetic predisposition resulting from small individual contributions from many genetic variants.

How does a PGS calculated from the previously mentioned GWAS of AFB and NEB (Barban and others, 2016) work? To examine PGSs of AFB and avoid false positives from examining the associations in a limited dataset, results were tested and replicated across four different datasets: HRS (United States), LifeLines (Netherlands), TwinsUK, and STR (Sweden). Using OLS models this study carefully examined results from the large scale GWAS on reproductive behaviour by Barban et al. (2016) and found that the PGS for AFB explains around 1% of the variance (for women) in AFB and around 0.2% for NEB. While these numbers seem small, in some cases when the variants are combined, they can explain 9% of the probability of women remaining childless or six months of the delay in AFB per standard deviation (SD). Using a Cox model that accounts for right-censoring in the AFB, 1 SD in the AFB PGS is associated with a reduction of around 8% in the hazard ratio of reproduction for women and 3% for men. The PGS of NEB is associated with a 1 SD increase in the PGS, decreasing the probability of remaining childless by 9% in women. Importantly, with the increased sample sizes in the more recent discovery studies (Mills and others, 2021), GWAS-derived PGS explain already up to 6% of the variance in reproductive traits, which is expected to further increase with ever-growing sample sizes.

The genetic tendency to have a later AFB is also linked to an overall shifting of the reproductive period, linked with both a later onset of menarche and menopause (Mostafavi and others, 2017). As with other studies that have used PGSs from GWAS discoveries on complex behavioural traits such as educational level, a certain amount of reflection is in order. The most recent meta-GWAS, which finds seventy-four significant hits for educational, explains around 3.2% of the observed variance (Okbay and others, 2016). We therefore turn to additional reasons for this variation now.

How Do Genetic Effects Vary Across Populations or the Sexes?

Birth Cohort and Country Variation

GWA studies often combine genetic data from individuals from different countries and historical time periods in order to gain a large enough sample size. By doing this they assume that the influence of genes on individuals is universal across time and place. As the review in this chapter until now has illustrated, previous twin studies estimated AFB and NEB to be around 30% heritable with GREML estimates suggesting that genetic differences should be able to explain around 10–15% of the differences in fertility between individuals in a population. However, large GWA studies, which aimed to uncover the specific genes that are related to fertility and other complex traits, have produced much lower estimates.

A recent study (Tropf and others, 2017) demonstrated that this may be attributed to the fact that GWAS methods rely highly on data from different countries and historical periods, which potentially "hides" heritability because combining these data sets could mask large differences. In other words, if the genes that are important for fertility differ across countries, birth cohort or

historical periods, it may be difficult to detect genetic variants when combining data from diverse populations. Using data from six countries (Australia, Estonia, Netherlands, Sweden, the UK and the US; overall 35,062 men and women) and several historical periods, the study found that 40% of genetic effects on education and timing of fertility (i.e. age when someone has her or his first child) are being "hidden" or "watered down" when data across populations in different countries and time periods are combined. For the number of children, this value increases up to 75%. In contrast, physical traits such as height are not impacted. The genes connected with height thus seem to be the same across populations.

Next to rare variants and insufficient sample size, GWAS discoveries might therefore be limited by heterogeneity across cohorts and birth cohorts under study (Tropf and others, 2017). Heterogeneity can arise on the phenotypic level if the phenotypic measurement differs across cohorts and birth cohorts, on the genotypic level if linkage disequilibrium differs across populations under study and by gene-environment interaction. The predictive power of the whole-genome methods increases up to fivefold when taking heterogeneity across cohorts and birth cohorts into account (Tropf and others, 2017). Given that fertility is largely environmentally determined and modified, it is likely that gene-environment interactions are important across the many cohorts included in GWAS discoveries as well as across birth cohorts. This in turn may be one reason for the comparably small predictive power of the polygenic scores. Combining data sets from vastly different countries and historical periods could be muddying the waters.

Sex Differences

Another aspect that deserves further attention in future research is sex differences in fertility and reproductive outcomes. This seems obvious since there are sex differences in biological makeup, in processes and diseases implicated in infertility and in behaviour. For women, ovulatory problems, tubal damage, endometriosis, cervix cancer and polycystic ovary syndrome are prominent causes of infertility, with sperm defects and testis cancer being central factors for men (Blundell, 2007). As we have seen from previous sections, these diseases are partly heritable. But there is also a behavioural component, since genes are implicated in different ways in relation to fertility and certain personality traits, including sociability, impulsivity and emotionality (Briley and others, 2017). These traits, which may have different effects on male and female fertility, have been shown to be heritable (Robinson and others, 2008).

Almost identical results in heritability estimates for men and women (Rodgers and others, 2001) might suggest that the same genes are important for male and female fertility. However, a study by Nisén et al. (Nisén and others, 2013), for example, shows that genes predicting childlessness in women are associated with low education among women and high education among men. Therefore, the genetic architecture of fertility might differ considerably between the sexes. Verweij and colleagues (Verweij and others, 2017) tested whether genetic loci operated differently in male and female fertility in the form of sexual dimorphism. Sexual dimorphism, or in other words, differences in secondary sex characteristics, can result in intralocus sexual conflicts, when genes that increase male fertility decrease female fertility, and vice versa. Using Swedish data, Verweij et al. (Verweij and others, 2017) estimated twin, GREML and AFB-PGS models on childlessness. They found that variation in individual differences in childlessness was explained by around 47% in the twin model and 59% (women) / 56% (men) in a GREML sibling model while the genetic correlation across sexes was significantly lower than 1. Using the PGS

of AFB they also found significantly higher odds of remaining childless — however, only for women. The study concluded that partly different sets of genes influenced childlessness in men and women.

Discussion and Conclusion

The aim of this chapter was to provide an up-to-date review and comprehensive overview of research on reproductive behaviour in the area of fertility and genetics. We first emphasized that when working in an interdisciplinary area such as fertility or reproduction, it is important to be cognizant of the varying terminology used across the disciplines. Genetic research on fertility behaviour also revived interest in understanding contemporary natural selection and evolution, since reproductive success codetermines the successful transmission of genes of an individual to the next generation. The link with fertility and genetics also has a dark history in early misguided work in eugenics, which we firmly condemn and distance from the more serious scientific research reviewed here.

Genetic Approaches to Fertility

Our review examined first behavioural genetic (twin model) approaches to fertility, followed by the more recent growth in molecular genetic approaches. We summarized how twin studies have demonstrated that the age at first birth (AFB) is around 0–35% heritable compared to around a 24–43% heritability of number of children ever born (NEB).

Molecular genetic studies, which use data from the whole human genome, first started with candidate gene studies, which proved difficult to replicate. This was then followed by GREML (Genomic-Relationship-Matrix based Restricted Maximum Likelihood) techniques that allowed researchers to produce more direct estimates of the proportion of phenotypic variance explained by genetic variance across the entire genome for unrelated individuals. Studies predicated that AFB and NEB were 10% and 15% explained by genetic variance in common genes, respectively.

Describing the percentage age of variation in genetic differences, however, does not uncover the actual genes or their biological functions. For this reason, researchers have turned to GWA studies. This is hypothesis-free testing across the genome to find associations with a particular fertility trait in order to isolate key genetic loci. We summarized results of previous studies, isolating hundreds of genetic loci for AFB, age at first sexual intercourse, NEB, and childlessness that were linked with human development, infertility and sperm differentiation. We then explained how these discoveries of genetic loci from GWAS can be compiled into a single polygenic score (PGS), which predicted up to 6% of the fertility outcomes. We also learned that the PGS for AFB and NEB are relatively strong predictors of the probability to remain childless (e.g. by 9% in women). Highlighting the evidence of ongoing natural selection in humans, we wish to emphasize that the relationship between fertility tempo, quantum and fitness and the derived evolutionary consequences are often still simplified. Future research should aim to integrate in-depth evolutionary demographic knowledge (Mcgraw and Caswell, 1996; Jones and Bird, 2014) with ongoing advances in molecular and quantitative genetics research (Barban and others, 2016; Tropf and others, 2015b; Guo and others, 2018).

Towards Gene x Environment Interaction

We acknowledged that reproductive behaviour is not only genetic but largely shaped by individual-level factors, but also family (meso-) and societal-level (macro-) forces, which we reviewed. We noted that until now, the majority of demographic and sociological research on this topic has focussed almost primarily on these aspects. We likewise acknowledged that they explain the majority of these complex outcomes and will continue to do so. Promising new approaches, however, should focus on how reproductive PGSs interact with these socio-environmental characteristics. It may be, for instance, that fertility issues arising from genetic predispositions for reproductive health problems, such as endometriosis or sperm defects, are more relevant in populations with higher average age at childbirth than in populations with younger ages at childbirth. Or, someone might be more genetically "hard-wired" to have more children, but if they come of age in an economic recession or work in precarious jobs, this might be more important than these predispositions. Likewise, it remains important to understand the physiological and psychological mechanisms, how genes influence fertility behaviour and outcomes. We anticipate that it is not the socio-environmental or the genetic predictors that will uncover fundamentally new findings, but rather a combined sociogenomic approach.

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SECTION 5:
THE MEASUREMENT AND INTERPRETATION OF SELECTION AND FITNESS

If there are quantities that, by their very estimation, could often distinguish evolutionary demographers as a group, they would be selection and fitness. An investigation into how evolutionary processes shape demographic patterns often requires that we calculate the strength of selection. Selection is "strong" on a heritable trait when it is highly correlated with fitness, and "weak" when there is little or no correlation with fitness (typically estimated by the coefficient in a regression). If heritable traits are associated with strong selection pressures, then their frequency in the population is likely to change relatively quickly. Examining variation in the heritability of traits and the strength with which selection acts upon them can help us understand how the changing ecological and social environment causes the population to change.

One set of traits that has been extensively studied in relation to fitness is the age pattern of fertility and mortality. In a famous study, Hamilton (1966) used stable population models to analyse the effect of variations in fertility and mortality at each age on the growth rate of the population, concluding that those variations with the greatest effect on the growth rate would be subject to the strongest natural selection (see chapter by Wachter).

Natural selection can act not only on biological traits, but also on cultural behaviours that either increase or reduce reproductive fitness. The cultural practices of a group can also influence the way that biological traits affect fitness, and thereby alter the way natural selection acts on those traits. A famous example is that pastoralism and consumption of milk products increases the fitness impact of continuing to produce the enzyme that enables digestion of milk at ages past weaning (although recent research suggests new complications).

Since all questions of evolution involve demographic processes, demographic tools are essential for advancing the research of evolutionary biology and ecology, and these chapters show how attention to demographic principles has greatly advanced our understanding of selection and fitness, and hence why mortality and ageing patterns look the way they do; this is where formal demography and evolutionary biology overlap.

Moorad leverages his expertise in formal modelling and evolutionary biology to provide us with a succinct overview of quantitative genetics that also serves as a recipe for how to handle computational challenges in the estimation of selection and fitness that are typical in human populations, like overlapping generations and social interactions. As models of quantitative genetics have iteratively improved, even species with complex life cycles, like us humans, can have selection pressures accurately estimated. This is also useful for modelling how patterns should change in future generations. A needed future application of Moorad's framework is to

extend the approach of using estimates of how phenotypic selection affects vital rates and age distributions to help inform projections of human population size.

Van Daalen and Caswell (in a collaboration of a member of the new generation of evolutionary demographers with one of the field's founders) develop definitional and mathematical tools for many applications in biology and demography. Here, they further our understanding of the concept of fitness and how it is calculated with demographic data. Additionally, they resolve how to include an under-appreciated aspect of variation in fitness: namely, the variation that arises from random or stochastic differences among individuals. If that sounds strange, consider this: lightbulbs constructed from identical materials and used in identical circumstances will have variable lifespans. This means that their demographic outcomes are variable due to stochastic factors that are independent of each individual's observable traits. Humans have much more variation in their makeup and genetic material than do lightbulbs, yet researchers often behave as if all of the variation in demographic patterns could be fully explained if they measured the right variables. However, this is simply not the case because a lot of variation is inherently stochastic: individuals will differ by chance, independently of education or health or the like. Van Daalen and Caswell offer an elegant presentation of and solution to this problem as it pertains to fitness in humans.

Carey has been mixing demography with evolutionary biology longer than just about anyone. His collaborations with Jim Vaupel on old-age mortality and his studies of ageing in the wild are among the most well-known and influential studies in the field. In this engaging chapter, he combines a personal discovery essay about a demographic identity (in the mathematical sense) with the biological context that surrounds it. Demographers and biologists alike will find much to appreciate in Carey's discussion of how challenging the concept of "age" is for the study of biological populations in the wild, as it is a factor that is quite easy to take for granted when studying human demographic rates via downloadable datasets.

The fourth chapter in this section is an overview of the human mortality profile by Orzack and Levitis. As a historical note, it was Levitis who passionately pitched the idea of an Evolutionary Demography Society at a small meeting in Rostock at the Max Planck Institute for Demographic Research, which now has thriving annual meetings. Using their experience in biology and demography, they argue that two concepts inherited from evolutionary ecology, but that are also common in economics, are over-used in evolutionary demography. These are natural selection and optimality. In short, Orzack and Levitis show the importance for HED of considering other kinds of evolutionary processes besides natural selection (in line with the Tinbergen section of this book), a case they make in a thorough discussion of the "U-shaped" mortality profiles observed in humans and most other species.

14. Measuring Selection for Quantitative Traits in Human Populations

Jacob A Moorad

Quantitative genetics offers a powerful suite of statistical approaches designed to describe and predict rates of phenotypic evolution. Its origin lies at the reconciliation of Mendelian and biometrical genetics and Darwin's theory of evolution by natural selection that occurred in the early twentieth century. Quantitative genetics has since played a major role in the science of animal and plant improvement since the mid-twentieth century and in the study of evolution since the 1970s and 80s. The goal of this chapter is to introduce this perspective to demographers, provide guidance on methods intended to characterize natural selection on traits of interest, and to illustrate the flexibility of this approach to deal with complications that are inherent to the study of human populations, such as overlapping generations and social interactions.

An important goal of evolutionary biology is to quantify the rate and direction of phenotypic change occurring in populations and to identify the portion of that change that is caused by natural selection. Understanding this response to selection requires sound measurements of the two elements of phenotypic evolution: (1) phenotypic selection: the association between fitness and the traits of interest, and (2) trait transmission or inheritance: the association between the traits of parents and their offspring. The approaches and data requirements for estimating these components differ, but a complete understanding of the response to selection usually requires estimates of both. Combining these components can describe retrospectively how natural selection caused transmissible genetic change that altered trait means from one generation to the next, and it can provide predictions about how natural selection will contribute to changes in the future. A strength of this approach to understanding evolutionary change is that when the appropriate analytical tools are implemented correctly to estimate one element of the response to selection (e.g. phenotypic selection), it is not necessary to estimate jointly the other (e.g. inheritance). This is important for comparative work because the response to selection can be thought of as a product of both components, and as a result, both can serve as independent indicators of maximal rates of phenotypic evolution. For example, evolution by natural selection can never occur faster than phenotypic selection.

In practice, the requirements for quantifying inheritance can be much more demanding than for measuring selection in terms of data quantity and technical know-how. As a result, phenotypic selection is the most often studied of the two components of phenotypic evolution, and the past few decades have brought about a proliferation of plant and animal selection studies performed in natural and artificial environments on a great diversity of traits. Not surprisingly, humans have also been subjects of phenotypic selection analyses, but a greater

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appreciation of the complexities related to these populations demands that special care be taken in the application of analytical methods. The first of these complications involves age structure and overlapping generations. These features characterize many non-human populations, of course, but human data is generally available on time scales far finer than generation time, and age structure is much more difficult to ignore. The second feature involves social interactions. Again, these are certainly not specific to humans, but there are no other species of which we know more about the importance of sociality. Demographers are intimately aware that these are important characteristics of human populations, and including an honest and informed accounting of these features into estimates of natural selection should be a goal of evolutionary studies of humans. This chapter is written with this goal in mind.

The methods discussed here are general to all systems; these have been discussed elsewhere in the primary literature, but it may be useful to collect them in a single overview intended for demographers who are specifically interested in measuring selection on phenotypes in human populations. The perspective taken here follows one of quantitative genetics, an area of study first developed for the purposes of animal improvement (Lush, 1937), but since developed and applied to the study of evolution by natural selection (Lande and Arnold, 1983, Arnold and Wade, 1984a,b). Much of my own work over the past few years has focused upon refining these methods to be useful for understanding evolution in age-structured populations (Moorad et al., 2011; Moorad, 2013a,b; Moorad and Wade, 2013; Moorad, 2014; Moorad and Walling, 2017; Moorad and Ravindran 2022), and here I have applied nearly all the methods discussed in this chapter to study selection in a human population.

I begin this discussion by contextualizing how phenotypic selection fits into the evolution of phenotypes; this is done to make clear the importance of avoiding conflating selection with inheritance (a problem inherent in many studies of selection). As I explain in the next section, selection is defined as a covariance between fitness and the trait(s) of interest (Robertson, 1966; Price, 1970); phenotypic selection does not imply a response to selection. As such, implementing an appropriate definition of fitness is key to appropriate estimates; I demonstrate in the second section (Relative Fitness) how individual reproductive value at birth is our most appropriate definition to be used for this purpose. However, there are many different ways to express phenotypic selection (even using the same measure of fitness). In the third section (Measures of Phenotypic Selection), I discuss what these measures mean, how they are estimated, and how they should be interpreted. Fourthly, I digress into a discussion of social interactions and how the quantitative genetic approach accounts for these (Complications Owing to Social Interactions). Fifthly, I discuss the complication of what to do if some individuals are logically precluded from expressing a trait of interest (age at menarche in males, for example) (Impossible Traits). Finally, I will introduce measuring genotypic selection as an alternative to phenotypic selection for predicting evolutionary responses to selection, and I will discuss the advantages and disadvantages of the two approaches (Genetic Selection for Quantitative Traits).

Phenotypic Selection and Evolutionary Change

As a first step to understanding phenotypic selection, it may be useful to articulate carefully where this fits into how we understand evolution by natural selection. The Price Theorem (Price, 1970, 1972) is often invoked as a fully general expression that formally accomplishes this goal by describing a between-generation change in the mean of some arbitrary trait z.

What is a trait? It can be literally anything that can be attributed in some way to an individual. For the purposes of this discussion, however, I will assume that the trait is observable. The specific shapes of trait distributions do not matter in principle; they can be continuous or discrete, Gaussian or not. Vital rates (age-specific survival and fertility) are traits of obvious importance to many evolutionary demographers. Other human traits of interest might be age at menarche or menopause, total lifetime reproduction, number of years lived past some age of interest, and survival to some age (a dichotomous trait).

Here we imagine an "ancestral" population in which every individual has some trait value z (we begin here with the univariate case where the evolution of one trait is considered without regard to any other, but we will generalize to the multivariate case later). Members of this ancestral population collectively produce offspring that wholly constitute a "descendent" population. The contribution of each ancestor to this new population, relative to the entirety of the ancestral population, is the ancestor's relative fitness, w. By the nature of this definition, w is non-negative with a mean of one. Finally, let us specify that for our purposes here, individuals are organisms, and the "descendents" are the offspring of the "ancestors". Following Price's Theorem, we can express the between-generation change in the mean of the trait as,

$$\Delta\bar{z} = \beta_{wz}\mathrm{cov}(z_d, z) + \delta \qquad [1],$$

where β_{wz} is the coefficient associated with the least-squares regression of relative fitness on trait values, z_d is the value of offspring phenotypes, and δ is the average change in trait values between offspring and their parents *unweighted* by relative fitness.

We can equate the change in the trait mean, $\Delta\bar{z}$, with an evolutionary change, and by doing so, we can identify the role that natural selection plays in this change, but first it is helpful to consider each of the three terms given in the right-hand side of eq. [1] in turn. The meanings of these are as follows.

1. The coefficient β_{wz} is known as the *selection gradient*; this is a slope that tells us how sensitive relative fitness is to changes in z. If we were to multiply this slope by the phenotypic variance of the ancestral population, we would have a *selection coefficient*, $s_z = \beta_{wz}\mathrm{var}(z)$. This is the covariance between relative fitness and ancestral trait values. This is equal to the difference between two trait means in the ancestral population: the first mean is weighted by relative fitness and the second is not.

2. The covariance between parent and offspring traits, $\mathrm{cov}(z_d, z)$, represents *transmission fidelity*. This gives us the amount of heritable variation, in absolute terms, associated with this trait in this population. This is often interpreted as *additive genetic variance*, or $\mathrm{var}(G)$. This concept may be expressed differently by restating it as the fraction of phenotypic variance in the ancestral population that is heritable. The covariance given in terms of *narrow-sense heritability* is $\mathrm{cov}(z_d, z) = \mathrm{var}(G) = h^2_z$. Note that h^2_z is also $\beta_{z_d z}$, the slope of the regression of offspring traits upon their parents' trait values, so that one can equivalently write $\mathrm{cov}(z_d, z) = \beta_{z_d z}\mathrm{var}(z)$ following the standard definition of regression coefficients.

3. The last term, δ, is the *transmission bias*. This accounts for all changes in the mean phenotype from one generation to the next that have nothing to do with natural

selection acting to change z. In practice, this is often attributed to changes in the environment or in the genome owing to the influx of new mutations.

From (1), we see that the response to selection, or the portion of the total change attributable to natural selection, is equal to both $\beta_{wz}\text{var}(G)$ and $h_z^2 s_z$. This is known as the "Breeder's Equation" (Lush, 1937). From either formulation, it is clear that both phenotypic selection and inheritance are required for a response to selection, and both independently act as mathematical limits to the potential for evolutionary rates of change owing to the force of selection.

Incidentally, some demographers are accustomed to scaling evolutionary change in terms of the length of time intervals (e.g. years in human studies), and it may seem strange to them to think of evolution expressed on the scale of generations. Because both of those conventional demographic approaches (e.g. Caswell, 2001) and the perspective advocated here assume that vital rates are stable over time, the difference in scaling is a trivial issue. Estimates of selection (or responses to selection) on the generational scale are converted to estimates on the time interval scale by dividing by mean generation time, where $T = \sum_{x=1}^{\infty} x l_x m_x e^{-rx}$ (Lande, 1982; Charlesworth, 1994).

Relative Fitness

As phenotypic selection is a covariance that always involves relative fitness, it is critical to quantify this value carefully. At its essence, relative fitness is simply a weighting factor applied to an individual to express its relative contribution to the next generation. In practice, determining what these weights are does not appear to be straightforward. Indeed, there has been much confusion on this point. Before we go further into a discussion of what relative fitness *is*, it may be illustrating to consider what it *is not*, at least in the context of the quantitative genetics perspective of phenotypic evolution considered here:

Fitness is not a characteristic of groups of individuals, where "group" is defined as belonging to the same population or sharing a common trait value. Groups of individuals can have fitness means, but these do not normally factor directly into expressions describing the response to selection. Fitness is best thought of as an attribute of an individual.

Fitness is not the contribution of some individual to the population at some arbitrary point in the future. As stated before, fitness describes the weighting of ancestral contributions to a descendent population. As such, the concept of fitness depends entirely upon the definitions of these populations. It should be appreciated from the previous section that these definitions are preserved when characterizing both selection and inheritance, and inheritance is always expressed on the scale of single generations (e.g. narrow-sense heritability follows from a parent-offspring regression). One could, in principle, define fitness based upon the number of grandchildren or great-grandchildren, but in these cases, the notion of heritability changes fundamentally to mean something quite different and potentially bizarre, such as the resemblance between great-grandparents and great-grand-offspring. This can potentially conflate the causal processes that we normally understand to be selection and inheritance, and estimates of the response to selection can be rendered invalid (Hadfield and Thomson, 2017).

Fitness is not the number of recruited or adult offspring because pre-adult death is an aspect of the performance of the offspring and not of the parent. Ideally, fitness relates to the number of zygotes produced by zygotes, but, as accounting for the reproductive success all new embryos

in a human population is impractical, using the number of newborns eventually produced by each newborn is a reasonable approximation. In practice, the clearest inferences follow from using the earliest age at which individuals can be observed.

Inclusive fitness is not a concept that is compatible with the modern quantitative genetic perspective taken here. Fitness is measured directly, and its definition need have no relationship to the performance of kin.

Some may object to elements of this list by invoking issues involving social interactions, such as resource transfers and potentially important effects of maternal or grandmaternal care. From a certain perspective, it may seem that these issues must require that the notion of fitness is quite complicated, or even arbitrary. However, if one is willing to accept some assumptions regarding demographic stability, fitness is actually a rather simple concept to understand and one that may be relatively easy to implement. As I discuss in a later section, the manner by which social interactions shape the evolution of phenotypes is accounted for in quantitative genetics not by redefining fitness, but by recognizing how these interactions redefine selection and inheritance in other ways.

In most evolutionary genetic studies, fitness is regarded as the total number of offspring born, R_0, total lifetime reproduction, or total breeding success (the terminology usually depends upon the field that implements it). Relative fitness is then simply this value divided by the mean value for the ancestral population. This is a perfectly adequate approach for populations in which generations do not overlap, but this is certainly not the case for human populations. In these cases, population growth must be considered as part of a satisfactory definition of fitness. While a few different definitions have been suggested, the most satisfactory is the individual's reproductive value at birth. The case for using this measure of fitness is made more explicitly in Moorad (2014), and here I will only discuss how the measure is defined and some of its implications and limitations.

Let us assume that the population has a constant Malthusian growth rate r, and every individual has some known number of new offspring that may vary with its age. For any individual i, its individual reproductive value at birth is,

$$w_i = \sum_{x=1}^{\infty} e^{-rx} B_{ix} \qquad [2],$$

where B_{ix} is the number of offspring alive at the first age class that are produced by individual, i, at age x. Demographers will be familiar with the notion of a reproductive value at birth from Fisher (1930), but it should be emphasised that Fisher defined it as the *average* of eq. [2] taken over all individuals at birth. For systems in which all offspring must have exactly two parents, then this measure must be discounted by half. Eq. [2] has a couple of obvious features that are worth pointing out:

1. The average of B_{ix} taken over all individuals i is equal to the product of: (1) the cumulative rate of survival to x and (2) the mean fecundity rate at x conditional upon survival to that age. Substituting this average into the right-hand side of eq. [2] recovers the Euler-Lotka relationship $1 = \sum_{x=1}^{\infty} l_x F_x e^{-rx}$. It follows that the mean of individual reproductive values at birth is one.

2. If the population size is stable, then $r = 0$, and relative fitness is equal to lifetime reproductive success. In these cases, nothing is lost if generational overlap is neglected, and $w = R_0/\bar{R}_0$

While the structure of eq. [2] explicitly demonstrates how population growth rates affect the determination of fitness, very little is known about the consequences of ignoring this feature in real populations (as is often the practice). Population growth modifies how realized fertility contributes to fitness, but this influence is amplified in late life when compared to its effect early in life. From this, a reasonable inference could be that estimates of selection for late-acting traits may be particularly sensitive to incorrect estimates or implementation of population growth rates (including using R_0 for fitness when $r \neq 0$). To my knowledge, only two studies have actually measured the association between w and R_0, and these two estimates of the correlations were not independent as they were applied to the same human population over overlapping time ranges. Moorad (2013a) reported correlations between 0.978 and 0.992 (depending upon birth year) for the female population of Utahns born between 1830 and 1894. For both sexes combined, and for the birth years 1860–1889 in the same population, Moorad and Walling (2017) reported a combined correlation of 0.986. Malthusian growth rates for this population were high (between 0.025 and 0.039), and this fact, coupled with extremely high correlations between w and R_0, might suggest that the consequences for ignoring age structure (and thereby assuming that $r = 0$) might be minimal in the general case. As noted before, however, estimates of selection for traits acting at late ages are expected to be the most sensitive to errors arising from neglecting growth rates. Because late-acting traits are expected to contribute little to the variance in fitness (see next section), very high correlations between w and R_0 may persist even when estimates of late-acting selection are heavily biased by errors in population growth rate. A reasonable recommendation would be to use individual reproductive value at birth instead of R_0 whenever possible. Provided that population growth rates can be determined from the data, then there seems no reason to prefer R_0. For cases where data are not adequate for estimating population growth rates, then one should question whether these data are sufficient to make evolutionary inferences.

By using individual reproductive value rather than R_0 as a definition of fitness, one effectively relaxes the, often implicit, assumption that population sizes remain constant over time. This assumption is replaced with the less restrictive requirement that population growth rates are temporally stable (r doesn't change over time). However, this assumption is also likely to be violated in most populations. Unfortunately, there is no clear definition of individual fitness that relaxes this assumption further. When growth rates are free to vary over time, the answer to the question, "What do you mean by fitness?" is, as always, dependent entirely upon the answer to the question, "Well, what do you mean by ancestral and descendent populations?" The answer to the latter question may be arbitrary to some degree (or at least sensitive to one's temporal perspectives), and more conceptual work is needed in this area to better understand this issue. One pragmatic approach to this problem that has been adopted in the past (e.g. Moorad, 2013a) has been to evaluate fitness using the population growth rate determined by individuals that share a common birth time. While this is not an ideal solution, this method does account for some complications arising from age-structure, and it represents an improvement over the alternative R_0 in this respect.

Measures of Phenotypic Selection

Once equipped with a well-founded definition of relative fitness, we can begin to ask how selection acts on phenotypes. We have already defined selection gradients and coefficients in the univariate case and demonstrated where these measures fit into a simple expression of evolutionary dynamics. However, we are often interested in understanding selection at a deeper level. For example, we may want to know something more about the causal relationships between fitness and phenotypes, or we may want to know how two or more traits evolve when they share some genes in common. In these cases, we need to consider multivariate phenotypic selection, and we must refine our definitions accordingly.

Let us now imagine that we have a suite of traits, represented algebraically by a vertical vector $\mathbf{z}$ of degree n. We can regress simultaneously relative fitness on these traits, and this will yield a vector of partial regression coefficients $\mathbf{b}$. Collectively, these comprise the *multivariate directional selection gradient*. Each element b_i expresses the sensitivity of relative fitness to changes in trait z_i holding all other traits $j \neq i$ constant. We can contextualize this gradient into expressions that define and predict multivariate phenotypic evolution by also imagining a $n \times n$ matrix $\mathbf{G}$ that contains the additive genetic variance along the diagonal elements and the genetic covariances in the off-diagonal elements (the genetic covariance is the product of the genetic correlation and the square-root of the product of the two additive genetic variances for the appropriate trait combination). This genetic covariance matrix, or "G-matrix", contains all of the genetic constraints that enable and shape how natural selection ($\mathbf{b}$) affects evolutionary changes over a single generation. This relationship between evolution, selection, and genetic constraint is made explicit in what is known as the *Multivariate Breeder's Equation* (Lande, 1979), which quantifies the multivariate response to selection. If we consider transmission bias specific to all traits $\mathbf{z}$, we can incorporate this response into a generalized version of the Price Theorem given in eq. [1],

$$\Delta\bar{\mathbf{z}} = \mathbf{Gb} + \boldsymbol{\delta} \qquad [3],$$

where $\Delta\bar{\mathbf{z}}$ is the change in trait means for all n traits; $\boldsymbol{\delta}$ is the difference between the phenotypes of the offspring and their parents (averaged over all offspring and unweighted by fitness) for all traits; and $\mathbf{Gb}$ is the multivariate response to selection.

In the univariate case described in the first section, the selection coefficients differed from the selection gradients only in the sense that they were weighted by phenotypic variances. This is not the case in the multivariate case, as selection gradients follow from partial regression coefficients while the simple covariance definition of selection coefficients remain unchanged. The relationship between the two can be succinctly expressed by imagining an $n \times n$ matrix $\mathbf{P}$ containing phenotypic variances on the diagonal elements and phenotypic covariances on the off-diagonal elements,

$$\mathbf{b} = \mathbf{P}^{-1}\mathbf{s} \qquad [4],$$

where $\mathbf{s}$ is a vector of selection coefficients (Lande and Arnold, 1983). It may be noticed from these definitions that any selection coefficient chosen from within $\mathbf{s}$ will be entirely unaffected by the decision of whether or not to include some other trait in the selection analysis (remember that each of these is a simple covariance). However, because some traits may be phenotypically

correlated with others, selection gradients do not share this context-free nature. The value of each selection gradient b_i is understood to be conditional upon the set of other traits included in the analysis. This implies that unless a suite of traits can be assessed that explain all of the fitness variance in the population, the estimates of selection gradients may be flawed reflections of the true selective forces acting on the population. This is because potentially important traits may be missed that correlate with both fitness and the traits considered in the analysis. In practice, this suggests that an emphasis should always be placed on collecting and analysing the greatest number of informative traits possible, as causal inferences made from these relationships between fitness and traits are expected to become more reliable as the proportions of fitness variance explained increase.

In fact, there are two situations in which all fitness variance is explained by a set of phenotypes, and selection gradients can be interpreted as perfectly reliable indicators of the causal relationships between traits and fitness. The first case is when the trait of interest is relative fitness. This is trivial (the selection gradient for relative fitness is always equal to exactly one) and warrants no further discussion. The second case is when $\mathbf{z}$ is comprised of all vital rates up to the last age of realized fertility. In this case, and for each vital rate, we are asking how individual fitness changes with a change in this vital rate (and with all other vital rates held constant). Eq. [2] defines fitness as a linear function of these traits, and this means that vital rates collectively describe all fitness. One can go through the exercise of actually performing the multiple regression of relative fitness, defined as in [2], upon all vital rates simultaneously. This has been done using human data (Moorad, 2013a) and in an analytical proof (Moorad, 2014), and in both cases, the estimated selection gradients agreed with vital rate "sensitivities" derived by Hamilton (1966) using a completely different method and interpreted elsewhere as selection gradients (Charlesworth, 1994). This equivalency must hold true if eq. [2] provides a valid definition of relative fitness and Hamilton's selection model for the evolution of ageing is sound.

Selection gradients and selection coefficients describe in slightly different ways the strength of associations between fitness and traits, and, as such, play an obvious role in the evolutionary dynamics of trait evolution. Accordingly, selection is most frequently quantified in these terms. Perhaps the most profound measure of selection, however, is the variance in relative fitness, because it provides a population-specific upper limit to the amount of adaptive change that population can experience as a result of selection for *all* traits. In practice, however, it is often interpreted as an upper limit to selection for *any* one trait in the population. In any case, the variance, often called the *opportunity for selection*, has emerged as a popular comparative metric in human studies to evaluate the potential for evolutionary change. It has long been appreciated that this total opportunity for selection, or simply I, can be partitioned into one component arising from fitness variation from pre-reproductive survival and another arising from fitness variation among adults (Crow, 1958). These components are identified as I(survival) and I(fertility); in reality, these are misnomers, as variation in adult survival contributes entirely to I(fertility). For this reason, Crow's method for partitioning I is crude and misleading, but it is still quite commonly implemented. A far better alternative leverages multivariate selection theory in order to provide finer scaled and more readily interpretable results.

Recall our suite of traits **z**. Given an appropriate vector of selection coefficients **s** and a phenotypic variance-covariance matrix **P**, it must be the case that the opportunity for selection generated independently by each trait is given by the vertical vector **i**, where

$$\mathbf{i} = \mathbf{s}\mathbf{P}^{-1}\mathbf{s} \qquad [4]$$

(Moorad and Wade, 2013). The sum of all elements within **i**, divided by I, is the multiple coefficient of determination, or $\mathbf{R}^2$, of the regression of relative fitness on traits **z**. To this point, the expression is perfectly general to all possible **z**. Studies of ageing can use this approach to improve on Crow's method by asking how much variation in fitness is generated by each vital rate independently of all others (e.g. Moorad, 2013a). Because all fitness variation is explained by all vital rates up to the last age of reproduction, the sum of **i**-elements is equal to I (and $\mathbf{R}^2 = 1$). The value of this approach is that it helps identify which traits at which ages have the greatest potential to drive adaptive change in the population. Incidentally, one can use the definition of selection gradients to rewrite [4] as $\mathbf{i} = \mathbf{sb}$. Putting this expression together with a sensible interpretation of Hamilton's finding that the strength of selection for an age-specific trait tends to decline as the age of its expression increases (1966), it appears that, all else being equal, late-acting traits (low b) will tend to contribute less towards the variance in relative fitness than early acting traits (high b). This provides some justification for the warning given in the section on relative fitness that the high correlations between w and R_0 should not be taken to mean that the two measures are interchangeable when considering phenotypic selection for late-acting traits.

Non-directional Selection

In this discussion of multivariate selection, I have qualified the selection gradient as *directional*. This means that the function that relates fitness to phenotypes is assumed to be linear. Differently put, the fitness benefit (or cost) associated with phenotypic deviation from its mean is in proportion to the magnitude of the deviation. Depending upon the questions being asked or the traits being investigated, this constraint placed upon the fitness function may be undesirable. For example, fitness may be a quadratic function of some phenotype, or phenotypic value for one trait may interact with values for another trait to cause fitness effects that are not captured properly by a first-order linear regression. In these cases, we can expand our expressions of phenotypic selection to capture these second-order polynomial (quadratic) effects. Before discussing how to do this, it may be helpful to review some of the common nomenclature used in this area:

Stabilising selection is a negative association between fitness and the squared deviations from the trait mean. If this is sufficiently strong, then fitness may favour intermediate values. Human birth weight in the mid-twentieth-century population is the classic example of this phenomenon, as infant mortality is minimized at seven pounds but increases in smaller and larger infants (Karn and Penrose, 1951).

Disruptive selection is a positive association between fitness and the squared deviations from the trait means. If this is sufficiently strong, then fitness may favour extreme values.

Interaction selection is any association between fitness and the product of the deviation of two trait values from their respective means. Here, combinations of trait values can have emergent properties that help determine fitness.

It's important to note that these forms of what we can collectively term *quadratic selection* can co-occur with directional selection. For example, stabilising and positive directional selection together could suggest that fitness increases as a trait value increases, but fitness gains diminish as the trait value become more extreme. Finally, note that some biologists use slightly different definitions of stabilising and disruptive selection that effectively combine the linear and quadratic effects of the phenotype on fitness. In this usage, stabilising selection refers only to the case where fitness is maximized at an intermediate phenotypic value and disruptive selection is found where fitness is minimized at intermediate phenotypic values. The different definitions can create some confusion, but the exact meaning of the terms should be clear (or at least decipherable) from the context. To be clear, I will use the former definitions (as described in points 1–2 above) in what follows.

Estimating quadratic selection for some collection of traits $\mathbf{z}$ involves first defining *quadratic selection coefficients*. These are the multivariate extensions of the univariate selection coefficients discusses above. For n traits, we define an $n \times n$ matrix $\mathbf{C}$ with any element c_{ij} defined as the covariance between relative fitness and the product of deviations from means for traits ij,

$$c_{ij} = \text{cov}\big(w, (z_i - \bar{z}_i)(z_j - \bar{z}_j)\big) \qquad [5].$$

From here, we can take two different approaches to estimating *quadratic selection gradients*, which are, of course, the quadratic analogues to directional selection gradients. If we are comfortable with the assumption that the traits $\mathbf{z}$ are multivariate normal before selection, then the matrix $\boldsymbol{\gamma}$ defines a matrix of quadratic selection gradients (Lande and Arnold, 1983),

$$\boldsymbol{\gamma} = \mathbf{P}^{-1}\mathbf{C}\mathbf{P}^{-1} \qquad [6]$$

where $\mathbf{P}$ is the phenotypic covariance matrix discussed earlier. For any trait i, $\gamma_{ii} < 0$ favours stabilising selection and $\gamma_{ii} > 0$ favours disruptive selection. For any trait pair ij, $\gamma_{ij} < 0$ indicates negative interaction selection and $\gamma_{ij} > 0$ indicates positive interaction selection.

Unfortunately, we can seldom count on $\mathbf{z}$ being multivariate normal. In these cases, we cannot estimate $\mathbf{b}$ and $\boldsymbol{\gamma}$ independent of each other, because these may become statistically intertwined owing to the emergence of mean-variance or mean-covariance relationships. The solution here is similar to the strategy that we adopted to deal with estimating multivariate directional selection for correlated traits: we use multivariate regression on all traits simultaneously, except we now define some traits to be the products of deviations from their means. To do so, we construct an $n \times n$ matrix $\mathbf{A}$ that resembles $\mathbf{C}$, except that instead of covariances between relative fitness and products of deviations from trait means, the elements are simply the deviations from trait means,

$$a_{ij} = (z_i - \bar{z}_i)(z_j - \bar{z}_j) \qquad [7].$$

We then vectorise $\mathbf{A}$ and append this to $\mathbf{z}$ to construct a new trait vector $\mathbf{z}'$, such that $\mathbf{z}' = \begin{bmatrix} \mathbf{z} \\ vec(\mathbf{A}) \end{bmatrix}$. Using this trait vector, we construct a new phenotypic covariance matrix . $\mathbf{P}' = \begin{bmatrix} \mathbf{P} & \text{cov}(\mathbf{z}, vec(\mathbf{A})) \\ \text{cov}(vec(\mathbf{A}), \mathbf{z}) & \text{cov}(vec(\mathbf{A})) \end{bmatrix}$. Finally, we define a new selection coefficient vector

$\mathbf{s'}$ by appending the first-order trait selection coefficients to the vectorised $\mathbf{C}$, such that $\mathbf{s'} = \begin{bmatrix} \mathbf{s} \\ vec(\mathbf{C}) \end{bmatrix}$. Following eq. [4], the new selection gradient that follows is

$$\mathbf{b'} = (\mathbf{P'})^{-1}\mathbf{s'} = \begin{bmatrix} \mathbf{P} & \text{cov}(\mathbf{z}, vec(\mathbf{A})) \\ \text{cov}(vec(\mathbf{A}), \mathbf{z}) & \text{cov}(vec(\mathbf{A})) \end{bmatrix} \begin{bmatrix} \mathbf{s} \\ vec(\mathbf{C}) \end{bmatrix} \quad [8].$$

The resulting selection gradient $\mathbf{b'}$ has $n \times (n + 1)$ elements. The first n elements are directional selection gradients. The remainder are transformed by de-vectorization into an $n \times n$ matrix that defines quadratic selection gradients corresponding to the traits $\mathbf{z}$. Note that the off-diagonal elements of this matrix should be equivalent to one-half $\boldsymbol{\gamma}$, as derived by the Lande-Arnold method, if all elements in the covariance matrix $\text{cov}(\mathbf{z}, vec(\mathbf{A}))$ are zero[1]. Otherwise, $\boldsymbol{\gamma}$ cannot be taken as a reliable indicator of quadratic selection gradients.

Complications Owing to Social Interactions

Demographers are well aware that individual humans are social animals, and as such, interactions are fundamental to our biology. These interactions can have evolutionary impacts on phenotypes when between-individual interactions affect either how fitness views phenotypes (natural selection) or how phenotypes emerged from genotypes (inheritance). In the first case, social interactions may cause the fitness of an individual to be sensitive to the phenotypes of social partners. Natural selection generated in this fashion is known as *group-level selection*, which can contribute to a conceptually flexible multivariate perspective of natural selection termed *multi-level selection*. In the second case, the phenotypes of individuals may be determined to some degree by the genes of social partners, and we call these social genetic effects *associative* or *indirect genetic effects* (Griffing, 1968; Moore et al., 1997; Bijma et al., 2007). Phenotypic evolution approaches can quantify and separate the influence of both multi-level selection and social genetic effects on a response to selection, but a useful discussion of the latter is beyond the scope of this chapter. A more detailed description of this concept, as applied to post-reproductive survival in human populations, can be found in Moorad and Walling (2017). Here, I will focus on multi-level selection, or the manner by which social interactions affect phenotypic selection and how we may quantify these influences.

It may be clear by this point that while the phenotypic evolution notion of relative fitness is very rigid, this perspective is actually very flexible in how it defines a trait (for example, we have already seen how directional and quadratic selection is defined using first- and second-order aspects of the same phenotype). In principle, we are free to choose any possible feature that describes an individual and include that in our fitness regression. Using the method of *contextual analysis*, we include aspects of the distribution of social partner phenotypes in our selection analysis (Heisler and Damuth, 1987; Damuth and Heisler, 1988; Goodnight et al., 1992). Perhaps the most useful of such an approach would be to identify for each individual i the mean phenotypic values of the social partners of i and attribute this contextual trait to that individual. Let us refer to this social trait mean z' to distinguish this from the individual's trait z. Using a single trait for the purposes of illustration (but recognizing that multivariate extensions

1 Stinchcombe, J.R., A.F. Agrawal, P.A. Hohenlohe, S.J. Arnold, and M.W. Blows. 2008. *Evolution 62(9)*: 2435–2440.

to this approach are straightforward), we would perform a bivariate regression of relative fitness on both the individual and contextual trait. This regression would yield two partial regression coefficients: b_{wz} and $b_{wz'}$. The first is the slope of the regression of fitness on the individuals' phenotypes, holding the contextual trait constant. This is known as *individual-level selection*. The other aspect of multi-level selection, *group-level selection*, is quantified by the slope of the regression of fitness on the contextual trait, holding the individuals' phenotypes constant. Note that there are no logical constraints on what the values of these selection gradients might be. They can be identical or different in both magnitude and direction. In any case, evolutionary dynamics can become much more interesting when it happens that group-level selection is important. If this component of selection is important, then evolution can occur much faster or slower than would be suggested by a selection analysis that ignored contextual traits. In nineteenth-century Utah, for example, there is weak individual-level selection that favours females to reproduce with more than one male ($b_{wz} = +0.0827$, where z is the individual trait: the number of husbands). This probably reflects increased reproduction in young widows who remarry. However, individuals whose mothers reproduced with more than one male also benefit, and this is reflected in a positive group selection gradient of roughly the same magnitude ($b_{wz'} = +0.0075$; where z' is the contextual trait: the number of the mothers' husbands) (Moorad, 2013b). If there is any genetic variation for this trait in this population (which is not a given), then we could infer that group selection accelerates the evolution of polyandry slightly. In other situations where the selection gradients are in different directions, and group-level selection is much stronger than individual-level selection, a naïve individual-level selection analysis could, in principle, predict evolution in the wrong direction!

We can contextualize how multi-level selection contributes to the response to selection by recognizing that this response has both a direct component acting on selection for z and an indirect component acting through z'. Summing these two together yields to response to selection,

$$\Delta\bar{z} = b_{wz}\mathrm{var}(z) + b_{wz'}\mathrm{cov}(z', z) \qquad [9].$$

Recognizing that the covariance in eq. [9] can be expressed as the product of a slope and a variance, eq. [9] can be restated in a more useful way,

$$\Delta\bar{z} = b_{wz} + b_{z'z}b_{wz'}\mathrm{v})\mathrm{var}(z) \qquad [10],$$

where $b_{z'z}$, the slope of the regression of social partner mean phenotype on the individual phenotype, can be interpreted (in the absence of indirect genetic effects) as the coefficient of genetic relatedness between the social partners and the individuals. In most human populations, this coefficient between full siblings or between offspring and parent will be one-half, and between half siblings and between grandchildren and grandparent this will be one-quarter.

It is important to note that it is up to the investigator to define the group of social partners that interact with the focal individuals (and this choice hopefully reflects some interesting social dynamic), but this definition will affect the interpretation of the multi-level selection gradients. This "group" need not even be a group in the sense that it consists of a plurality of individuals — it can be a single individual, such as a mother, as in the example given above. In this case, the term "group-selection" may appear inappropriate, so *family-level* selection may be more palatable to some. Furthermore, there is no limit to the number of contextual traits

that can be applied to the same phenotypes. For example, it may be appropriate to consider a *trivariate* form of multi-level selection for some phenotype of interest in which maternal and grandmaternal trait values were included as contextual traits. For the purposes of predicting a response to selection, eq. [10] would need to be expanded to include two group selection terms (each weighted appropriately by relatedness of one-half and one-quarter).

Impossible Traits

Many demographers are interested in conditional traits, or those traits that are expressed in only certain individuals that meet some specific condition. For example, age of menarche is a trait limited to females, but a formal selection analysis should be applied to all individuals within the investigated population. In fact, all individuals *must* have all trait values included in the analysis to ensure that the **P**-matrix in eq. [4] is invertible. It is clear that these trait values must be imputed in those situations in which some traits are logically precluded from happening in some individuals. The appropriate value to impute is the mean value of the trait taken from the portion of the population that expresses that trait. However, a new trait should be added to the analysis to indicate whether or not a value was imputed (Moorad and Wade, 2013). The multivariate phenotypic selection analysis should include a selection gradient that applies to this indicator, or dummy trait, and one would interpret this to be the strength of selection acting on dichotomous expression of trait.

In the menarche example above, the indicator trait could be "female" (0 for male, 1 for female). For the sake of simplicity, I am ignoring the fact that some females will not live long enough to experience menarche (allowing for this would require a second indicator variable). Provided that we consider no other traits beyond the indicator trait (z_1, female) and the conditional traits (z_2, age at menarche), then our multiple regression that relates relative fitness to the traits of interest takes the form,

$$w = a + \beta_{wz_1} z_1 + \beta_{wz_2} z_2 + \varepsilon \,,$$

and the partial regression coefficients indicate selection gradients. The first coefficient β_{wz_1} represents the strength of selection for being born female. As human populations tend to have slight male bias at birth, one would expect that this term should be slightly positive in most cases. The reason for this is that because all humans have exactly one biological mother and one father, males and females collectively contribute equally to offspring production (the ultimate source of fitness). However, males are more numerous and thus can expect to have slightly less fitness each than the females. The second coefficient β_{wz_2} is selection for age at menarche in females. However, females make up less than half of all individuals at birth, so this partial regression coefficient will need to be weighted by the fraction of females in order to provide a selection gradient fit to be applied to predict a response to selection (Moorad and Wade, 2013).

Genetic Selection for Quantitative Traits

In the multivariate context, selection gradients provide a superior picture of fitness causality than selection coefficients, because the latter will combine both the direct effects of a phenotype on fitness and the indirect effects caused by correlations with all other traits that may have a more direct relationship with fitness. In principle, selection gradients will partition and identify

only the direct contribution, and this will provide a more complete model of causality. As discussed above, however, the causal model suggested by estimated selection gradients may be sensitive to the decision of whether or not to include particular traits in a selection analysis (Rausher, 1992; Morrissey et al., 2010). For this reason, biologists have been cautioned to treat selection gradients as only tentative suggestions for causal relationships between fitness and phenotypes to be tested by experimental manipulations (Wade and Kalisz, 1990). This is not possible for human populations for obvious reasons.

Rather than concern themselves overmuch with identifying causality, however, investigators may wish to know simply how much natural selection changes the mean of one trait in a single generation. To know this, one may independently estimate a selection coefficient (using the covariance between fitness and ancestral trait values) and narrow-sense heritability (using other quantitative genetic methods), and then take the product of these two estimates. However, this is not the most efficient use of data, and estimating the standard errors for this product is not straightforward. Fortunately, one can estimate directly the evolutionary change owing to the effects of natural selection using the genetic covariance between relative fitness and the trait of interest. This genetic covariance is interpreted as genetic selection for the trait. This approach appeals to the "Robertson-Price Identity" (Robertson, 1966; Price, 1970) that identifies the trait of interest to be the *genetic* or *breeding value* for that trait instead of the trait itself. In this way, the univariate Breeder's Equation $\Delta\bar{z} = \beta_{wz}\mathrm{var}(G)$ becomes $\Delta\bar{z} = \mathrm{cov}(G_w G_z)$, where this covariance is estimated directly from the data, usually by implementing a quantitative genetic bivariate "Animal Model" (Lynch and Walsh, 1998; Kruuk, 2004); this is a linear mixed-modelling approach that incorporates pedigree information in conjunction with phenotype data to yield estimates of **G**-matrices. A technical explanation for how Animal Models can be used to estimate genetic covariances is beyond the scope of this chapter, but the interested reader is recommended to read Wilson et al. (2010) for an accessible introduction to the subject intended for ecologists. It may also be useful to read Moorad and Walling (2017); at the time of this writing, this is currently the only Animal Model application of the Robertson-Price Identity used to estimate genetic selection in a human population. However, it should be noted before delving into Animal Models that the data requirements for estimating genotypic selection (in terms of sample size) can be far greater than that needed to estimate phenotypic selection. Information on several thousands of phenotyped and related individuals over multiple generations may be necessary for reasonably precise estimates of genetic covariances.

Final Remarks

The phenotypic evolution approach emphasises the role that the distribution of individual values of phenotypes and relative fitness play in trait evolution. This is, of course, the causal mechanism of evolution by natural selection articulated by Charles Darwin, but it is not a perspective that is shared by other approaches that may be familiar to demographers. For example, population projection matrices can be used to estimate selection gradients correctly in some situations. These approaches do not explicitly consider individual data, except as a means to summarize trait averages associated with shared states (e.g. age or size). As a result, among-individual variation, a property that is at the conceptual heart of natural selection, is not easily dealt with. It is my firm belief that individual-based methods employed by phenotypic evolution and quantitative genetics offer a superior approach to measuring a diversity of metrics

related to natural selection and inheritance in most cases. Some will disagree, but I hope that this chapter makes clear to all readers that these regression-based methods exist, and they are accessible and appropriate tools for demographers interested in understanding evolution in human populations.

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15. Demographic Sources of Variation in Fitness

Silke van Daalen and Hal Caswell

Heritable variation in fitness is required for natural selection, which makes identification of the sources of variation in fitness a crucial question in evolutionary biology. A neglected source of variance is the demography of the population. Demographic processes can generate a large amount of variance in fitness, but these processes are stochastic and the variance results from the random outcomes of survival, development and reproduction, and will therefore be non-heritable. To quantify the variance in fitness due to individual stochasticity, the mean and variance of lifetime reproductive output (LRO) are calculated from age-specific fertility and mortality rates. These rates are incorporated into a stochastic model (a Markov chain with rewards) and the statistical properties of lifetime reproduction — including Crow's Index of the opportunity for selection — are calculated. We present the basic theory for these calculations, and compare results with empirical measurements of the opportunity for selection. In the case of a historical population in Finland, 57% of the empirically observed opportunity for selection can be explained by individual stochasticity resulting from demographic processes. Analysing the contribution of demography to variance in fitness will improve our understanding of the selective pressures operating on human populations.

Introduction

Natural selection on a trait is an automatic consequence of three conditions: (1) there is variation among individuals, (2) the variation is heritable and (3) the trait is correlated with fitness, so that individuals differing in the trait experience differential reproductive success (Darwin, 1859; Lewontin, 1970; Brandon, 1978; Endler, 1986). Disentangling the underlying sources of variation in fitness, and of traits correlated with fitness, is a critical component of evolutionary biology, because not all variation is heritable or correlated with fitness.

Quantitative genetics provides powerful statistical tools for partitioning phenotypic variance into its components (e.g. Falconer, 1960; Kempthorne, 1957). The total phenotypic variance is customarily partitioned into genetic variance, environmental variance and variance that occurs as a result of gene-environment interactions. The genetic variance is further partitioned into additive and non-additive components (see Figure 1). Additive genetic variance is due to the linear contributions of alleles to the trait, and is the component of variance that determines the response to selection (Falconer, 1960; Lande, 1979). Non-additive variance arises due to dominance effects and epistatic effects. Heritability in the broad sense is the ratio of the genetic variance to the total variance. Heritability in the narrow sense, which determines the response

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to selection, is the ratio of additive genetic variance to the total variance (Crow and Kimura, 1970, p. 124).

In this chapter, we distinguish demographic analyses from other kinds of population calculations. By the demography of a species, we refer to the life cycle and its stages, the differences among individuals due to those stages, and the stochastic outcomes (surviving or not, reproducing successfully or not) of demographic processes in these stages. The familiar analysis of variance in quantitative genetics was developed with only minimal consideration of demography. As we will show, the contributions of these demographic processes (known as individual stochasticity; see Caswell, 2009) can be sizeable and should not be ignored. Methods now exist to calculate the demographic contributions to variance from standard life table information (Caswell, 2011; van Daalen and Caswell, 2015, 2017) and we will present these methods, together with examples, below.

Fitness and the Response to Selection: Crow's Index

Selection requires genetic variance, and the rate at which a trait responds to selection depends on the genetic variance in the trait and on the correlation of the trait with fitness. Fitness is, of course, perfectly correlated with itself, and so the response of fitness to selection is a useful starting point for analysis. Crow (1958) derived an index that measures the opportunity for selective improvement in fitness from the variance in fitness.

Suppose that the population contains k trait values and that individuals with trait value i have fitness w_i and occur with frequency p_i. The mean fitness at a given time is the sum of all possible fitness values weighted by their proportions, $\bar{w}(t) = \sum p_i w_i$. The frequency of trait i will change over time according to its current frequency and fitness:

$$p_i(t+1) = \frac{w_i p_i(t_i)}{\bar{w}(t)}$$

where the mean fitness scales $p_i(t+1)$ so that it sums to one.

Mean fitness in the next generation is $\bar{w}(t+1) = \sum p_i(t+1) w_i$, which, by replacing $p_i(t+1)$, can be written as

$$\bar{w}(t+1) = \frac{\sum w_i p_i w_i}{\bar{w}(t)}$$

The change in mean fitness from t to $t+1$ over time is $\Delta\bar{w} = \bar{w}(t+1) - \bar{w}(t)$. Crow (1958) writes this change as a proportion, relative to the mean fitness at time t, to obtain

$$\frac{\Delta\bar{w}}{\bar{w}(t)} = \frac{\bar{w}(t+1) - \bar{w}(t)}{\bar{w}(t)} = \frac{\sum p_i w_i^2 - \bar{w}(t)\sum p_i(t) w_i}{\bar{w}(t)^2} = \frac{V(w)}{\bar{w}(t)^2} = I$$

This is obviously related to Fisher's fundamental theorem of natural selection, which states that "the rate of increase in fitness of any organism at any time is equal to its genetic variance in fitness at that time" (Fisher, 1930: p. 35).

Crow's index I gives the proportional rate of increase in fitness when fitness is perfectly heritable, so that all the variance is genetic. The rate of change of any other trait would depend on the correlation of that trait with fitness (Crow, 1958). Crow's I has been referred to as "the index of total selection", "the intensity of selection", and "the opportunity for selection", the

latter of which most accurately represents its interpretation (Crow, 1958; Arnold & Wade, 1984; Cavalli-Sforza & Bodmer, 1999). Crow's I is an upper limit to the rate of the response to selection, but this limit is realized only if fitness is completely heritable and selection is not frequency-dependent.

Fitness and its Components

The definition and measurement of fitness are matters of great debate in ecology and evolution (e.g. Mills & Beatty, 1979; Metz, Nisbet & Geritz, 1992; Roff, 2008; Barker, 2009). It is clear that fitness is a demographic concept, because it measures the rate at which a particular phenotype or genotype is able to propagate copies of itself to future generations (Fisher, 1930; Dobzhansky, 1951; Hedrick, 1983; Barker, 2009, Metz, Nisbet, and Geritz, 1992). Such a rate is a demographic outcome.

Crow's definition of fitness avoids this; it simply states that the number of individuals with trait i increases by a factor ω_i in each generation, without specifying how that factor is determined. Fisher (1930) suggested the use of the intrinsic rate of increase r (the Malthusian parameter in Fisher's terminology) as a measure of fitness. It is calculated from survival and fertility schedules as shown in Table 1 (Fisher, 1930; Charlesworth, 1994). Metz and others have made a case for taking a similar, but more stringent measure of fitness: the rate of increase of a rare mutant in a resident population in a given environment, as measured by the dominant Lyapunov exponent (Metz, Nisbet, and Geritz, 1992; Metz, 2008). The discrete-time version of the population growth rate is $\lambda = e^r$.

All these demographic measures incorporate the life cycle, the changes that happen to individuals as they develop through the life cycle and some measure of rate of increase. Most evolutionary studies, however, must be satisfied with components of fitness that capture some aspects of survival, reproduction, growth, etc. even if they do not suffice to compute λ. The component perhaps most closely related to λ is lifetime reproductive output (LRO). The mean LRO, if measured as the number of daughters per female, is equivalent to the net reproductive rate R_0 (see Table 1), which is the per-generation rate of increase and, as such, serves as an indicator of population growth, decline or stability (Heesterbeek, 2002; Caswell, 2001). Both R_0 and LRO are often taken as a proxy for fitness (Grafen, 1988; Clutton-Brock, 1988; Newton, 1989; Partridge, 1989; Stearns, 1992; Roff, 1992; Charlesworth, 1994).

Measurement of LRO for a sample of individuals from a population provides an empirical estimate of the mean and variance, and thus of Crow's I, as

$$I = \frac{V(\mathrm{LRO})^2}{E(\mathrm{LRO})^2}$$

Such calculations are regularly carried out by demographers, anthropologists and population biologists (e.g. Clutton-Brock, 1988; Brown, Laland, and Borgerhoff Mulder, 2009; Courtiol et al., 2012). We will discuss these further below.

Lifetime reproductive output is, however, a demographic consequence of the complete set of stage-specific vital rates throughout the life cycle. It integrates the rates of survival, development and reproduction across age classes or stages, no matter how those stages are connected. Thus, LRO can be calculated from life tables or projection matrices, provided that they contain information on age-specific mortality and fertility (Caswell, 2001; 2011).

Table 1: Mathematical definitions of a few familiar fitness measures.

Measure	Variable	Equation
Total fertility rate	TFR	$\int_0^\infty m(x)dx$
Net reproductive rate	R_0	$\int_0^\infty \ell(x)m(x)dx$
Intrinsic rate of increase	r	$1 = \int_0^\infty e^{-rx}\ell(x)m(x)dx$
Population growth rate	λ	e^{rt}

Individual Stochasticity in LRO

Demography is a source of variance in fitness. In general, variance among individuals arises from two sources. One is heterogeneity: genuine differences among individuals, which translate into differences in the rates of mortality and fertility experienced by those individuals at any age or stage. This is the variance that is decomposed into the familiar environmental and genetic components (Figure 1). The other source is individual stochasticity, variance that arises from the stochastic outcomes of probabilistic transitions (living or dying, giving birth or not, maturing or not, etc.) within the life cycle. Variance due to individual stochasticity is unavoidable in any quantity that results from demography, but it is invisible in fitness calculations that ignore the demographic structure of the population.

Consider an extreme example, where every individual experiences the age-independent mortality rate μ. The longevity of individuals has an exponential distribution with a mean of $1/\mu$ (life expectancy), and a variance of $1/\mu^2$. This variance is a result of individual stochasticity, because by assumption we have eliminated every source of heterogeneity from this example.

The same principle holds when the vital rates depend on age — conditional on age, individuals experience the same rates and probabilities, but may differ in their outcomes. Calculating the amount of variance in LRO produced by stochastic events in the life cycle has been a long-standing problem, which has recently been solved (Caswell, 2011; van Daalen, and Caswell, 2017). In the next section we will present these results, and we will apply them to Finnish population data as an example.

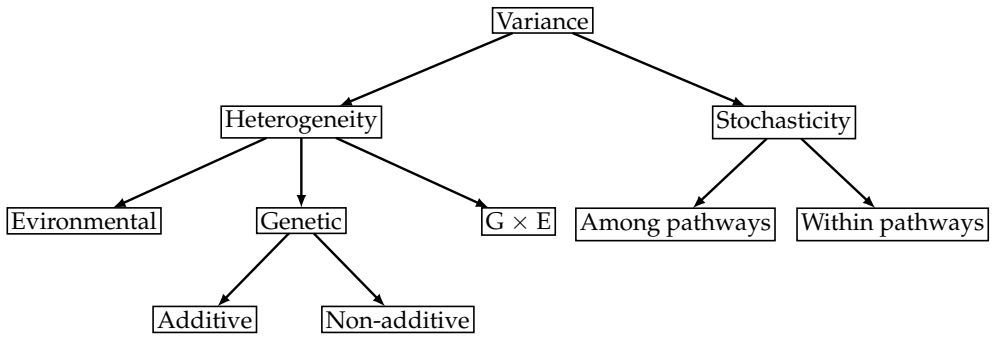

Fig. 1 Variance among individuals is caused by heterogeneity, i.e. actual differences between individuals, and by stochasticity, i.e. differences in outcome by chance.

A Markov Chain Model for Stochasticity in LRO

Individual stochasticity can be calculated by incorporating demographic processes into a stochastic model for individuals. An individual in age class i may survive and advance to the next age class (with probability p_i) or die (with probability $1 - p_i$). It will reproduce with probability f_i.[1] The probabilistic nature of surviving or dying at a given age class causes random variation among the pathways individuals follow through their life course. Similar random variation is caused within pathways by probabilistic fertility (Figure 1).

These probabilities are captured in a stochastic model framework referred to as absorbing Markov chains with rewards (Caswell, 2011; Caswell and Kluge, 2015; van Daalen and Caswell, 2015; 2017). The Markov chain describes the movement of individuals among a set of states, in this case, among age classes. An individual of any age has a probability of surviving to the next age class. These probabilities appear on the sub-diagonal of the transition matrix of the Markov chain (see Box I). Individuals who die are captured into the absorbing state of death. This model keeps track of all possible trajectories that individuals take through their life course, from birth to eventual death, and the probabilities of each.

At each step in its trajectory, an individual may accumulate offspring. These offspring are treated as "reward" in the Markov chain model. Rewards accumulate until the individual dies. Thus, defining rewards as offspring in this analysis leads directly to a measure of lifetime reproductive output. The statistical moments of rewards are incorporated into a set of reward matrices (see Box II). For humans, we assume that the fertility at age i is the probability of producing a single child, which implies the higher moments of the reward matrix follow a Bernoulli distribution. With this structure, a Markov chain with rewards model incorporates the full range of stochasticity, as it arises partly as a consequence of probabilistic survival and transitions, and partly as a consequence of probabilistic success at reproduction.

Box I - Markov Chains

The Markov Chain is specified by a transition matrix $\mathbf{P}$, which includes a submatrix $\mathbf{U}$ describing transitions and survival. Given ω age classes, the matrix $\mathbf{U}$ contains survival probabilities p_i on the subdiagonal and zeros elsewhere; e.g., for $\omega = 4$,

$$\mathbf{U} = \begin{pmatrix} 0 & 0 & 0 & 0 \\ p_1 & 0 & 0 & 0 \\ 0 & p_2 & 0 & 0 \\ 0 & 0 & p_3 & [p_4] \end{pmatrix},$$

where the optional p_4 in the lower right corner creates an open-ended final age class.

The transition matrix $\mathbf{P}$ is

$$\mathbf{P} = \left(\begin{array}{c|c} \mathbf{U} & \mathbf{0} \\ \hline \mathbf{M} & \mathbf{1} \end{array}\right)$$

where $\mathbf{M}$ is a $1 \times \omega$ matrix of age-specific mortality probabilities. The final state is death; the 1 in the lower right corner indicates that death is a permanent absorbing state.

1 If we ignore multiple births, probabilistic fertility for humans takes the form of a Bernoulli random variable, so that reproduction is 1 with probability f_i or 0 with probability (1- f_i).

Box II - Reproductive "rewards"

In Markov chain models, reproduction can be incorporated as a reward associated with a transition from one age class to the next. Individuals moving from age class i to age class j thus collect the reward r_{ji}. Age-specific fertility rates f_i can be treated as the age-specific probabilities of collecting a reward, under the assumption women produce 1 child at a time (disregarding twins), making the reward a random Bernoulli variable;

$$r_{ji} = \begin{cases} 1 \text{ with probability } f_i \\ 0 \text{ with probability } (1 - f_i) \end{cases}.$$

In order to calculate the statistical moments of lifetime accumulated rewards, we must define the different moments of the *reward matrix*. $\mathbf{R}_k$ is a matrix of the kth moment of the transition-specific rewards r_{ji}. Under the assumption that rewards depend only on the current age class of the individual, the first moment reward matrix becomes

$$\mathbf{R}_1 = \left(\begin{array}{ccc|c} f_1 & \dots & f_\omega & 0 \\ \vdots & \ddots & \vdots & \vdots \\ f_1 & \dots & f_\omega & 0 \\ \hline f_1 & \dots & f_\omega & 0 \end{array}\right)$$

with the last column corresponding to rewards accumulated by individuals who are dead; these are, unsurprisingly, always zero. Due to the fact that reproduction is treated as a Bernoulli random variable, the higher moments of the reward matrix are easily obtained as

$$\mathbf{R}_2 = \mathbf{R}_3 = \mathbf{R}_1.$$

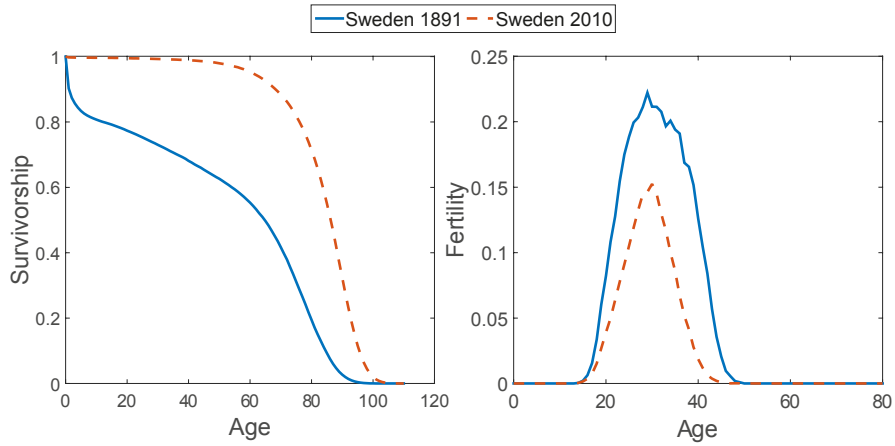

Fig. 2 Survivorship (left panel) and fertility (right panel) for Sweden at two different points in time, 1891 (solid blue line) and 2010 (dashed red line).

The accumulated number of children over the lifetime of an individual, i.e. lifetime reproductive output, is then a function of the Markov chain $\boldsymbol{P}$, the reward matrix $\boldsymbol{R}_i$, and the fundamental matrix $\boldsymbol{N}$. The latter is obtained from the Markov chain and provides information

on the occupation times of different age classes and longevity (see Box III). Calculating Crow's index of the opportunity for selection requires the first two moments of LRO (see Box IV), but calculation of all higher moments is also possible (van Daalen and Caswell, 2017).

As an example, consider Sweden in 1891 and 2010 (Figure 2). Survival was higher in 2010 than in 1891; fertility was lower in 2010 than in 1891. The Markov chain with rewards calculation shows that, given these rates, a Swedish woman in 1891 would produce an average of three children (of either sex) over her lifetime. The variance in lifetime reproduction, among a cohort of women identically experiencing the rates of 1891, would be 5.6. Crow's index would be $I = 0.62$. This variance is due to the random outcomes of the age-specific probabilities of survival and reproduction. By 2010, mean LRO had declined to two children. The variance, again among individuals identically experiencing 2010 rates, would be 1.8, with Crow's $I = 0.46$ (see Table 2).

Table 2: Mean lifetime reproductive output, variance and opportunity for selection calculated using Markov chains with rewards for Sweden at two points in time. The model was parameterized using mortality and fertility as shown in Boxes I and II.

Model outcome	Sweden 1891	Sweden 2010
Mean LRO	2.997	1.972
Variance in LRO	5.595	1.788
Opportunity for selection	0.623	0.460

Box III - Longevity and the fundamental matrix

U contains all the information of a life table, and makes it possible to calculate survivorship, life expectancy, variance in longevity, and other statistics. We will use the fundamental matrix

$$\mathbf{N} = (\mathbf{I} - \mathbf{U})^{-1},$$

where $\mathbf{X}^{-1}$ is the inverse of the matrix $\mathbf{X}$. The entries of $\mathbf{N}$ correspond to the mean amount of time spent in any age class, given that you start in any age class. For example, the first column of $\mathbf{N}$ will be a vector of mean time spent in age class i for any individual starting life in the first age class. By summing over all the columns in $\mathbf{N}$, as

$$\boldsymbol{\eta}^{\mathsf{T}} = \mathbf{1}^{\mathsf{T}}\mathbf{N},$$

a vector of remaining life expectancy from each starting age is obtained. The first entry of this vector is mean longevity, or life expectancy at birth.

Box IV - Lifetime reproductive output

With the ingredients described in the previous boxes, we can calculate the different statistical moments for the lifetime number of children for women at any starting age. The vector $\tilde{\boldsymbol{\rho}}_k$ represents the kth moment of accumulated rewards for individuals who start their life in any of the living classes. The first moment of lifetime reproductive output is mean lifetime reproductive output, obtained as

$$\tilde{\boldsymbol{\rho}}_1 = \mathbf{N}^{\mathsf{T}}\mathbf{Z}\left(\mathbf{P}\circ\mathbf{R}_1\right)^{\mathsf{T}}\mathbf{1},$$

where $\mathbf{Z}$ is a matrix that cleaves off the absorbing state of death, $\mathbf{N}^{\mathsf{T}}$ refers to the matrix transpose of $\mathbf{N}$, the product $\mathbf{P}\circ\mathbf{R}_1$ is the element-by-element product of $\mathbf{P}$ and $\mathbf{R}$, and $\mathbf{1}$ is a vector of ones. The second moment of lifetime reproductive output is

$$\tilde{\boldsymbol{\rho}}_2 = \mathbf{N}^{\mathsf{T}}\left[\mathbf{Z}(\mathbf{P}\circ\mathbf{R}_2)^{\mathsf{T}}\mathbf{1} + 2(\mathbf{U}\circ\mathbf{R}_1)^{\mathsf{T}}\tilde{\boldsymbol{\rho}}_1\right].$$

From these, the variance in lifetime reproductive output can be obtained, as

$$V\left(\tilde{\boldsymbol{\rho}}\right) = \tilde{\boldsymbol{\rho}}_2 - \tilde{\boldsymbol{\rho}}_1\circ\tilde{\boldsymbol{\rho}}_1.$$

The mean and variance combined provide the opportunity for selection,

$$\mathcal{I} = V\left(\tilde{\boldsymbol{\rho}}\right)\mathcal{D}\left(\tilde{\boldsymbol{\rho}}_1\circ\tilde{\boldsymbol{\rho}}_1\right)^{-1},$$

where $\mathcal{D}\left(\mathbf{y}\right)$ puts the entries of the vector $\mathbf{y}$ on the diagonal of a matrix containing zeros elsewhere. Further details and derivations of these equations are shown in van Daalen and Caswell (2017).

It is important to be clear about what this variance reflects. The calculations assume that every woman experiences the same fertility and mortality rates at every age, so there is no heterogeneity involved on which to select. This lack of heterogeneity, genetic or otherwise, means that this estimate of Crow's I is based on demographic variation with a completely non-heritable basis. We therefore refer to it as an apparent, rather than a real opportunity for selection.

These values of Crow's I due to individual stochasticity are not unusual. In an analysis of fertility during the second demographic transition, we calculated Crow's index for a set of forty developed countries (van Daalen and Caswell, 2015), based on age-specific demographic data from the Human Mortality Database (Human Mortality Database, 2014), the Human Fertility Database (Human Fertility Database, 2014) and the Human Fertility Collection (Human Fertility Collection, 2014). We found values of Crow's I in the range of roughly 0.25 to 1.0, increasing slightly between 1960 and 2000, with a slight decrease after 2000 (see Figure 3). Van Daalen and Caswell (2017) found values in a similar range for two hunter-gatherer populations and the high-fertility Hutterites.

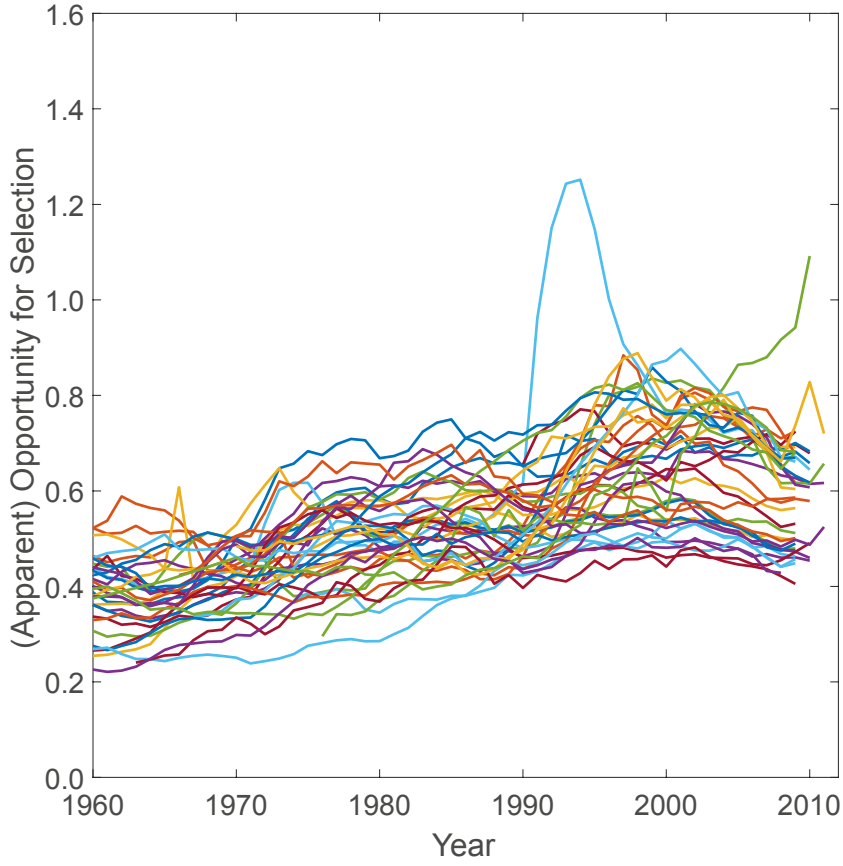

Fig. 3 Apparent opportunity for selection calculated using the Markov chain with rewards method for forty developed countries between 1960 and 2010. Values fall broadly within a range of 0.2–0.8.

Empirical Estimates of the Opportunity for Selection in Human Populations

Given individual lifetime data, it is possible to make empirical estimates of Crow's *I*. Such data have been used to investigate reproduction in a number of human populations. The index is often partitioned by sex (Crow, 1962; Wade, 1979; Brown, Laland and Borgerhoff Mulder, 2009), by episodes of selection (Crow, 1958; Arnold and Wade, 1984) or by the type of selection. The latter partitioning was developed by Wade (1979; 1995), who derived an expression for the opportunity for sexual selection from Bateman's observations on variance in number of mates (Bateman, 1948).

Table 3: Empirical measures of opportunity for selection (OFS) in reproductive success of women in 18 populations, with a median of 0.34, and an interquartile range of 0.16–0.46.

Country or population	Mean LRO	Variance	OFS
Paraguay, Ache	7.8	3.6	**0.06**
Chad, Arabs	8.3	5.1	**0.07**
C.A.R., Aka	6.2	5.2	**0.14**
Kenya, Kipsigis	6.6	5.9	**0.14**
Chad, Dazagada	6.4	6.5	**0.16**
Tanzania, Pimbwe	6.1	7.3	**0.20**
Botswana, Dobe !Kung	4.7	4.9	**0.22**
Mali, Dogon	3.2	2.3	**0.22**
Brazil, Xavante	3.6	3.9	**0.30**
Venezuela, Yanomamo	3.4	4.4	**0.38**
Tanzania, Hadza	3.6	5.1	**0.39**
Norway 1700-1900	4.5	8.3	**0.41**
USA social survey	2.0	1.8	**0.45**
Dominica locals	5	11.6	**0.46**
Iran, Yomut Turkmen	3.9	7.1	**0.47**
Finland 1745-1900	3.5	7.6	**0.62**
Pitcairn Isl. genealogies	4.7	23.2	**1.05**
Sweden, 1825-1896	2.4	9.7	**1.68**

Brown, Laland and Borgerhoff Mulder (2009) have compiled such empirical estimates of the sex-specific opportunity for selection in eighteen human populations. In Table 3 we have tabulated their estimates as female opportunity for selection. The values (median=0.34, interquartile range=0.16–0.46) are similar to those produced by individual stochasticity in typical human life tables, with only three populations exceeding 0.5. The estimates of opportunity for selection were higher in males in most populations, something that might reflect sex roles, but for which there was not sufficient evidence (Brown, Laland and Borgerhoff Mulder, 2009).

Brown, Laland, and Borgerhoff Mulder (2009) also found differences among mating systems (i.e. monogamy, serial monogamy, polygyny, etc.) in the degree to which male opportunity for selection outweighed female opportunity for selection. A more robust study by Moorad et al. (2011) showed that a shift in the mating system of a frontier population in Utah between 1830 and 1894 reduced the opportunity for selection over time. The opportunity for selection was high in males in 1830 (approximately 1.1) and decreased by almost half around mid-century, corresponding to the shift from polygyny to monogamy. For women, the opportunity for selection was quite stable across this time period (around 0.5–0.6).

Nulliparity as an Issue

A confounding issue in empirical measurements of variance in LRO is the treatment of individuals that do not reproduce at all (nulliparous individuals), either because they die before reproducing or simply never produce children. These individuals are often excluded from estimates, thereby underestimating the variance in lifetime reproduction. The study by Moorad et al. (2011) is an apparent exception, but to our knowledge, the only study of opportunity for selection in human populations that includes explicit counts of nulliparous individuals is that of Courtiol et al. (2012). They used detailed church records of preindustrial populations in

Finland between 1760 and 1849 to obtain counts of lifetime reproductive output for all women, nulliparous or not.

Courtiol et al. (2012) estimate the opportunity for selection for women as $I = 2.03$, which is distinctly higher than other empirical estimates (Table 3) and higher than estimates calculated from life tables (Figure 3). They found no evidence for effects of social status on the opportunity for selection. The opportunity for selection was again higher in males, estimated at around 2.52. The larger values in this study are most likely due to the inclusion of nulliparous individuals in the estimates of Crow's I, and as such they are a benchmark estimate, at least for a preindustrial European population.

Pre-industrial Finland: Variance and Stochasticity

The empirical estimate of the opportunity for selection reported by Courtiol et al. (2012) for Finnish women in the late eighteenth to mid-nineteenth century provides a valuable opportunity for comparison with the level of variance due to individual stochasticity that is implied by the mortality and fertility schedules of that era. To make such a comparison, we require mortality and fertility schedules as comparable as possible to those of the Finnish population represented by the parish register data. If the variance in LRO due to individual stochasticity is similar to the observed value, invoking heterogeneity to explain the variance is, strictly speaking, not necessary without additional evidence (Steiner and Tuljapurkar, 2012).

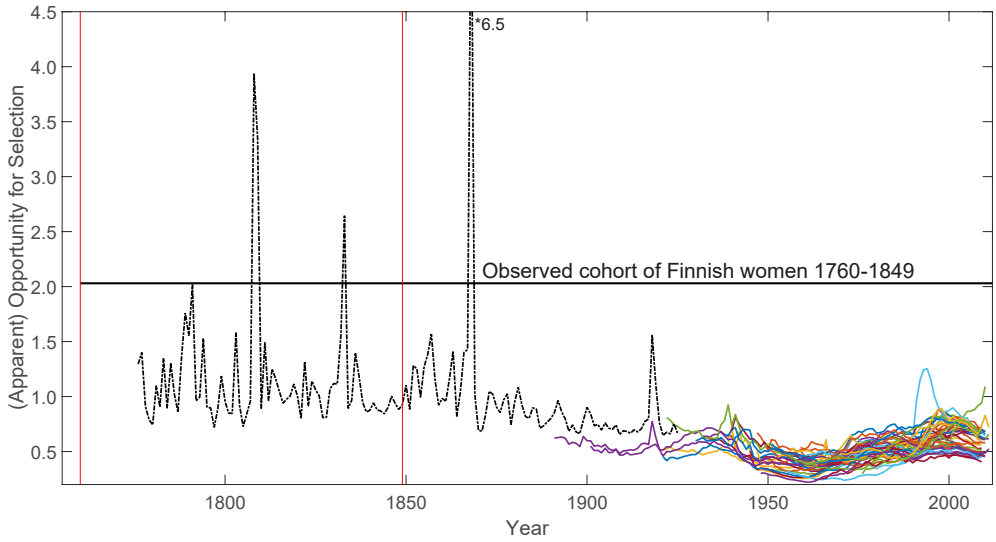

Fig. 4 The straight back line represents the empirically measured opportunity for selection for Finnish women living between 1760 and 1849; the window of time wherein this measure was obtained is indicated by vertical red lines. The black dash-dotted line represents apparent opportunity for selection obtained from life table data on Finnish women living between 1776 and 1925. Peaks correspond to famines and wars, times when mortality was higher. The apparent opportunities for selection for forty developed countries are shown as a reference in the bottom-right corner of the figure (coloured lines).

Turpeinen (1979) reported estimates of age-specific mortality and fertility for Finland, from 1776 to 1925. We interpolated Turpeinen's results from five-year age classes (or, for the first

years of life, two-year age classes) to single-year age classes using cubic splines. We used the resulting mortality schedules to create the matrix **U**, and the fertility data to create the reward moment matrices $\mathbf{R}_1$ and $\mathbf{R}_2$, for each year (see Boxes I and II), and calculated the resulting values of Crow's *I*, as shown in Figure 4.

The mean value of opportunity for selection between 1776 and 1849 was 1.15, which is 2–3 times the value for current developed countries. This value declined gradually from the mid-nineteenth century to the typical modern values. This is at least partly a result of the reduction in mortality since that time, because such changes reduce *I* (see the sensitivity analysis results in van Daalen and Caswell, 2017).

Like all empirical estimates, the values of Crow's *I* reported by Courtiol et al. (2012) reflect both sources of variance in LRO (see Figure 1). Compared to their value of 2.03, the value of apparent opportunity for selection implies that slightly more than half of Crow's *I* can be accounted for by individual stochasticity. The remainder, approximately 0.8, could be due to heterogeneity. This heterogeneity could be genetic, or it could be non-genetic, such as marital status, parity or geographical location (Figure 1).

Discussion

Natural selection is, at heart, a demographic process, concerned as it is with the differential propagation of genes, genotypes or traits (Metcalf and Pavard, 2007). This demographic basis is recognized in the calculation of fitness (measured as some rate of increase that integrates survival and reproduction) and fitness components (measured by indices that capture some, but not all, aspects of survival and reproduction).

To this familiar concept, we must also add demography as a source of *variance* in fitness and its components, due to individual stochasticity. The existence of random events within the life cycle makes this stochasticity an unavoidable result, implicit in any demographic model. It has now been shown, by a variety of methods, that individual stochasticity creates significant amounts of variance in human and non-human populations (e.g. Caswell, 2009; Tuljapurkar, Steiner and Orzack, 2009; Steiner, Tuljapurkar and Orzack, 2010; Caswell, 2011; Steiner and Tuljapurkar, 2012; van Daalen and Caswell, 2015; Hartemink, Missov and Caswell, 2017; van Daalen and Caswell, 2017).

Human life tables imply a degree of individual stochasticity in LRO that is sufficient to create values of Crow's *I* that are on the same order as empirical measurements of variance in lifetime reproduction. This result has several implications. It provides a baseline against which empirical measurements can be compared. It serves as a neutral model (*sensu* Steiner and Tuljapurkar, 2012), eliminating all sources of heterogeneity, and implies the need to search for evidence of heterogeneity in order to invoke it as a source of the variance. The variance produced by individual stochasticity can be expected to reduce the efficacy of natural selection, by masking variance produced by genetic differences (Steiner and Tuljapurkar, 2012).

In the case of the high-quality empirical measurements of lifetime reproductive output in pre-industrial Finland, roughly 60% of the empirically measured value of Crow's *I* can be accounted for by individual stochasticity arising from the demographic properties of seventeenth-century Finland as reported by Turpeinen (1979).

Whether the Finnish population serves as a general or an exceptional example, we cannot say. The Finnish data are exceptional with regard to their inclusion of nulliparous individuals

(Courtiol et al., 2012), whereas other studies try to compensate their results for nulliparity (Moorad et al., 2011). It is clear that leaving out unsuccessful individuals changes estimates of the variance and the opportunity for selection (Klug, Lindström and Kokko, 2010; Courtiol et al., 2012). Although in many studies there will be logistical limits, including nulliparous individuals in empirical studies is essential for comparisons that allow insight into the underlying sources of variance, in addition to providing representative data with which to parameterize models.

Showing that individual stochasticity can account for some fraction (or all) of the observed variance does not prove that it does so. To measure the contributions of individual stochasticity and heterogeneity, one must incorporate the relevant source(s) of heterogeneity into the Markov chain model, and assign reproductive rewards to both age classes and heterogeneity categories. It is then possible to decompose the variance in LRO into components due to heterogeneity and stochasticity (Caswell et al., 2018). Variance decomposition has been applied to longevity in both humans (Hartemink, Missov and Caswell, 2017) and animals (Hartemink and Caswell, 2018, Jenouvrier et al., 2017) and to LRO (Jenouvrier et al., 2018; van Daalen and Caswell, in prep.). The data requirements are more demanding, but the matrix framework for such analyses exists.

The methods presented here (and in more detail in Caswell, 2011; van Daalen and Caswell, 2017) make it possible to calculate the individual stochasticity in lifetime reproductive output implied by any set of mortality and fertility schedules. This opens the way for increasingly detailed study of the demographic contribution to variance in fitness, and its implications for human evolutionary demography.

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16. Ageing in the Wild, Residual Demography and Discovery of a Stationary Population Equality

James R. Carey

In the late 1990s, while exploring methods for estimating population age structure using the post-capture longevity of fruit flies sampled from the wild (referred to as residual demography), I discovered an identity in which the fraction of individuals x days old in a stationary population equals the fraction that die x days later. I co-authored a paper containing this identity in 2004 as part of a larger publication with my biodemography colleagues, in which we extended the concept for practical application. In 2009, demographer James Vaupel published a proof of this identity and referred to it using the eponym Carey's Equality. The Vaupel paper was then followed six years later (2015) by a surprise — the identity had been published in French thirty years earlier in the grey literature by demographer Nicolas Brouard. Remarkably the identity had never been cited in either the searchable (journal) literature or in any of the mainstream demography texts, treatises, encyclopaedias or reference books. Here I tell the story of how I discovered this identity, why it is important, implications for human demography and lessons learned along the way.

Introduction

A feature of many interdisciplinary fields such as biodemography is that questions are often asked that had previously not been considered by scientists in either of the "parent" disciplines (i.e. demography and biology). Presumably this is because the questions were not thought of in the first place, or, alternatively, because the answers were perceived in the respective fields to be of no general interest, to serve no conceptual purpose or to solve no pertinent problem.

In this chapter I describe a personal experience in which a question emerged during discussions with several of my biodemography colleagues in the late 1990s that appeared not to have been asked before (at least in the same way): *What can information gathered on field-captured Mediterranean fruit flies (medflies) monitored through death in the laboratory tell us about population ageing in the wild?* This question was the precursor that, in the late 1990s, led to my discovery of a mathematical identity unique to stationary populations stated as "the fraction of individuals x days old in a stationary population equals the fraction of individuals that die x days later." This identity was published in a jointly-authored paper with Hans Müller, Jane-Ling Wang and other colleagues (Müller et al., 2004), followed six years later by its proof

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the *Carey's Equality eponym* (Vaupel, 2009a), which, in turn, was followed by a paper with original analytical concepts and new theoretical insights into the identity (Rao and Carey, 2015).

The story thread focuses primarily on my quest to find a practical method for estimating age structure in medfly populations that, along the way, yielded the serendipitous discovery of this population identity. For important context, this journey was preceded by an earlier one that started when I was a participant in a 1987 workshop supported by the National Institute on Aging (NIA) that was spearheaded by the late Richard Suzman (who was then Associate Director of the NIA Division of Behavioural and Social Research) and organized by demographers Sheila Ryan Johannson and Kenneth Wachter at the University of California, Berkeley (Carey and Vaupel, 2019). Titled "Upper limits to human life span", this workshop brought together biologists and demographers to discuss issues concerning ageing and longevity in the oldest-old (persons > 85 years old), but with a particular focus on the question concerning the existence of a specific lifespan limit in humans.

These discussions ultimately led to my involvement as a principal investigator in two different NIA-funded programs. One of these focused on oldest-old mortality (directed by James Vaupel) and the other focused on ageing in the wild (directed by myself). One of the major findings from one of the studies my colleagues and I conducted in the Vaupel-directed program was that mortality in a 1.2 million medfly cohort slowed at older ages (Carey et al., 1992). This outcome supported the hypothesis that medflies and likely many other species including humans do not possess specific limits to lifespan. Because this study used the medfly as a model system in the laboratory, the theme of the NIA-funded program that I directed was concerned with ageing in the wild. The results of these field studies on the medfly were designed to complement the laboratory studies as well as expand perspectives on ageing in evolutionarily-relevant (i.e. natural) environments.

Importance of Age in Demography and Biology

Arguably the greatest difference between population studies of humans and population studies of non-human species is the gulf in the availability of age-specific data. Whereas it is nearly ubiquitous in the former, it is mostly absent in the latter.

Without age information, human demography would be unimaginable in some types of studies and impossible in others, e.g. constructing Lexis diagrams, disaggregating age-period-cohort effects, tabulating actuarial rates, predicting future births and deaths, analysing migration trends, projecting population numbers or developing population policies. Indeed, demographers concerned primarily with human populations consider age as central to and as inextricable from their discipline as the concept of supply and demand is to economists, Darwinian selection is to evolutionary biologists and differential calculus is to mechanical engineers. Without age data, the field of demography would be reduced to a shadow of its current self at best and completely disappear at worst. Aside from population studies in a few sub-specialties in human demography (e.g. remote indigenous peoples), the absence of age data in human population studies is the rare exception.

The situation is the near-exact opposite in the vast majority of population studies concerned with non-human species. For example, the accuracy is extremely low and the costs generally extremely high for virtually all of the methods used to estimate insect age (Lehane, 1985) including wear-and-tear (Tyndale-Biscoe, 1984), cuticular hydrocarbon layering (Gerade et al.,

2004), accumulation of bio-compounds (Lehane, 1985), and transcriptional profiling (Cook, McMeniman, and O'Neil, 2008; Cook and Sinkins, 2010). No ageing method has ever been routinized as part of a standardized surveillance program in applied insect ecology such as for monitoring insect disease vectors (mosquitoes, tsetse flies) where insect age is an extremely important component in disease transmission (Cook, McMeniman, and O'Neil, 2008). For vertebrates there are some exceptions including (1) long-term mark-recapture studies on selected species of birds and large mammals (Nussey et al., 2006; Ozgul et al., 2009) and (2) ecological studies spanning many taxa that use post-mortem techniques to estimate age including otolith layering in fish (Campana and Thorrold, 2001; Limburg et al., 2013) and tooth wear in wildlife (Dinsmore and Johnson, 2012). Although there are a number of relatively recent papers for estimating age- and stage-specific life history parameters (Cochran and Ellner, 1992; Metcalf et al., 2009; Davison, 2011; Horvitz and Tuljapurkar, 2008), this is a different concept than that for estimating the age of individuals.

The profundity of not having information on individual and population age in studies of non-human species is not recognized by the majority of mainstream demographers because of their exclusive focus on humans. But this lack is deeply frustrating to the majority of population biologists and applied ecologists. This is because the absence of information on age and age structure in populations of non-human species severely limits the scope and depth of demographic analysis and modelling in several important respects. Firstly, the majority of the most sophisticated demographic models in the literature are developed for and concerned with human populations. These methods both assume and require information on individual-age and population-age structure. Therefore, without age data on non-human species, many of the classical demographic models including cohort life tables and age-structured population models apply in theoretical and laboratory contexts rather than in the wild settings where they are the most relevant.

Secondly, age is a major source of risk which, as a general concept, underlies the quantification of various age-specific force-of-transitions, for example in sexual maturation, marriage and divorce, reproduction, disease acquisition, disablement, retirement and death. Because force-of-transition concepts apply to changes of state in species across the Tree of Life (Jones et al., 2014), the lack of age information limits demographic analysis.

Thirdly, the results of demographic studies in the laboratory are of marginal value without the availability of age data for cohorts and populations in the field. These limitations frequently preclude opportunities to refine, adapt and expand powerful demographic tools for use in analysis of populations of non-human species. They also restrict the range of possibilities for creating new demographic concepts and building new models based on the treasure-trove of life history (and thus demographic) characteristics observed across the Tree of Life.

In light of the near-absence of methods for estimating age structure in fruit fly populations and the importance of finding a method, several of my biodemography colleagues and I set about trying to develop new concepts for studying ageing in the wild. We believed that it might be possible to achieve a better outcome for estimating age structure in insect populations using demographic models than with costly and mostly inaccurate high-tech methods used to estimate the age of individual insects.

Discovery and Formulation in Four Stages

A retrospective examination of the process that took me from an idea to the discovery of the life table identity occurred in four stages. Remove any of the first three stages or even one of the within-stage details, and I would likely not have discovered the identity. The last stage was model formulation after having identified the key equivalency. Key parts of the following sections are taken from a paper by myself and co-workers written at approximately the same time as this one (Carey, Silverman, and Rao, 2019).

Stage I: Framing the Concept

The germ of the idea that ultimately led to the discovery of the population identity was motivated by my view, and that of many insect ecologists, that the conventional methods for estimating individual age and age structure in wild populations described earlier are sorely lacking. Thus, the question that arose in 1998 on a research retreat in Crete (Greece) involving myself, mathematical demographer Anatoli Yashin and geneticists Lawrence Harshman and Linda Partridge was: "*What can be learned about aging in the wild from information gathered on field-captured fruit flies of unknown age monitored in the laboratory?*" This initial question, the concept of which is illustrated in Figure 1, was framed around the potential use of biological information in what Partridge referred to as "residual demography", e.g. the post-capture levels and patterns of egg laying; challenge assays such as starvation and desiccation resistance; health status and remaining longevity. None of us had an inkling that this biological idea would lay the groundwork for the discovery of a population identity.

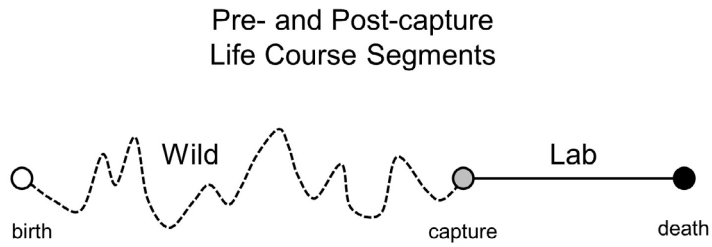

Fig. 1 Schematic diagram of the captive cohort concept in which the life course of an individual is divided into pre- and post-capture segments. An individual is born (open circle), lives an unknown fraction of its life in the wild (dashed line), is captured (shaded circle), and is monitored through death (black-filled circle).

Stage II: Simulation Studies

Inasmuch as I am trained as an insect ecologist and not as a mathematical modeler per se, I asked several of my mathematical demography colleagues what statistical concepts would be required to estimate the age structure of a fruit fly population using the post-capture survival information of wild-caught individuals of unknown age.

Suppose a fly of unknown age is captured in the wild and lives for twenty-three days in the laboratory. Assuming Gompertzian mortality rates and a maximum lifespan in the laboratory of sixty days for this (hypothetical) species, what is the best estimate of the age of this individual fly when it was captured, and the confidence intervals for this estimate?

The consensus among my colleagues was that answering this question would require development of a sophisticated Bayesian statistical model. Given the time and statistical expertise required to build a model designed to answer this type of question, the biological studies needed to both parameterize and validate it species-by-species, and the likelihood that both the accuracy and precision of the model would be low, I decided that statistical model-building should not be my next step. Instead, I decided my most parsimonious next step should be simulation studies designed to answer the question: "*What are the survival patterns of a group of fruit flies that are each captured at random ages in a computer-generated stationary population?*"

Example results of simulations based on laboratory data collected on 1,000 individual medflies (Carey et al., 1998) revealed distinct patterns that were much different than those for a cohort of newly-enclosed fruit flies (Figure 2). This was no surprise, since survival of the group involved individuals of different ages and thus different composite mortality rates. But on seeing the consistency of the survival patterns, my immediate thought was that there had to be a mathematical explanation that would account for their similarity. Follow-up simulations using replicates with sample sizes in the many thousands of individuals yielded virtually identical post-capture survival curves thus confirming the idea not only that there existed an underlying mathematical model, but that it might be simple. Although in retrospect the results of these simulations now appear obvious, they were not obvious to me at the time.

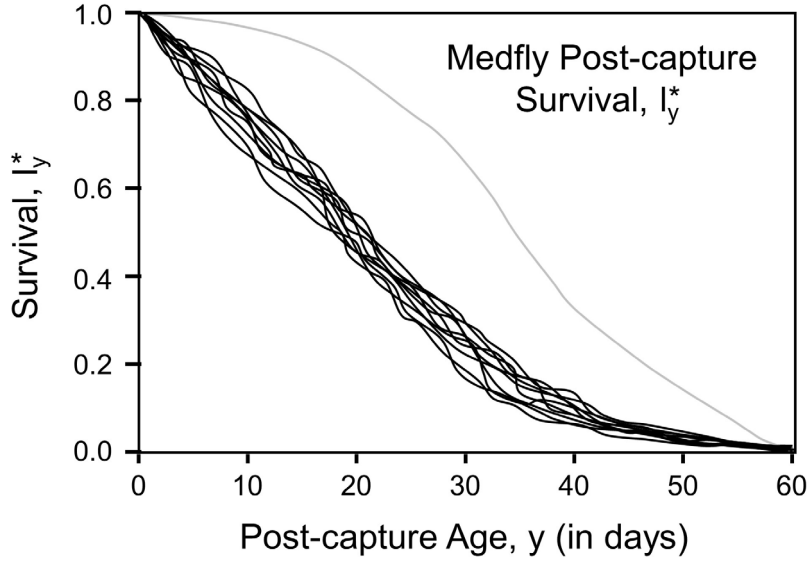

Fig. 2 Survival (l^*_y) curves generated from simulation studies consisting of 10 replicates of 100 medflies sampled at random ages and monitored through their remaining lifetimes (i.e. post-capture segments, see Fig. 1). The grey line is the age-specific survival schedule of a birth cohort where age is in chronological rather than post-capture time (denoted y). Medfly survival data used for simulation from Carey and co-workers (Carey et al., 1998).

Stage III: Construction of a Heuristic Life Table

In order to move from a field sampling concept to the development of an analytical framework, I decided to construct a simple heuristic pencil-and-paper life table model as an aid to understanding the relationship between the population as a whole and what I was then referring to as the *captive cohort* — the group of individuals of mixed ages that were survived forward from their age of capture through death. Ultimately this framework elucidated the connection between population age structure and the captive cohort death distribution.

Shown in Table 1, the life-table-based model consisted of sub-components framed as separate but interconnected sub-tables. The first of these sub-tables (columns 1–4 in Table 1) contains the basic metrics of the stationary population with age x in column 1, and the number in the population at each age (N_x) in column 2 (i.e. 40, 30, 25, 5 and 0 individuals at ages 0, 1, 2, 3, and 4, respectively). The corresponding survival l_x within this population and its age structure c_x are given in columns 3 and 4, respectively.

The framework for the second sub-component of the model in Table 1 (columns 5–9) was central to the eventual discovery of the population identity for three reasons. Firstly, it includes a new age index for the newly-formed group referred to as *captive age*, denoted y, defined as the time units from capture ($y = 0$) through death of the last individual in the sample cohort. Secondly, it revealed the initial age structure of the sample based on the assumption of random sampling. This is shown in the first row for columns 6 through 9, the fractions of which correspond to the proportion of the population sample for each of the respective ages. For example, I assumed that if 0.30 of the stationary population in the wild is in age class 1 then (on average) there will be 0.30 of the captive sample in this age class. Thirdly, these proportions are then subject to their respective age-specific survival rates. For example, the 0.30 individuals in age class 1 at captive age l_y* are reduced to 0.25, 0.05 and 0.00, respectively, for captive ages 1, 2 and 3, respectively. This sub-table helped me visualize the internal mortality dynamics of the population sample.

The concept for the third subcomponent of Table 1 (columns 10–11) was to bring life table methods to bear on the captive cohort. Inasmuch as the sum of the fractions surviving in each of the sample sub-cohorts at each captive age (y) represents the total of the original surviving, these sums represent the survival schedule of the captive cohort, l_y*. Although I found the survival column (column 10) interesting, it revealed nothing about the deeper mathematical connection between the population and the captive cohort. It was only when I computed the d_y*column did I experience a Eureka moment — the values for the death distribution (column 11) were exactly equal to the values in the age distributions (column 4).

In his fascinating paper on the role of serendipity in science, Yaqub (2018) would likely classify my discovery of this stationary population equality as a "Mertonian serendipity" — discovery as the outcome of a targeted search that solved a problem in hand (age-structure estimation) via an *unexpected route*. The *expected route* would have been through the use of Bayesian models.

Stage IV: Model Formulation

In a section titled "A key demographic identity" my statistical colleagues Hans Müller and Jane-Ling Wang formulated a mathematical model based on the framework and concepts that I developed as given in Table 1 (Müller et al., 2004). Assuming population stationarity in this hypothetical case, the fraction of individuals age x in the population is given by $c_x = l_x \sum l_y = c_0 l_x$. The death rates in the marked sample life table at captive age x' are by definition $d_y^* = l_y^* - d_{y+1}^*$. These death rates are generated by subjects that enter the marked sample life table at various unknown ages, and survive to captive age y. For all individuals that enter the marked sample cohort at age z, the contribution to d_y^* is therefore

$$c_z \frac{l_{z+1}}{l_z} \cdots \frac{l_{z+y}}{l_{z+y-1}} \left(1 - \frac{l_{z+x'+1}}{l_{z+y}}\right) = c_z \left(\frac{l_{z+x'}}{l_z} - \frac{l_{z+x'+1}}{l_z}\right) = c_0 (l_{z+y} - l_{z+y+1})$$

where l_z refers to the survival of the captured individuals at age z.

The contributions of individuals entering the marked sample life table at various ages are additive. Therefore, adding the contributions over all ages of entry z:

$$d_y^* = \sum c_0 (l_{z+y} - l_{z+y+1}) = c_0 / l_y$$

and therefore

$$d_y^* = \sum c_x$$

This formula states that the fraction of a population age x days equals the fraction of the population that die x days later. The shorthand for this equality is *age structure equals death distribution*. In his proof of this equality, Vaupel expressed the relationship differently — the proportion of the population with x years of life remaining is equal to the proportion dying x years in the future (Vaupel, 2009a; p. 8). The shorthand for this expression of the same equality is *life lived equals life left*.

Vaupel notes that since births equal deaths in a stationary population, Carey's Equality generalizes this relationship by showing that the proportion of individuals younger than x equals the proportion whose remaining lifespan is less than x. Similarly, the proportion of individuals x or older is equal to the proportion of individuals who will still be alive in x days (years).

Table 1 Illustration of the relationship between the age structure in a stationary population (columns 1–4), the captive (sample) cohort life (columns 5–9), and the captive cohort life table (columns 10–11). Framework from Table 1 in Hans Müller and others (Müller et al. 2004). The identity illustrated in this table as well as one for medfly stationary populations are visualized in Figs. 3–5 of the paper by Carey et al. (2019).

Wild Population				Captive Cohort (mixed chronological ages)						
				Post-capture Age (time since capture)	Population Sample at Capture				Captive Life Table	
Age	Number age x	Survival	Age structure		Age 0	Age 1	Age 2	Age 3	Survival to age y	Deaths at age y
x	N_x	l_x	c_x	y	$x=0$	$x=1$	$x=2$	$x=3$	l_y^*	d_y^*
(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)
0	40	1.000	0.40	0	40	30	25	5	1.00	0.40
1	30	0.750	0.30	1	30	25	5		0.60	0.30
2	25	0.625	0.25	2	25	5			0.30	0.25
3	5	0.125	0.05	3	5				0.05	0.05
4	0	0.000	0.00	4	0				0.00	0.00
	100		1.00		100	60	30	5		1.00

A Surprise Retrospective Story

Given the depth and scope of the demography literature in general (Poston and Micklin, 2005) and of the mathematical demography (Land, Yang, and Yi, 2005) and life table literature in particular (Guillot, 2005), I was interested to know whether this identity had already been published in some form. My online searches in the demography, statistics and mathematical biology literature revealed that there were no previous papers on this identity in either the primary literature or in the searchable secondary and grey literature. Later perusals of demography texts (Preston, Heuveline, and Guillot, 2001; Keyfitz and Caswell, 2010), handbooks (Poston and Micklin, 2005), methods books (Siegel and Swanson, 2004; Caswell, 2001), dictionaries (Peterson and Peterson, 1986), encyclopaedias (Demeny and McNicoll 2003a, 2003b) and treatises (Caselli, Vallin, and Wunsch, 2006f) also indicated that this result was not present in the earlier literature.

To my astonishment, three years ago I learned from Tim Riffe that he had discovered a 1986 paper describing an equality in stationary populations that was expressed differently than mine but similarly to Vaupel's *life-lived and left* version (Riffe, 2015). In his article, titled "*Structure et dynamique des populations. La pyramide des années à vivre, aspects nationaux et exemples régionaux*", French demographer Nicolas Brouard (1986; pp. 160–61) wrote:

> La population stationnaire a un intérêt pour le sujet qui nous préoccupe, car dans une population rigoureusement stationnaire la pyramide des âges est aussi égale à la pyramide des années à vivre. On montre [2] en effet que dans une population stationnaire, il y a autant d'individus ayant vécu x années que d'individus ayant x années à vivre.
>
> [The stationary population has an interest in the subject that concerns us, because in a strictly stationary population the age pyramid is also equal to the pyramid of the years to live. It is shown [2] that in a stationary population, there are as many individuals having lived x years as individuals having x years to live.]

Although this description of the identity differs from mine (i.e. age structure equal death distribution), it is virtually identical to the description presented by James Vaupel (i.e. life lived equals life left; Vaupel, 2009a). In correspondence with Brouard (Brouard, 2018) I learned that, in an exercise of the final examination (February 1984) at the Institut de Formation et Recherche Démographiques (IFORD), he asked the students to prove the identity in discrete time. Also, Brouard inserted the proof of the theorem in continuous time in the chapter entitled "Infinitesimal approach of mortality" of his mimeographed manual "Mouvements et modèles". I learned from Brouard that at that time not only were French demographers required to first publish their findings in French, but, interestingly, young researchers like him did not have ready access to IBM Selectric typewriter math typeballs — the state-of-technology in personal typesetting at the time. He thus implied in his correspondence that otherwise he would have published the book containing his identity "Mouvements et modèles" earlier. His book was not published until 1989 when the word-processing software LaTeX became widely available. However, its distribution was extremely limited because it was published as a training manual for IFORD in the African country of Cameroon. Brouard noted in his correspondence with me (Brouard, 2018):

> Others were not interested by the equality which [...] appeared not very useful per se [...] after the weak response of [more senior] French demographers to my articles I moved away from formal demography[.]

Brouard's story reveals how his limited access to typesetting technology, the restricted distribution of the publications, the necessity of publishing in French rather than in English, the perceived absence of any utility of the identity and the lack of interest by his more senior colleagues conspired to keep his discovery (Brouard's Theorem) hidden from mainstream demography for thirty years.

Discussion

The need to find a method to estimate age structure in insect populations was the driving force behind development of the methodological component of my research program on ageing in the wild in the late 1990s. One of the important outcomes of the quest was the

discovery of the identity that Vaupel referred to as Carey's Equality. Although this discovery and its connection with Brouard's earlier publication was important from the standpoint of basic demography, the identity itself could not be used to estimate age structure in actual wild fruit fly populations because: (1) like most field populations, fruit fly populations in the wild violate the assumption of stationarity; and (2) conditions flies experience pre-capture (field) are different than those they experience post-capture (laboratory). Nonetheless, the discovery of the identity was important, especially from a practical standpoint because it established the theoretical foundation for using the post-capture death distribution of individuals of unknown ages to estimate age structure in the populations from which they once belonged. More realistic models to estimate age structure in actual field populations of fruit flies were developed by my UC Davis statistical colleagues, Hans Müller and Jane-Ling Wang, and their students, based on the use of mortality data from reference fruit fly life tables and iterative mathematical methods (Müller et al., 2007; Müller et al., 2004). Example applications of these methods for estimating age structure in wild populations of insects along with their constraints are given in the papers by Carey, Papadopoulos and their colleagues (Carey et al., 2008; Carey et al., 2012; Papadopoulos et al., 2016).

Papers related either to this identity, similar concepts or stationary population theory more generally, which were published many years prior to the paper that described this identity (Müller et al., 2004), include the two papers by Brouard (Brouard, 1986, 1989), the classic papers by Ryder (1965, 1973, 1975) on replacement populations, the article by Kim and Aron (1989) showing the equivalency of the average age and average expectation of remaining life in a stationary population, the book section by Keyfitz (1985; p. 74) containing a general formula for the average expectation of life in a stationary population, and the demography text by Preston and his co-authors (Preston, Heuveline, and Guillot, 2001; pp. 53–58) outlining the basic properties of a stationary population. More recent papers connecting life lived to life left (or age structure to death distributions) include one by Goldstein (2009) proving the earlier finding of Kim and Aron (1989) but in a different way; a paper by Rao and Carey (2015) with original approach and new conceptual insights into Carey's Equality; symmetries between life lived and left in finite stationary populations (Villavicencio and Riffe 2016a); the relationship of random age and remaining lifetimes by Finkelstein and Vaupel (2014); and the paper by Vaupel and Villavicencio (Vaupel and Villavicencio, 2018) in which they extend stationary population identity to the stable, non-stationary case.

Although Brouard's Theorem and Carey's Equality both describe the same demographic identity, the contexts in which they were discovered and the concepts upon which they are based are fundamentally different. From my experience working with scientists in the professional worlds of both human (classical) demography and biodemography, the conceptual differences between the two flow naturally from these two worlds — the "life lived equals life left" perspective from demography, and the "age structure equals death distribution" perspective from biodemograpy. Indeed, until James Vaupel published the proof of the stationary population identity (Vaupel, 2009), I had never before thought of age structure (denoted c_x) as a "life lived" concept, or of the death distribution (denoted d_x) as a "life left" concept. Even though they are literally identical, they are neither semantically nor conceptually identical.

As a biodemographer working through each of the stages in the discovery process that I described earlier, my focus was on the potential use of life table theory as a basis for estimating

the age structure of a life table (stationary) population. I am reasonably certain that I would never have conceived of measuring 'life left' as a way to estimate 'life lived' instead of the use of the conventional and more transparent life table concepts in which the death distribution provides a means to estimate age structure.

I consider development of the general idea of using the death distribution of wild-caught fruit flies to estimate population age structure distribution to be the single most important concept that emerged from brainstorming sessions on 'residual demography' with my colleagues two decades ago. The 'accidental' discovery of the stationary population identity itself is personally gratifying for several reasons. These include bringing Nicolas Brouard's original contribution to light and thus providing him with due credit, contributing to the basic demographic literature so that the basic identity becomes part of the demography pedagogy (e.g., Wachter, 2014), and using it as the conceptual basis for the development of the captive cohort method as a new technique for estimating age structure in wild populations. These contributions all resulted from the original discussions with my colleagues in Crete when we asked the question situated within the inter-zone between biology and demography: what can residual demography tell us about ageing in the wild?

Implications for Human Population Studies

A career-altering decision I made in my first year as an assistant professor of entomology at the University of California Davis in 1980 was to audit Kenneth Wachter's course in introductory demography at the University of California, Berkeley. Although this required a half-day's commitment several days each week for three months (i.e. the Davis-Berkeley round trip), the experience exposed me to methods of formal demography that had been expressly developed for and applied to human populations, tied me into a network of professional demographers and motivated me to join several population associations (e.g. Population Association of America; International Union for the Scientific Study of Populations) and present papers at their meetings. It also gave me both the incentive and the confidence to, where possible, generalize my findings on non-human species in order to contribute to the mainstream demographic literature, particularly as they apply to human populations (e.g., Carey, 1997; Judge and Carey, 2000; Carey and Judge, 2001; Carey, 2003).

I believe the stationary population identity that I discovered in the context of medfly research has a number of important implications in both basic and applied contexts for human demography. I briefly describe several of these next.

Human Evolution

It is virtually certain that the population growth rates of various species of early hominids (e.g. *Australopithicus* spp.) in general and of prehistoric *Homo sapiens* in particular were stationary, or nearly so, the vast majority of the time (Johnson and Brook, 2011; Lee and Tuljapurkar, 2008; Boone, 2002). Indeed, it is estimated that during most of the Holocene, human populations worldwide grew at a long-term annual rate of 0.04% (Zahid, Robinson, and Kelly, 2016) which, for practical purposes, have age structures that are nearly indistinguishable from stationary populations.

Using the concept of the stationary population identity, life-table rates of prehistoric populations (Gage, 1998; Kaplan et al., 2000) imply that up to half of the population is 20 years

old or greater, and at least a quarter are over 30 years old. This implies (from the identity) that 50% and 25% of the prehistoric populations had respectively 20 and 30 years remaining to live together. These basic demographic metrics provide important perspectives on the extent to which individuals in prehistoric societies shared lives (and thus their skills, language, stories, art, music and culture).

Historical Demography

An interesting application of the population identity, similar to its original application in fruit fly demography, was offered by Villavicencio and Riffe (2016b), who suggested that the concept might be applied to incomplete historical data. In the Barcelona Historical Marriage Database — which collects information about marriage licenses of Barcelona (Spain) from the mid-fifteenth century until the early twentieth century — individuals are first identified in their marriage record and then followed up. But no information is available about their birth date or their age at marriage (Villavicencio, Jordà, and Pujadas-Mora, 2015). Interestingly they used a Bayesian statistical modelling approach (Colchero and Clark, 2012; Colchero, Jones, and Rebke, 2012) as was first suggested by me for estimating the post-capture lifespans of fruit flies. The alternative that Villavicencio and Riffe suggested was the possibility of using the life-table identity to estimate the pre-marriage ages of this historical population.

Demographic Principles

Inasmuch as stationary population theory and basic concepts are inextricably linked to population stationarity (Preston, Heuveline, and Guillot, 2001), the stationary population identity contributes to a number of fundamental principles upon which the field rests. For example, demographic transition theory is based on the concept of offsetting birth and death rates at the beginning and end of the transition (Mesle and Vallin, 2006); population momentum is based on the concept of stationarity as its end point (Caselli, Vallin, and Wunsch, 2006a); and zero population growth (ZPG) is, by definition, based on the concept of the cessation of population growth (Caselli, Vallin, and Wunsch, 2006a), and thus implies stationary populations. The stationary population identity can now be integrated into each of these basic demographic principles.

Replacement-level Populations in the Twenty-first Century

To study the world population today is to study many countries whose populations are essentially quasi-stationary. For example, the Population Division of the United Nations noted that in 2010–15 there were eighty-three countries with below-replacement fertility that accounted for nearly half (46%) of the world's population (UnitedNations, 2017). The vast majority of European countries are experiencing population growth rates within $^{+}$0.5% of replacement-level change. And, with nearly 20% of the world's population, China's population growth rate, currently at slightly over 0.5%, is not far from replacement-level and is predicted to fall to zero by mid-century. Extending Vaupel's example (using 2005 life table rates for the United States) to the quasi-stationary populations of the world shows that around 50% of hypothetical individuals are 41 years old or older. Since the population change in nearly half of the world's population is near replacement, this implies that around a quarter of the world's "life-table

populations" will be alive in 2060, a date approximately forty-one years from now that is often considered as being in the distant future.

Future World

One of the most fascinating contributions in the 143-chapter treatise in population studies (Caselli, Vallin, and Wunsch, 2006b, 2006c, 2006d, 2006e) is Chapter 78 by Vallin and Casalli entitled "The Future of Mankind" (Vallin and Caselli, 2006). In exploring one variant of the scenario of a "one-child, 150-year life expectancy" world, they note that up to 84% of the population would be made up of centenarians alone. Applying the stationary population identity concept suggests that more than eight out of ten individuals in this hypothetical futuristic population would share one hundred years of life together. This scenario illustrates an extreme case of Joel Cohen's concept of Methuselah's choice (Cohen, 1995) — the necessity of an extraordinarily low birth-rate (=1/150) in a stationary world of long-lived individuals.

Concluding Thoughts

I believe there are several lessons that can be learned from this story. The first is that the proverb "necessity is the mother of invention" can also be reframed as "… the mother of discovery". My quest to find a practical solution to the problem of population age structure estimation led to the discovery of the basic stationary population identity. A second lesson pertains to the virtues of pencil-and-paper modelling in science — it is both immediate and simple (Wong and Kjaergaard, 2012). This step forced me to think about the essence of the problem rather than becoming buried in its complexities. Simple is not the same as simplistic. A third lesson pertains to the discovery of new demographic principles. If the stationary population identity remained hidden for most of the 350-year history of the life table, there are almost certainly more undiscovered ones. I believe the most fertile ground for new demographic discoveries will be within the interdisciplinary paradigm of biodemography.

I will close with what I consider to be the wonderment of the discovery. How could this particular mathematical property of (arguably) the most studied of all demographic models — the life table — have remained hidden for so long? And once discovered, still not have found its way into the mainstream demographic literature for thirty more years? Why do all deaths that occur in a stationary fruit-fly population at twenty-five days post-capture, for example, sum exactly to the number that were precisely twenty-five days old in the original population? How is it possible to compute precisely the age structure of a stationary population without age data on even a single member? Think about that.

The concept appears not to make sense on its face. It is a discovery that *is not obviously true*. But once proven (Brouard, 1989; Vaupel, 2009b) becomes one that *obviously is true*.

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17. Human Mortality from Beginning to End: What Does Natural Selection Have to Do with It?

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Evolutionary demographers who study human traits usually focus solely on natural selection as a cause of a trait's evolution. However, demographic stochasticity, genetic drift and phylogenetic inertia can also significantly influence trait evolution. We describe why accounting for these influences is necessary in order to correctly test hypotheses about the adaptive nature of human demographic traits. For example, "U"-shaped mortality from the beginning to the end of life is found in many vertebrates, which implies that phylogeny must be considered in understanding the evolution of this trait in humans. Even when these other evolutionary influences have negligible effects on a human demographic trait, it is incorrect to assume that the observed trait must be optimal. Current data and analyses are not sufficient to properly confirm the claim that "U"-shaped mortality rate in humans is the result of natural selection in humans or that it is optimal. We describe the additional data and analyses that are needed in order to properly test these claims.

Human life can be hazardous. For example, it is likely that 60% or so of conceptions die before birth, with most deaths occurring in the first month of pregnancy (see Léridon 1977; Macklon et al. 2002; Boklage 2005; Orzack et al. 2015; Jarvis 2016; Orzack and Zuckerman 2017 and references therein). The mortality rate during pregnancy declines rapidly thereafter and less than 1% of fetuses alive at the beginning of the third trimester die before birth.

This steep pre-birth decline precedes the beginning of what many demographers refer to as the "U"-shaped trajectory of age-specific mortality rate from birth onward (Gompertz 1825; Makeham 1860; Heligman and Pollard 1980; Gage and Mode 1993; Carnes et al. 1996; Levitis and Martínez 2013). This "law of mortality" is taken by many to describe the age-specific pattern of mortality from birth onward in all or most human populations. It is thought to have these features:

1. A decline in age-specific mortality rate to a low value "soon" after birth. This decline is the left limb of the "U".
2. A low age-specific mortality rate (the bottom of the "U") that often lasts well into adulthood. In some populations, there is a mortality increase and decrease around the time of the transition from juvenile to adult.

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3. An increase in age-specific mortality rate in later life. For example, in all populations in the Human Mortality Database (https://www.mortality.org), the mortality rate of 70-year-olds is higher than that for 60-year-olds and lower than that for 80-year-olds. This increase is the right limb of the "U".

From here on, we refer to the entire trajectory from conception onward as the "U"-shaped trajectory of age-specific mortality rate.

What Are the Possible Causes of the "U"-shaped Trajectory?

We begin by noting that many analyses seek to explain only the right limb of the "U", i.e. the later-age increase in age-specific mortality rate. Some investigators have sought a physiological explanation (e.g. Rubner 1908; Pearl 1928). More recently, many investigators have sought an adaptive explanation, i.e. one invoking natural selection as a cause. At first glance, such attempts would seem ill-conceived if not foolish. After all, senescence and death are things one would expect *not* to evolve. Isn't survival the consequence of the process of evolution via natural selection? In fact, senescence and death can be two consequences of this process (Bidder 1932; Williams 1957; Hamilton 1966; Kirkwood and Holliday 1979; Kirkwood and Austad 2000). All of these explanations involve the distinction between current evolutionary "fitness" (as determined by survival and reproduction) and future evolutionary fitness, but their causal details differ. For example, Williams (1957) proposed that senescence occurs because a "pleiotropic" mutation, one decreasing mortality rate earlier in life and increasing it later in life, can be favored by natural selection (see also Abrams 1993; Williams et al. 2006; Gaillard and Lemaître 2017). The reason is that a decrease in early mortality results in a greater number of descendants than would result from a later increase of the same magnitude, just as an earlier contribution to a savings account "outweighs" a later contribution of the same magnitude because of interest accrued. In contrast, Medawar (1946) and Hamilton (1966) proposed that late-age deleterious mutations potentially compromise future reproduction less as compared to early-age mutations. Accordingly, the intensity of natural selection against deleterious mutations decreases with age and so the age-specific mortality rate increases. See Charlesworth (2000) for details.

Despite these causal differences, William's hypothesis and the Medawar/Hamilton hypothesis are *adaptive* explanations. Natural selection is assumed to be the *only* evolutionary force acting on the trait. Given the constraint of either pleiotropy or of deleterious mutations, the population is expected to evolve to the predicted mortality rate trajectory, which is locally optimal, i.e. it results in a higher fitness than the alternative trajectories delineated by the model (see below). It is incorrect to refer to the Medawar/Hamilton explanation as "non-adaptive" (cf., Dańko et al. 2012).

What Are We to Make of Such Adaptive Explanations?

There is a two-part answer to this question. The first part is that the adaptive explanations provide important insights. For example, they demonstrate the necessity of considering the temporal expression of influences on survival and reproduction. They also illustrate how natural selection can cause the evolution of a partially-deleterious trait. The second part of the answer is that neither explanation is as illuminating as claimed by its original proponents. For

example, consider the prediction of Hamilton's (1966) model that the age-specific mortality rate increases monotonically after the age of first reproduction, which underlies his famous claim (p. 12) that "….senescence is an inevitable outcome of evolution". One can distinguish between a "strong" form of Hamilton's prediction, which is that age-specific mortality rate increases monotonically, and a "weak" form of his prediction, which is that age-specific mortality rate eventually increases. Evidence *for* the strong prediction necessarily supports the weak prediction but not vice-versa. Evidence *against* the strong prediction does not necessarily refute the weak prediction but evidence against the weak prediction necessarily refutes the strong prediction.

What Evidence Do We Have About the Age-specific Mortality Rate?

Some of the diversity of the trajectories of the age-specific mortality rate is depicted in Figure 1 in Jones et al. (2014) (see also Vaupel et al. 2004; Cohen 2017; Jones and Vaupel 2017). For some species, the age-specific mortality rate increases monotonically, which supports the strong and weak predictions. For others, the mortality rate eventually increases after the age of first reproduction, which contradicts the strong prediction and supports the weak prediction. Other studies not compiled by Jones et al. also reveal this result. For example, Orzack et al. (2011) reported for a long-lived seabird that the age-specific mortality rate of reproductive individuals first decreases and then increases. (The age-specific probability of successful reproduction also decreases but then increases.)

Even when aggregated data from individuals of a species reveal a monotonically-increasing age-specific mortality rate, there can be individuals who live well past most others, thereby demonstrating that the length of an individual life can depend upon fixed and/or random differences among individuals (Tuljapurkar et al. 2009). This demonstrates that mortality need not be a unitary phenomenon within a species (see also Zuo et al. 2018). Such heterogeneity may indicate that there is no fixed length of life. Even if there is a fixed limit to lifespan, longer life may be accessible to most individuals. Determining the influences that govern extremes of the age-specific mortality rate trajectory is the goal of studies of human centenarians (Yashin et al. 2000; Andersen et al. 2012; Barbi et al. 2018) and of organisms that can live much longer than humans (de Magalhães et al. 2007; Keane et al. 2015).

Figure 1 of Jones et al. (2014) indicates that the age-specific mortality rate decreases at some time during life in twenty-four of forty-six species depicted. Of these, seventeen exhibit a decrease and an increase (inconsistent with the strong form of Hamilton's prediction but consistent with the weak form). Seven others reveal only a decrease. In addition, there are two species that appear to be "non-senescent", i.e. to have a constant age-specific mortality rate.

Do these nine species constitute evidence *against* the weak prediction (and therefore against the strong prediction)? If so, they might indicate that an increase of the mortality rate with age could be avoided altogether. These data suggest that the weak prediction is false but they are *not* sufficient to demonstrate that such an increase can be avoided. Why? One reason is that demonstrating the absence of an increase in the mortality rate means demonstrating that an effect does not exist. Accordingly, it is essential to assess whether a study could detect the presence of an effect. Typically, one does this by estimating the statistical power to detect a trend (see Petrascheck and Miller 2017). Such an estimate of the statistical power to detect a given (small) increase in the mortality rate is lacking for the nine species.

Another reason why these data do not falsify the weak prediction is that the absence (or presence) of an increase in the age-specific mortality rate is a claim about the entire lifetime. The implication of this can be illustrated by considering the analysis of the small aquatic invertebrate, *Hydra magnipapillata*, which is one of the two "nonsenescent" species depicted by Jones et al. The mortality rate trajectory they present is based upon the data collected by Schaible et al. (2015), who also present data consistent with a constant age-specific mortality rate for a second species, *H. vulgaris*. The latter species was previously studied by Martínez (1998) who also reported a "lack of senescence" of age-specific mortality rate. These important and well-done studies of laboratory cohorts (some tracked for up to eight years or so) reported constant age-specific mortality rates that are so small that they would imply, if maintained, that 5% of the individuals in a cohort would be alive after hundreds if not thousands of years (see Table 1 of Schaible et al. and also Figure 1 of Jones et al.). The constancy of the age-specific mortality rate is impressive given that an individual is just a few millimeters long.

However, these data do not underwrite the claim of Schaible et al. (2015, p. 15701) that *Hydra* has a "non-senescent life history" or the claim of Archer and Hosken (2016, p. R202) that *Hydra* "escapes senescence". The reason is seemingly contradictory: there are too few deaths. Doesn't this confirm the claim of non-senescence? No. The reason is that the weak prediction about the age-specific mortality rate is a prediction about the entire life. To this extent, *entire* lives (or nearly so) must be measured in order to make a claim that the age-specific mortality rate does *not* eventually increase. (The converse is not true in that one could base a claim *for* such an increase on a small interval of the lifetime.) What *would* support the claim for no eventual increase is a *high* constant age-specific mortality rate, such that we observe the death of all or most all individuals. Instead, in the case of *Hydra*, we have a "censored" data set (because of the low constant mortality rate) and it is the censored portion of the lives (the end) that is needed to support a claim as to the absence of an increase in the age-specific mortality rate. In contrast, assessments of the age-specific mortality rate in, say, humans are not censored in the sense described here because they are based on observations of completed lives. If human data were censored so as to include mostly uncompleted lives, one could also infer that humans have a 5% chance of living for hundreds of years. For example, the National Center for Health Statistics (2018) report (p. 52) that the annual mortality rate in the United States for children less than one year old was 0.0059 in 2015. If this rate were to remain constant as the cohort gets older, it implies that approximately 5% of the cohort would be alive at age 500.

We also note that current analyses do not support the claims by Martínez (1998, p. 217) that there is no "[age-specific] decline in reproductive rates" and by Schaible et al. (2015, p. 15701) that there are "constant age-specific…reproduction rates". One reason is the censoring mentioned above; most lives are uncompleted. Even given such censoring, the data suggest either a decline in age-specific reproductive rate (as later acknowledged by Martínez, see his Figure 2 and p. 220) or an increase that is sometimes followed by a decrease for some cohorts (see Schaible et al. Figure 3). Their claim (p. 15703) that the age-specific reproductive rate "eventually reached a cohort-specific constant level" conflicts with visual impression of a lack of constancy and the statistical basis for the claim about eventual constancy is not presented (see also Estep 2010).

Despite ambiguity about the eventual trend of age-specific mortality rate, the Martinez and Schaible et al. data do not support the strong prediction that the rate increases monotonically.

This is weaker than a conclusion that there is no increase (and no senescence) but it is important. There are many possible reasons for the discrepancies between observed trajectories of the age-specific mortality rate and those predicted by the adaptive models outlined above. For example, the evolution of age-specific mortality rate could be influenced by mutations and natural selection in ways not assumed by Hamilton (Cichoń 2001; Baudisch 2005; Dańko et al. 2012).

What Should We Conclude About the Causes of the "U"-shaped Mortality Rate Trajectory in Humans?

We start by recalling that what needs to be explained is the *entire* "U"-shaped trajectory of mortality rate. The mortality rate is likely high just after conception but declines rapidly during pregnancy. After birth, it is relatively low and possibly constant for several decades. It then increases rapidly for several decades and becomes high. The age-specific mortality rate may even become constant but high at very old ages (Barbi et al. 2018; Newman 2018a, b; Wachter 2018). Do these "phases" of mortality each have a different evolutionary explanation, or are they best understood as having one explanation? The answer to this question is unknown. The separation of the pre-birth and post-birth trajectories arose mainly because the former trajectory was poorly characterized until recently. It still remains much less well characterized than the latter trajectory. Hence, the separation of the two arose because of a lack of data, instead of from empirical results indicating that the two trajectories must have distinct evolutionary causes. However, some investigators do believe that these trajectories need different evolutionary explanations (e.g., Medawar 1952; Hamilton 1966).

Most research has focused on only one part of the post-birth trajectory: the increase in age-specific mortality rate later in life. This is when most people die and so there are huge amounts of data available (e.g., Oeppen and Vaupel 2002; Colchero et al. 2016). Abundant data attracts investigators. In contrast, much of early pre-birth mortality is hidden from view and relatively few people attain the age of 100.

The evolutionary process described by Hamilton (1966) does not predict a downward age-trend in pre-reproductive mortality. To explain this discrepancy, Hamilton posited that it is selectively advantageous for parents to eliminate likely-inviable offspring as early as possible so that the saved energy can be invested into later likely-viable offspring. He may be correct to assume that pre-birth mortality and later mortality require different adaptive explanations. In order to assess whether his explanation is correct, it is important to reconcile the apparent contradiction between the high level of pre-reproductive mortality and the assumption that natural selection is powerful enough to result in an optimal age-specific mortality rate trajectory. All other things being equal, if natural selection is this powerful, it should also be powerful enough that all offspring be viable until they reproduce or at least that mortality of offspring occur immediately after conception so that minimal energy is wasted.

Hamilton's model also predicts death at the end of reproduction because survival thereafter cannot increase offspring number and thereby increase evolutionary fitness. Post-reproductive survival is anecdotally observed in many but not all animal species. There are conflicting claims about its frequency and extent in natural populations because of methodological and empirical challenges (Cohen 2004; Reznick et al. 2006; Levitis et al. 2013; Croft et al. 2015; Lemaître and Gaillard 2017; Ellis et al. 2018; Johnstone and Cant 2019). Sometimes even determining the end of reproduction is difficult. However, one can distinguish between the many species in

which a minority of individuals live past the end of reproduction and the many fewer species in which many individuals survive well past the end of reproduction. Both patterns contradict Hamilton's prediction but the evolutionary implications of the contradictions differ. The first pattern suggests that natural selection acted in the way Hamilton posited but that it is not the sole important influence on the mortality rate trajectory. In contrast, the second pattern suggests that the selective process he described does not capture an important aspect of the evolution of the mortality rate trajectory. Hamilton (p. 37) argued (following Williams 1957) that the second pattern ("typical" post-reproductive survival) is an adaptation that evolved so that a mother avoids the hazards of further reproduction and is thereby alive to provide care, energy and knowledge to her extant offspring. This would imply that there has been natural selection to end reproduction before the expected end of life. Another possibility is that post-reproductive individuals survive because they provide such resources to grand-offspring. This would imply that there has been natural selection to extend survival past the expected end of reproduction. These hypotheses involve the inter-generational transfer of resources, the potential evolutionary influences of which have been analyzed theoretically (Lee 2003, 2008; Chu and Lee 2006; Chu et al. 2008; Gurven et al. 2012). As explored elsewhere in this volume, current data for humans better support the hypothesis that post-reproductive survival of human females is due in part to the evolutionary advantage of transfers of resources from grandmothers to grand-offspring (Hawkes and Blurton Jones 2005; Hawkes 2010; Lahdenperä et al. 2011; Chapman et al. 2019).

Whatever the ultimate explanation of post-reproductive survival in humans proves to be, it is clear that Hamilton's 1966 model does not provide a complete causal explanation of the entire "U"-shaped mortality rate trajectory in our species. The weak prediction that age-specific mortality rate eventually increases is correct. However, the strong prediction arising from his model is incorrect. As Hamilton acknowledged, his model also does not provide an explanation for the pre-birth decline of age-specific mortality rate or for post-reproductive survival. In addition, it does not predict a possible late-age transition between increasing and constant age-specific mortality rates.

How Can We Develop Better Causal Understanding of Mortality Rate Evolution?

In order to answer this question, we describe the causal scheme contained in Hamilton's and Williams' models. They assumed that the trajectory of age-specific mortality rate is optimal, that is, it has evolved because it results in a greater number of descendants as compared to plausible alternatives. In addition, those investigators who have focused specifically on the human-mortality-rate trajectory have assumed it to be the result of natural selection acting either in our species or in our recent ancestors. There are two reasons why these assumptions can lead to incorrect understanding:

The first reason is that it privileges natural selection as a causal explanation. Since Darwin and Wallace's discovery of natural selection in the nineteenth century, evolutionary biologists have debated a variety of claims about the influence of natural selection on trait evolution as compared to the influence of other processes. Two such processes, genetic drift and demographic stochasticity (Parsons et al. 2010; Der et al. 2011), cause traits to evolve without the influence of natural selection. In all real populations, only a finite number of individuals reproduce and the number of offspring each produces is finite. Accordingly, the distribution of

traits among the parents is different from the distribution among their offspring. Genetic drift and demographic stochasticity necessarily influence "micro-evolution", the process of short-term evolution within species, to a small or large extent. They can also contribute to the process of long-term evolution (Wright 1931, 1932, Kimura 1968, 1983; King and Jukes 1969).

Another potential influence on a current trait is evolution in a past environment (Felsenstein 1985; Orzack and Sober 2001; Hansen and Orzack 2005). The influence of this "phylogenetic inertia" has been studied extensively in recent decades, with an important focus being how to control for it when testing hypotheses about adaptation in the current environment. If a trait is present in, say, two related species or populations, it is conceivable that it evolved once in a common ancestor, instead of evolving twice independently. Phylogenetic inertia and current natural selection can jointly contribute to a trait's evolution (Orzack and Sober 2001; Hansen and Orzack 2005). These considerations underscore the need for assessment of the nature of *evidence* about natural selection in the past and current environments. Methods for doing so are reviewed in Hansen et al. (2008) and O'Meara (2012). Ignoring common ancestry among species or populations can falsely increase the apparent amount of independent data one has to test an adaptive hypothesis.

For example, Wilson (2005) claimed that religious belief is a group adaptation because it increases cooperation and reduces exploitation among adherents. He based this conclusion on his assessment of data on thirty-five religions. Each is assumed to provide independent evidence. Each religion has distinctive features (see pp. 426–27) but it is unclear that they each provide independent evidence for (or against) his hypothesis. For example, the religions listed include "Tibetan Buddhism, tenth century", "Tibetan Buddhism, general", and "Tibetan Buddhism, fifteenth century" (not to mention various forms of Buddhism in India, Japan and Korea). The Tibetan forms are not identical. But this does not mean that whatever group benefit each may provide arose independently within each group. If instead, a group benefit arose in the "ancestral" form of Buddhism that gave rise to these three religions, they provide just one independent piece of evidence for the hypothesis. It is even possible that a group benefit to religious belief arose in a religion ancestral to *all* of these religions. If so, there would be only one possible evolutionary event in the sample, instead of thirty-five. An analysis in which the potential dependencies among the data are accounted for is required to assess whether the group-benefit hypothesis is true.

Such an accounting is also necessary in the context of assessing hypotheses about the evolution of the human-mortality-rate trajectory. A "U"-shaped mortality trajectory occurs in a variety of mammals, including other primates (e.g. Caughley 1966; Gage 1998, and references therein). Barring evidence that the most recent species from which *Homo sapiens* evolved did not have a "U"-shaped mortality trajectory, we must account for the possibility that humans have this trait because it evolved in our ancestral lineage prior to the evolution of our species. It may or may not have been adaptive when it evolved. If it were adaptive, it may or may not have been optimal. It cannot be taken to be self-evident that the "U"-shaped mortality trajectory in humans is adaptive (much less optimal) either in the current environment or in the environment inhabited by *Homo sapiens* prior to the "modern" environment of the last few thousand years (see below).

The second reason why a focus on optimality and natural selection can lead to an incorrect understanding is that optimality does not have necessary priority as an explanation for any

trait even if natural selection is an important influence on its evolution. This is true even for mortality and reproduction, which are the "stuff" of evolutionary fitness. These traits and others defined with respect to numbers of individuals can evolve via neutral evolution, i.e. in the absence of natural selection (e.g. see Kolman 1960; Poethke 1988; Orzack and Tuljapurkar 1989; Orzack and Hines 2005; Proulx and Adler 2010). In addition, natural selection need not cause even the average trait in the population to evolve to match the optimal trait, much less cause the trait of an individual to be the optimal trait (see Birch 2016 and references therein). One possible reason is that the optimal trait does not breed true.

The common focus in human evolutionary demography on optimality appears to be in part due to the use by practitioners of the common assumption in economics that individuals (or businesses) possess optimal consumption and production behaviors (see examples and discussion in Friedman 1953; Winter 1964; Ursprung 1988; Schoemaker 1991; Hodgson 1994; Rogers 1994). Other concepts in economics that may provide insights to evolutionary biology and evolutionary demography (e.g., Ward 1992; Nonacs and Dill 1993; Hammerstein and Hagen 2005; Bendor et al. 2009) have been much less used by biologists and demographers. Why this is so is unclear; see Samuelson (1985).

A Claim that the Age-specific Mortality Rate Trajectory in Humans is Optimal

Chu et al. (2008) derived a model that predicts that the optimal age-specific mortality rate trajectory from birth onward (not from conception) is "U"-shaped. Their important model predicts that the age-specific mortality rate declines after birth because the selective advantage of a reduction in mortality increases with age. This increase occurs because the amount of energy invested in the offspring increases with age. Their model also predicts that individuals survive past the end of reproduction because surviving individuals can still transfer resources such as knowledge and resources to offspring. The authors state correctly (p. 171) that "Age-specific mortality is U-shaped for many species…". Their title, *Explaining the Optimality of U-Shaped Age-Specific Mortality*, reflects the authors' beliefs that 1) these observed U-shaped trajectories are optimal and 2) that the apparent qualitative match of the shape of observed trajectories and the U-shaped trajectory predicted by the optimality model reveals *why* it is optimal. The notion appears to be that the model correctly represents the biology that has led to the evolution of an optimal trajectory.

What should we make of Chu et al.'s claim about the "U"-shaped mortality rate trajectory in humans? Their claim that the trajectory is optimal could be true. However, this is a conclusion that needs to be substantiated by evidence; it cannot be assumed to be true or even likely true.

In order to illustrate the analyses needed to assess the adaptive significance of the trait, we focus on the high pre-birth mortality (although Chu et al.'s model does not strictly apply to this period of development). The analyses that we use to assess this hypothesis are similar to those needed to assess the rest of the mortality rate trajectory.

As noted above, perhaps up to 60% of conceptions die within the first month or two of pregnancy. The proximate cause of much of this mortality is thought to be aneuploidy (the absence of one of two copies of a chromosome or the presence of an extra copy) caused mostly by errors during meiosis, the process by which haploid gametes are produced, see Guerneri et al. 1987; Hassold and Hunt 2001; Plachot 2001; Menasha et al. 2005). Most aneuploidies appear to

be fatal because the genetic information needed for normal development is unbalanced (Torres et al. 2008). The high frequency of aneuploidies in human ova has often been attributed to the long duration of female meiosis (Shuttleworth 1909; Jenkins 1933; Penrose 1933, 1934). The production of an ovum begins before a woman is born and pauses until sexual maturation (De Felici et al. 2005). After that time, usually a single ovum matures each month until menopause occurs. Accordingly, at least ten years and as many as fifty or so years could elapse between the time an ovum's precursor cell arises and the time the ovum is mature. It is possible that the length of this process is a cause of aneuploidy. There could be other causes (Brook et al. 1984; Nagaoka et al. 2012). For example, the incidence of aneuploidy of chromosome 21 among newborns appears to decline with maternal age before it increases (Erickson 1978) suggesting that hormonal imbalance may be an influence.

The production of a single mature ovum each month implies that at most 600 or so of the hundreds of thousands of primary oocytes in a woman's ovaries become fertilizable. The consequence of this sampling process is stochastic variation in the frequencies of genetic variants (generated by mutation and by the process of genetic recombination during development of the oocyte; see Hou et al. 2013). This sampling is expected to result in the loss of rare mutations (Ewens 2012). The mutations lost could include those that change the mortality rate trajectory in such a way that it results in higher fitness, as even advantageous mutations are most likely lost due to genetic drift. A quantitative calculation might reveal that the influence of natural selection and the influence of genetic drift are comparable in magnitude. This would imply that the trajectory is relatively immutable across species and that the potential for adaptive evolution is reduced. This reinforces the need for the investigator to provide evidence for the influence of natural selection on the trajectory. In the end, consideration of other evolutionary influences may not alter our conclusions about the power of natural selection. However, whatever the outcome, this kind of analysis is essential.

We next consider the influence of phylogenetic inertia on pre-birth mortality in humans. There is evidence that some degree of fetal wastage is widespread among vertebrates, invertebrates, and even plants and fungi (Levitis 2011; Levitis et al. 2017). It occurs in a variety of placental mammals (Brambell 1942, 1948; Casida 1953) including primates (Turner et al. 1987; Harley 1988; Palombit 1995; Knapp et al. 1996; Takeshita et al. 2016).

This suggests that an evolutionary explanation that relies solely upon human-specific biology may be incorrect. To resolve this, we need better comparative data on the amount of fetal wastage in at minimum our closer primate relatives, including chimpanzees, gorillas, and orangutans. If there is a significant difference in the amount in fetal wastage in humans as compared to the amount in these and other primates, it is circumstantial evidence that the amount in humans has evolved after our lineage split from those leading to these other species. We can then look to specific aspects of human biology in order to explain this difference. There is some evidence that the amount of fetal wastage in humans is higher than that of other primates (Corner and Bartelmez 1953) but we lack adequate comparative data and this is an unresolved issue. This analysis again illustrates how consideration of other evolutionary influences can alter our conclusions about the power of natural selection.

How Does One Test the Hypothesis of Optimality?

Let's imagine that our "U"-shaped age-specific mortality rate trajectory has in fact evolved entirely via natural selection in our species. Proving optimality requires evidence that it results in higher fitness than plausible alternatives. Would it be reasonable to conclude from the qualitative match between the "U"-shapes of the observed and optimal trajectories that Chu et al.'s claim of optimality is optimal?

The notion of optimality embodies the Darwinian idea that natural selection occurs when the trait of *an* individual outperforms other traits that an individual might possess. All other things being equal, this superior performance by the individual implies that the population should evolve to consist entirely (or nearly so) of individuals with that trait. Accordingly, assessment of whether a trait is optimal *requires* assessment of whether individuals are identical in the trait they express; this need not mean that individuals are identical at any given time, as the comparison is made over the entire time over which trait expression influences fitness. Assessment of optimality also *requires* a quantitative test of the optimality model's prediction. Orzack and Sober (1994a, b) describe why these analyses are necessary to support a claim of optimality.

In order to assess whether the observed mortality rate trajectory is optimal, we must determine whether individuals differ with respect to the mortality trajectory they would express if each had multiple lives to live. If such data were available for a reasonably large set of randomly-chosen individuals, one could compare the trajectories with a log-rank test (Harrington and Fleming 1982). A significant test statistic would suggest that there are differences among individual trajectories and imply that natural selection has not been powerful enough to cause the optimal trait to be fixed in the population. If there is no evidence for such heterogeneity (as determined by standard statistical criteria, such as a change in the Akaike Information Criterion), the observed and optimal distribution of lifespans can be compared quantitatively, say, with a goodness-of-fit test. If there is no evidence for a discrepancy between them (as determined by standard interpretation of the observed test statistic), one can conclude that current evidence supports the claim that the age-specific mortality rate trajectory is optimal (as compared to the non-optimal alternatives delineated in the optimality model). Orzack and Sober (1994a, b) explain how various combinations of qualitative and quantitative test outcomes support different inferences about the power of natural selection to influence a trait's evolution.

Of course, any heterogeneity among individuals in a real population with respect to their potential mortality rate trajectories is unobserved because each individual dies once. The observed age-specific mortality rate trajectory is aggregated over individuals and so it can by itself *never* underwrite a claim for optimality at the level of the individual. Accordingly, by itself even a quantitative match of the observed and optimal trajectories underwrites at most the claim that natural selection has had an important influence on the evolution of the mortality trajectory. This is not a trivial accomplishment but it leaves unresolved whether the trajectory is optimal. In contrast, a discrepancy between the observed and optimal trajectories can underwrite a claim *against* optimality.

The Importance of Understanding Evolutionary Causation

Any endeavor to assess the influence of natural selection must attend to consistency between causation in the observed biology and assumed causation in the adaptive model being investigated. This imperative can be illustrated by considering the cause of the pre-birth mortality in humans. As noted above, the rate of this mortality appears to decrease dramatically during pregnancy. Could this trend be explained by extrapolation of the causal framework in Chu et al.'s (2008) model? Much of the earliest mortality is likely the death of embryos and fetuses that are incapable of normal development and could not eventually reproduce if they did not die. Accordingly, the mortality rate declines because after these deaths occur most but not all of the remaining individuals are capable of normal development and most will be born alive. In contrast, in Chu et al.'s model, the decline of early mortality after birth arises from the increase in the selective advantage to a parent of protecting energetic investment in current offspring. This benefit increases as the offspring gets older. This explanation for the post-birth attenuation of the mortality rate cannot provide a causal account for most of the pre-birth decline of the "U"-shaped trajectory because the latter arises from the elimination of inviable offspring. Energetic investment in the inviable offspring is not being protected. We emphasize that Chu et al. make no claim that their model explains pre-birth mortality in humans.

At present we lack an adaptive explanation of the age-specific mortality-rate trajectory from conception onward. Such an explanation must account for the apparently distinct causes of the pre-birth and post-birth declines in the trajectory. It has been claimed that a mother can "suppress" offspring if the present environment is less suitable for those offspring than is the future environment for future offspring (Wasser and Barash 1983; Wasser and Isenberg 1986). Proponents assert that this explanation is in keeping with the adaptive explanation for such mortality in other mammals. There is evidence that pregnancy failure is associated with stress (see Table 1 in Wasser and Isenberg 1986), but this is not sufficient by itself to demonstrate that failure is adaptive, much less optimal. Further assessment of this claim will require analysis of an optimality model that includes the indirect transfer of energy from current offspring (by their termination) to future offspring as well as the direct transfer of energy from parents to current offspring.

Going Forward in Human Evolutionary Demography

The important distinctions between the hypotheses that natural selection has had some influence or an important influence on a trait and the hypothesis that a trait is optimal usually go ignored by evolutionary demographers. The consequence has been inferential ambiguity about the power of natural selection to influence trait evolution. A resulting danger is that investigators may make contradictory conclusions about the occurrence of optimality given the same data in part because they use unspecified "private" criteria in their judgment of optimality (see examples from evolutionary biology in Orzack 2014).

Human evolutionary demographers would do well to avoid such inferential ambiguity by exercising care when testing hypotheses about the realized influence of natural selection on trait evolution. Human evolutionary demography will become a more meaningful endeavor if two changes occur. One is the adoption of higher standards for the evaluation and testing of hypotheses about optimality and adaptation, which depends in part on having data on current

trait function and on the history of trait evolution. Human evolutionary demographers do understand the potential of the latter influence on trait evolution in a narrow sense in as much as they often invoke the action of past natural selection. For example, Robson and Kaplan (2003, p. 150) claim that long human life expectancy (and high intelligence) evolved in response to life in the "hunter-gatherer societies that prevailed for the two million years of human history". Similarly, Kaplan and Lancaster (2003, p. 179) claim that human patterns of fertility, mating and parental investment are a "constellation" of traits that "derives from the hunter-gatherer way of life, which characterized the vast majority of human evolutionary history" and Volk and Atkinson (2013, p. 182) claim to "generate a reliable estimate of [infant mortality and child mortality] levels in the EEA". The EEA or "environment of evolutionary adaptedness" was defined by Bowlby (1969, p. 58) as

> [...] the environment in terms of which the adaptedness of man's instinctive equipment must be considered [...] [it] is the one that man inhabited for two million years until changes of the past few thousand years led to the extraordinary variety of habitats he occupies today.

Our point is not to agree or disagree with the specific claims made above. Instead, our point is that human evolutionary demographers already traffic in the notion that history matters. The notion is that only *human* history matters (although sometimes this is extended to include some of our nearest primate relatives, such as the chimpanzee). This may be true but it is not self-evidently true, despite how special the traits possessed by humans are (see also Irons 1998). Either way, this acknowledgement of the potential influence of past natural selection on current human demographic traits illustrates that the path forward towards improved practice in human evolutionary demography can be rooted in part on current conceptual understanding. When invoking the action of past natural selection, human evolutionary demographers need only extend the potentially relevant history of trait evolution to include other primates and probably other vertebrates.

The second change needed in order that human evolutionary demography continues to make progress is for practitioners to understand the potential of forces *other than natural selection* to influence trait evolution. Such an understanding has been instrumental in allowing evolutionary biologists to better understand the evolution of a myriad variety of non-demographic traits (e.g. Wright 1932; Lande 1976; Kimura 1983; Hartl et al. 1985; Lynch and Hill 1986; Lynch 1990; Proulx and Adler 2010; Koonin 2016; Šustar and Brzović 2016; Charlesworth and Charlesworth 2018) and demographic traits (e.g. Tuljapurkar et al. 2009; Steiner et al. 2010; Orzack et al. 2011; Steiner and Tuljapurkar 2012) in many organisms. Just as evolutionary biology needs demography in order to achieve its explanatory potential (cf. Metcalf and Pavard 2007), so too does human evolutionary demography need evolutionary biology.

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SECTION 6: EVOLUTION OF THE HUMAN LIFE CYCLE

The related explanatory challenges of a long human lifespan and the long duration of post-reproductive life have received extensive attention from demographers and anthropologists, evolutionary or otherwise. A key difference between non-evolutionary and evolutionary approaches is in the framing of the question, and, at times, the nature of the analysis. The emphasis on evolutionary tradeoffs anchors the problem in models that consider energy budgets and allocation across the life course; for example, how late life mortality is linked to slow growth rates during childhood or fertility at younger ages. In the classic approach, at each age any organism has some amount of energy to expend, acquired either through its own efforts or given to it by others. This energy is then allocated among different goals: reproduction, survival, maintenance and growth. Life history theory often asks how can energy be allocated over the life cycle to maximize fitness? For lots of organisms, including all mammals and birds and many others, this optimal pattern would be to start the life cycle with allocation to growth and survival with no reproduction, and then at some age of sexual maturity to cease investing in growth and begin allocating that energy to reproduction.

In this section we have four chapters addressing the human life cycle.

The first, by Lee and Boe, analyzes the way hunter-gatherer social arrangements support the human life history strategy, and then in turn influence the way human life histories evolve. Humans invest heavily in each child, and hunter-gatherer children remain nutritionally dependent until age 18 or 20. This life history strategy has brought phenomenal success to humans, but also important problems and risks. With multiple dependent children, the death of the mother would cause a catastrophic fitness loss. Even when both parents survive, the growing number of dependent children over the family life cycle results in considerable energetic needs of the family, costs that would be both difficult and dangerous for a couple to bear, and one impossible for a mother alone. Human sociality solves these problems. Humans share food within groups and as adults make net transfers to the young throughout their lives and even well into old age. On the one hand, this provides life insurance, so that if the mother or father dies others may help feed the children. On the other hand, the rising dependency burden of children over the family life cycle is diluted and shared by contributions from other kin and non-kin. These social arrangements, in turn, influence the way natural selection shapes fertility and mortality over the life cycle.

Emery Thompson and Sabbi use their expertise in evolutionary anthropology and primatology to give us one of the most complete, and fascinating, overviews of Great Ape (which includes humans) demography that has ever been published, showing where humans fit in the context of our closest living relatives (see also the phylogeny chapter by Jones et al in

the Tinbergen section). The authors consider a wide range of social and demographic patterns in the comparison and find the degree to which variation among the apes has been explained by constraints of size and energy budget may have been over-estimated. They also give us innumerable empirical findings grounded in the best datasets available, such as Mountain Gorillas actually having a "faster" pace of life (with early reproduction and rapid reproductive paces) than previously thought and the Orang-utans perhaps the "slowest" (with late ages at sexual maturity and long birth intervals, up to eight years, likely linked to their high survival rates). But their chapter also highlights the relative dearth of data available on the demography of non-human great apes, perhaps reminding human demographers of the unique challenges faced in other parts of the discipline.

In an impressive analysis by leaders in the field of historical demography who often employ evolutionary concepts, Dillon et al. address the problem of post-reproductive survival in humans using detailed and nuanced analysis of multiple historical datasets (using some of the statistical methods advocated by Wilführ et al., see section on Family and Culture). Their thorough analysis, across four generations, supports previous findings that suggest that paternal grandparents are more often associated with higher fertility than maternal grandparents, while their data also allows them to explore interesting effects of maternal and paternal grandparent that vary by distance from the focal family, region and strength. Such work is a very nice illustration of the strengths of an evolutionary demographic approach, combining a data-rich descriptive approach common in demography with hypotheses informed by evolutionary theory. Such analyses lay the foundation for more detailed theoretical work, which is needed to explain why associations between kin availability and fertility differ by lineage.

Tuljapurkar, a leading theorist in evolutionary demography who has contributed to many areas of research, succinctly points the field toward some under-appreciated mechanisms that will be essential for fully understanding the evolution of menopause in humans. We have already heard about antagonistic pleiotropy (see chapter by Wachter), where genes may be selected for that have positive effects at early ages but negative effects at later ages (because individuals with such genes live long enough to pass them on, and thus there is no selection against the late-age negative effect). Tuljapurkar identifies the inability of antagonistic pleiotropy to account for aging patterns in humans and gives a concise overview of ideas that need much more attention in the field, such as genes that may have positive effects early and late in life.

18. Sociality, Food Sharing, and the Evolution of Life Histories

Ronald Lee and Carl Boe

Life history theory has focused on the life cycle trade-offs faced by individuals who are constrained by the energy they can forage for themselves at each age. However, humans are deeply social and adults transfer food to children for many years, freeing them from this energy constraint but also bringing the risk that parental death could entail the death of all dependent offspring. Multiple simultaneously dependent offspring also bring a family life-cycle squeeze in which dependency ratio doubles. Food sharing and alloparenting ameliorate both problems, providing life insurance and smoothing the life-cycle squeeze, while permitting humans to rely on food resources that would be too uncertain for isolated individuals. Food sharing and intergenerational transfers in turn affect the way natural selection shapes life histories. We use microsimulations to study evolution of life histories. Births inherit the mother's genome subject to mutations. Individuals live under different social arrangements and forage with productivity depending on population density. Natural selection on life histories occurs. We examine the way the size and relatedness of sharing group arrangements alter the evolution of life history traits through mutation and natural selection. We consider which social arrangements, with their corresponding evolved life histories, are most successful in a group competition where all face the same density constraint. There is a trade-off between costs and benefits of sharing. We find that intermediate levels are most successful, unless childhood conditions strongly influence later life productivity.

Introduction

The life histories of some species might be understood through the constraints and opportunities they face as isolated individuals. In life history theory, an individual starting life allocates whatever energy it can acquire among the competing goals of survival, growth, and reproduction (Stearns, 1992; Urlacher et al, 2018, estimate the survival-growth trade-off for a group of Amazon Basin forager/horticulturalists). Each allocation strategy entails some level of reproductive fitness. The strategy generating the highest fitness would tend to evolve through natural selection. Under some simplifying assumptions, mathematical models find that the optimal strategy invests first in growth and survival, and then at some "age of maturity" switches to investment in reproduction and survival. This life history pattern is called "determinate growth", and it is approximately the strategy of mammals and birds, but generally not of fish and reptiles, or plants.

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What about humans? In one sense, the determinate growth story fits us well. But in other ways it misses most of what is unusual and important about our life history strategy. Humans' life histories are deeply enmeshed in familial relationships and in broader social relationships as well (MacDonald and MacDonald, 2010). Like all mammals, a newborn human does not at first forage for its own food, but rather receives it as an intergenerational transfer, in the form of maternal lactation. This enables the offspring to invest much more in its growth and survival, beyond the limits of any meagre food it could have acquired on its own. The intergenerational transfer of food relaxes the constraints that limit the individualistic life history strategy and open up possibilities for slower and longer growth and development. The calorically hungry human brain (Kuzawa et al, 2014) is possible only through such intergenerational transfers (Lancaster et al, 2000; Kaplan and Robson, 2002).

But human parental investments continue for a very long time after lactation ends. In the Ache, Piro and Machiguenga (Kaplan, 1994, Lee, 2000), the Tsimane (Hooper et al, 2015) and the !Kung (Howell, 2010), the average child does not begin to produce the number of calories it consumes until age 18 or 20.[1] The net cost of raising one surviving child from birth to age 20 is around ten years of average consumption (average consumption over ages 0–50; Lee et al, 2002). Since average birth intervals were three or four years, a woman would often have multiple dependent children while her foraging productivity was limited by the energetic costs of lactating and/or carrying young offspring. She could not possibly manage this on her own and required and received help from others beyond her mate, such as single males (Hill and Hurtado, 2009) or grandparents and other older adults (Hawkes et al, 1998; Hawkes, 2003; Hooper et al, 2015). These simple and well-known features of the human life history then go hand in hand with other features of the life history and, particularly importantly, with human forms of sociality (Hrdy, 2009). The fitness benefits arising from different family members, which surely involve contributions of many kinds in addition to food, are critically reviewed by Sear and Mace (2008).

As we will discuss at greater length below, the basic demographic problem posed by the human life history strategy is the long and deep stage of child dependency, which on the one hand runs the risk of parental death wasting the entire prior investment in fitness, and on the other hand leads to a crushing dependency burden at certain life cycle stages even for a fully cooperating parental pair. These problems are solved by human sociality, which in turn may lead to further problems, as will be discussed later. The literature on these and related problems arises partly in the context of evolutionary studies, but also in the study of modern-day societies, and in what follows we will try to link these two contexts.

Age and Economic Roles in Hunter-Gatherer Societies

Figure 1 displays age profiles of caloric production and consumption by age, averaged for the Ache, Piro, Machiguenga[2] and !Kung (Kaplan, 1994; Howell, 2010), with males and females combined. We see that production first equals consumption around age 20, and that production

1 This is an average of males and females. Tsimane female children become self-sufficient some years earlier than males (Hooper et al, 2015).

2 Hillard Kaplan kindly sent me the data underlying the table in his (1994) paper. After some adjustments (Lee, 2000) the average of these three Amazon Basin groups was then averaged 50–50 with the !Kung data, to give equal weight to the Latin American and African experience. But results for each of these four

continues to rise until a peak around age 50.[3] People continue to produce substantially more than they consume, on average, through to the last ages observed (age 65), and we note that in the larger sample of the Tsimane that has a larger sample at higher ages, net production is still positive at age 80 (Hooper et al, 2015, Figure 1(a)).

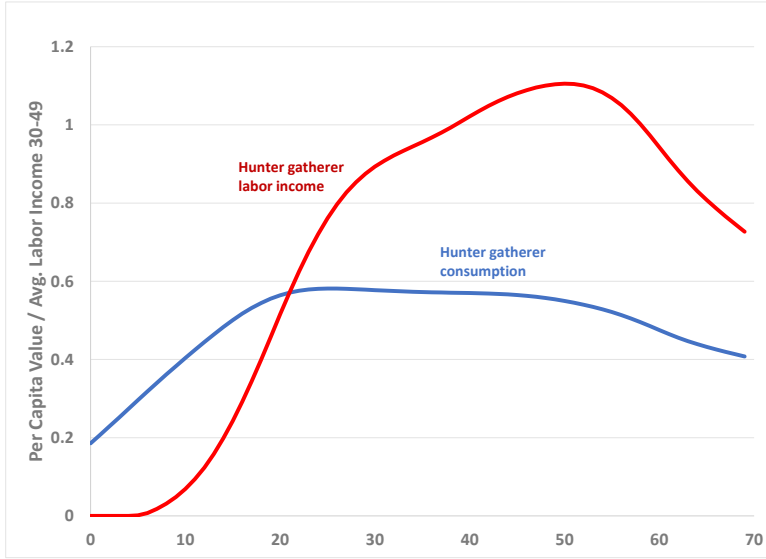


Fig. 1 Age profiles of hunter gatherer production and consumption: averages of estimated profiles for Amazon Basin Ache, Piro, Machiguenga (50%, based on Kaplan, 1994) and Botswana !Kung (50%, based on Howell, 2010).

The first problem posed by the substantial dependency of offspring, as compared to independent newborns who are on their own once born, is that the death of the mother entrains the death of the offspring (Queller, 1994) and the complete loss of the investment in fitness. If this is a problem for wasps (as in Queller) it is a much bigger problem for humans. The solution for insects and for humans is found in reproduction in a larger social group, in which others may take over if the mother or father dies.

The second problem is that the simultaneously dependent offspring place an increasing burden on two collaborating parents up until around twelve to fifteen years after the start of mating. The Russian economist Chayanov (1925/1986) was first to analyse the economic life cycle of the family in this way using survey data from Ukrainian peasants in the early twentieth century. He considered a hypothetical couple marrying at age 20 and having a birth every two years. Combining this pattern of household age composition with age schedules for production and consumption, he found that the ratio of consumers to producers, now known as the "Chayanov ratio", doubled after fourteen years of marriage. Similar results have been

groups were quite similar, and also quite consistent with the Tsimane age profiles for net production in Hooper et al (2015).

3 Caloric consumption is not completely satisfactory as a measure, because calories derived from fruits and vegetables are not regarded by these societies as being as nutritious as calories derived from meat. Older people may switch from hunting to gathering or horticulture, thereby maintaining a high caloric productivity, but with what may be some decline in nutrition and quality that is not shown on this graph.

found for calculations based on a group of Mayan subsistence swidden agriculturalists (Lee and Kramer, 2002), where the ratio more than doubled by the tenth year, and by Tobin (1967) for the US. In the relatively favourable conditions of the Ukraine, peasants met this rising ratio with increased hours of work, a result known as "Chayanov's Law", but in the harsher conditions of hunter gatherers that option would have been more restricted.

However, elder hunter gatherers continue to produce more than they consume on average, and the surplus is transferred to their children and grandchildren, helping to offset the rising dependency ratio — a possibility that Chayanov did not consider. Hawkes (2003) and her collaborators (Hawkes et al, 1998; Voland et al, 2005) have emphasized the importance of the grandmother's role, while Gurven and Kaplan (2006) find greater contributions from older men, but the contributions of these elders was surely important in any case. Hunter-gatherers achieved further flattening of the rising Chayanov ratio by sharing food within groups of around three to five households and eight to twenty-five individuals containing both kin and non-kin (Binford, 2001; Gurven, 2004; Hooper, 2015).[4] Of course, this sharing was not perfect, and households shared more generously with the households of others who were related to them, but non-kin participated to some degree as well (Hooper et al, 2015, provide detailed data and analysis). Within these sharing groups the household-to-household variations in Chayanov ratios tended to average out to a more stable level of dependency.

The economic role of children in the household economy is also potentially important. In settled agricultural societies there are safe and productive opportunities for children to contribute to household output. Cain (1977) found that male children in a Bangladesh village broke even by age 12, and Caldwell (1976) argued that the important economic contributions of children motivated parents in agricultural societies to have high fertility. These views have been questioned (Kaplan, 1994; Lee, 2000; Lee and Kramer, 2002; Robinson et al, 2008). In the Mayan subsistence agriculture group children began to breakeven around age 16, and actually contributed around 60% of the total family output from year twenty to year thirty-five of the average marriage, but, even so, the Chayanov ratio more than doubled as reported above. In hunter-gatherer settings, foraging was apparently more dangerous for children, and in any case they were not very efficient workers. Their limited contributions lead to a later break-even age near 20 (Figure 1).

The Evolution of Life Histories

With this background on the nature and importance of human sociality, we will now consider how this social context might have altered the forces of natural selection that shaped the human life history. Hamilton (1966) was the seminal mathematical study linking genetics and mutation to the evolution of the life history through natural selection. In his setup, each birth inherits a genome from its parents, but mutations also occur and each mutation can be thought of as a deleterious error that will raise mortality at some particular age. If a particular mutation raises mortality at an age before the start of reproduction, let's say at age 11, then this mutation strongly reduces the recipient's reproductive fitness and it would tend to be strongly

4 According to Binford (2001) hunter gatherers lived, travelled and foraged in these smaller groups during the lean season in each region, while congregating in larger groups of around 500 in seasons when food was plentiful.

selected out of the population. If instead the mutation raises mortality at age 35, when much of a female's potential reproduction is already behind her, it would reduce her fitness much less (in proportion to the small share of total lifetime reproduction remaining after this age), and the force of selection against it would be correspondingly weaker. If the mutation raises mortality at age 50 then, in Hamilton's theory, it would have no effect on reproductive fitness at all and so would not be deselected. Such mutations would accumulate, leading to a rapid increase in mortality after menopause. It seemed to Hamilton that this approach made more sense for mortality, where deleterious mutations might release no energy for other uses, than for fertility, where increases or decreases would entail trade-offs affecting other aspects of the life history. For this reason, we also will focus on mortality in what follows, although we believe that this problem of trade-offs also undermines Hamilton's analysis of mortality in ways that we hope to avoid with our approach — for example, the survival or death of a child affects the resources available to siblings and the parents, thereby indirectly affecting their survival.

In reality, human mortality does not rise explosively after the age of menopause, but rather continues its gradual Gompertzian ascent, and Hamilton recognized that this posed a problem for his theory (see Burger, 2017, on the evolution of human mortality schedules). Hunter-gatherer females have substantial post-reproductive survival, as has been well documented (Gurven and Kaplan, 2007) and as is widely accepted by demographers and anthropologists. According to the grandmother hypothesis (Hawkes et al, 1998; Hawkes, 2003) post-reproductive females continue to enhance their reproductive fitness by assisting their children and grandchildren. A similar argument can be made for older men, as discussed earlier.

Hamilton's theory also implied that mortality following birth would be low and flat until the age of reproductive maturity,[5] whereas (as he discussed) actual mortality is very high following birth and then declines to a low point near reproductive maturity. This is a second problem for his theory.

Lee (2003) developed a mathematical model that sought to extend Hamilton's theory by incorporating the role of intergenerational transfers of food in promoting fertility and survival, and showed that in this case the force of selection against mutations affecting mortality at any age would be proportional to a weighted sum of the Hamilton effect (the proportion of lifetime fertility remaining) plus a transfer effect (the proportion of lifetime net transfers remaining to be made to kin). If this species makes no transfers after birth, then the Hamilton weight is 1.0 and the transfer weight is zero. In a species making transfers after birth, however, evolution would move the system toward the level of fertility that optimized the quality-quantity trade-off for births, at which point the transfer effect weight would be unity and the Hamilton effect weight zero, as for humans. In this theory, as in the grandmother hypothesis, there is post-reproductive survival because older people continue to enhance their reproductive fitness. Mortality is high at birth because expected net future transfers are zero, with expected future transfers to be received by a baby balanced by expected future transfers to be made by it to its own offspring. But as children grow older, transfers already received increase with no change

5 It is sometimes thought that in Hamilton's theory, evolved childhood mortality should be inversely proportional to Fisher's reproductive value, which would closely match the actual pattern of child mortality, but this is not correct. It should be inversely proportional to the share of lifetime reproduction remaining after each age, and that share remains 100 percent from birth until the age of reproductive maturity.

yet in those to be made in the future, so the expected future net transfers turn positive, and selection against deleterious mutations rises, so mortality falls. This is one explanation for why mortality declines following birth. Another way of putting this is that when an infant dies, all the transfers that would have been made to it in the future are saved and can be used for a replacement birth, so the death is not costly. But when a 12-year-old dies, all that has already been invested in that child is lost and cannot be replaced, so the death is very costly.

This model captures the average effects of intergenerational transfers but it assumes that each sharing group has a stable population distribution, on average. This assumption would be harmless if the consequences of age distribution variations were linear, but they are not. A mother who survives until a child is halfway to independence will not leave half a surviving child, she will leave zero surviving children, for example. The stability-on-average assumption does not incorporate the catastrophic effect of maternal death. Another troubling assumption is homogeneous genetic lineages. The simulation model described next does not require these assumptions. It is also able to incorporate more ethnographic detail for social group living and food sharing.

Modelling and Simulating the Evolution of Life History in a Social Context[6]

The starting point is 100,000 single-sex individuals of different ages who are subject to probabilistic fertility and mortality at five-yearly simulation steps. Probability of death depends on age, genome and food consumption. Probability of giving birth depends on age (according to an initial hunter-gatherer age-specific fertility schedule) and food consumption in the previous five-year simulation cycle. Each birth inherits the genome of its mother (consisting of some number of deleterious alleles raising mortality at each age and setting an initial age schedule of mortality for that birth) but mutations also probabilistically occur, modifying the inherited age schedule by further raising mortality at certain ages.

Individuals live in households that share food. A matriarchal household contains all individuals descended from an oldest single living female, for example a grandmother, her two surviving daughters and the children of each. If the oldest household member dies, this household would split into two new matriarchies under each of the mothers. Food in such households derives from age-specific production (foraging) according to an age schedule like that in Figure 1, and the age-production profile is shifted downwards in inverse relation to population density. An individual's production also depends on her level of consumption in the prior period and on her level of consumption as a young child, which is assumed to influence her adult size, health and vigour. This turns out to be a key feature, and it will be discussed in more detail later.

Based on this treatment of production, each household will have a certain level of total output each simulation cycle. This output is allocated to the household members in proportion to a consumption age profile like the one in Figure 1, the level of which is adjusted so that total household consumption equals total household production. In this way the age composition and dependency structure of the household affects household production and the level of

6 The code in R for the microsimulation program used in this paper is posted at https://github.com/carlboe/SocEvoSims. We encourage others to experiment with this simulation program and to modify it for use in different ways.

consumption by every individual, which is the central feature of the simulation. Every individual in the simulation is in this way assigned a level of production and consumption, and the difference between these two indicates a transfer made to others or received from others. The simulation is then run for many five-year cycles, typically over a span of 75,000 years. Because productivity is inversely related to population size, and the population growth rate (through fertility and mortality) depends on productivity, population size converges to an equilibrium level and to a stable age distribution and age schedule of production, consumption, mortality and fertility.

As an aside, here I will briefly discuss the relevance of population equilibration for hunter-gatherer populations. In these simulations, populations do reach a stable equilibrium. The model underlying the simulations incorporates individual-level demographic uncertainty because fertility and survival are probabilistic. However, this form of individual-level uncertainty largely cancels out for population-level outcomes such as population size and age-specific death rates. The model does not incorporate other kinds of uncertainty, such as variation in food availability due to changes in weather or climate, or variation in mortality due to epidemics. Lee (1987:453) incorporated both equilibration and random macro-perturbation in simple population models calibrated to preindustrial human conditions. He found that population size swung widely around its hypothetical equilibrium, with a standard deviation from equilibrium of 7% and with swings away from equilibrium that last hundreds of years. Indeed, a simulated population size series with equilibration looked indistinguishable from another with no equilibration over a simulation period of one thousand years when both were subjected to the identical random shocks. It was only in the next thousand years that they diverged. Thus "the gentle nudge of homeostasis becomes a dominant force in the longer run" (1987:454). The presence or absence of a tendency toward equilibration could only be discerned through studying the mechanisms at work, and not through change or stability total population numbers. For this reason, I disagree with the "forager population paradox" based on observed boom-bust cycles in contemporary hunter-gatherer populations.

The matriarchal social system described above, which we will label "M.100", is the simplest system simulated. Actual hunter-gatherer societies had larger and more complex food sharing groups as discussed earlier. In other simulated systems, related matriarchal households are grouped together. Here we will emphasize a system in which individuals group together if they are third cousins or closer (have a common great-great grandmother). We label this system "K5.100" because kin groups span five generations. Here and above, the "100" means that the M or K5 groups retain 100% of their output and do not share at all with other groups or households. Another system takes into account Binford's (2001) conclusion that hunter gatherer sharing groups contained between eight and twenty-five individuals during the lean season. In this system, K5 households are grouped together to form sharing groups in this size range, and in the simulations this means that most of households in sharing groups have no kin ties with other households. We label these SG.K5 for "sharing group composed of K5 households". As time passes, the groups may shrink below eight members, in which case they fuse with another small group, or may grow above twenty-five in which case they split into two sharing groups. Because food sharing was biased toward own kin in such sharing groups (Gurven, 2004; Hooper, 2015), the share of food placed in the common pool by each K5 group can be specified, and here we report results for SG.K5.50 which indicates that 50% of each household's output is kept within it, with the remainder placed in the common pool of the sharing group. In some

variations there is reshuffling of the K5 components of a group every five years, indicated by a "D" for dispersion. This is intended to reflect the fact that membership in sharing groups is in fact fluid, and also that young adults would often leave the group to join a mate.

A reviewer suggests that it would be interesting to make the dispersion and reshuffling non-random. The reviewer points out that hunter-gatherers like to be in groups with good hunters, e.g. Ache (Wood and Hill, 2000) and Hadza (Wood, 2006) and with cooperators (Apicella et al, 2012). At the same time, since both the man and the woman in a couple want to live with their own kin (Dyble et al, 2015), sorting into groups based on genetic relatedness is limited. Our model has only one sex, and within each kind of social arrangement simulation, all are equally strong or weak cooperators. But there is heterogeneity in hunting ability arising from childhood nutrition, and sorting into groups based on hunting ability would certainly lead to the kind of positive feedback loop that we will discuss later.

Social Sharing and the Evolution of Age-Specific Mortality

With this background, consider Figure 2 which plots the long-run equilibrium evolved age schedules of mortality,[7] averaged over the last 300 years at the end of the 75,000-year simulation period[8] for different social arrangements. Experiments find that the same schedules evolve every time, over multiple trials, and that nearly identical mortality schedules evolve regardless of the initial mortality-age schedule assumed, including completely flat age schedules. All these average simulated age schedules have very tight confidence bounds, which are not shown.

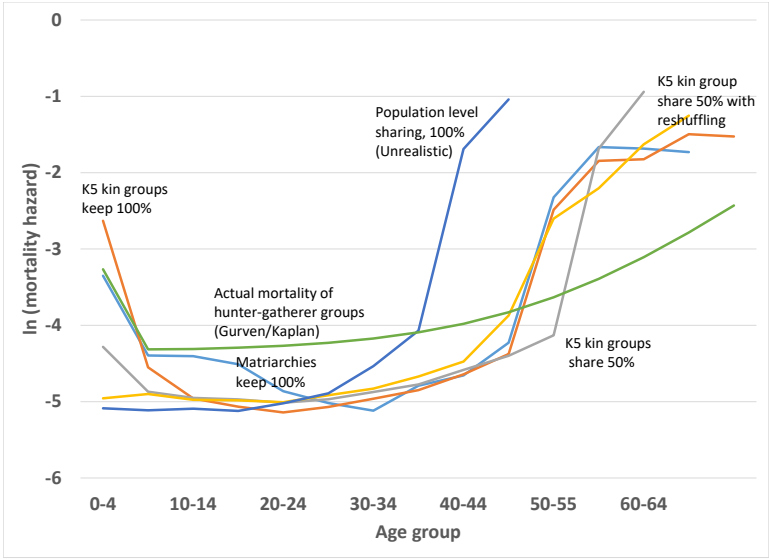


Fig. 2 Evolved mortality under different social arrangements after simulation runs 15,000 cycles (75,000 years), and contemporary hunter-gatherer mortality (Gurven and Kaplan, 2007, Siler fitted curve). Line of age-specific death rate plotted at midpoint of 5-year age group.

7 These are not strictly speaking equilibria, because at the highest ages mutations continue to accumulate despite the weak selection against them, but for practical purposes we may think of them as equilibria.

8 Averaging smooths out the jaggedness in cycle-to-cycle age schedules, which arises from randomness and the size of the simulated populations.

Note: "Actual mortality" is calculated from the parameters of the Siler curve for average non-acculturated hunter-gatherers given in Gurven and Kaplan, 2007, p. 327. K5 indicates all group members are at least third cousins. A matriarchy group contains all descendants of a single living female. Reshuffling indicates that the K5 components of a group are shuffled every five years. Keep or share refers to the proportion of a kin group's output that is kept in the kin group rather than shared with others.

We can begin with the Pop.100 simulation which assumes a completely unrealistic social system in which 100% of output is shared with the total population. This simulation leads to the Hamilton (1966) outcome with almost no post-reproductive survival and low and flat infant/child mortality, as discussed earlier. The reason is that with universal sharing, there is no fitness advantage in any given female surviving past menopause since her offspring would be fed by the total population in the event of her death. By the same token, the rising expected net future transfers by older children do not lead to lower mortality, because they have no impact on the fitness of these children through kin selection. Kin selection is taken out of the picture by universal food sharing. This case is a convenient benchmark and deviations of simulation outcomes from this benchmark case will reflect differences in food sharing rules.

It is instructive to note that the Pop.100 system achieves perfect sharing, completely eliminating the life-cycle squeeze and providing complete life insurance since in the event of a mother's death her children will be provisioned like all other children. Yet here we see that there is a downside to this complete sharing, because natural selection is unable to act on individual mortality variation, and consequently post-reproductive survival cannot evolve. As we shall see later, this means that Pop.100 leads to a chronically unfavourable dependency ratio, and equilibration at a low population density. As a result, this social arrangement would be vulnerable to deselection in intergroup competition.

The M.100 simulation corresponds most closely to the setup in Lee (2003). In it, matriarchal households retain 100% of their product, sharing none with other households. Mortality starts high at time of birth. One way to think of this is that if an infant dies, all the food that would have been used to rear it can now instead be used to feed siblings and the mother, the mother can soon get pregnant again, and the resource cost of the child death is consequently very low and has little impact on reproductive fitness. Mortality then declines from birth to a low point around age 30, after which it rises. This is quite different than a modern mortality-age schedule, but it is more consistent with the mortality patterns of hunter-gatherer groups studied over the past half century as summarized and analysed by Gurven and Kaplan (2007, Figures 2 and 10). They found that mortality remained quite low through mid-adult years until beginning a rapid rise at around age 40, after which it doubled every eight or nine years. Nonetheless, they find substantial post-reproductive survival. Remaining life expectancy at age 45 averages 20.7 years across hunter gatherer groups (Gurven and Kaplan, 2007:327), somewhat higher than in these simulation results.

There is an interesting bulge in M.100 mortality at ages 10–14 and 15–19. This reflects the reduced consumption during the family life-cycle squeeze when a mother and perhaps grandmother must also feed a number of children without help from a broader kin group. Put differently, this bulge is due to economic circumstances, not to a bulge in accumulated mutations affecting these ages, and therefore not heritable.

In the K5.100 simulation we see that infant mortality is even higher than in the M.100. The future resources freed by the death of child can be captured even more efficiently by the other kin, so the resource costs of the child death are even lower in this case, and the mutations leading to higher infant mortality are only weakly selected out of the population. However, starting at ages 5–9 the K5.100 mortality is lower than the M.100, because the family life cycle squeeze is much reduced by the larger size of the sharing group, and also because if the mother dies, other kin are available to raise her surviving offspring — the life insurance effect. After age 15–19, however, the M.100 and K5.100 are very close. Why does mortality at 60–64 and above flatten out in the M.100 and K5.100 results? We believe it is because at these higher ages it is more likely that surviving older women will have grandchildren in whom to invest, so selection against mutations promoting their death is stronger.

The SG.K5.50 groups are larger, with 8 to 25 members, consisting of multiple unrelated K5 families. These families keep half their output and put half in a common pool for broad sharing. In this case infant mortality is very much lower with less than one tenth as many accumulated harmful mutations affecting this age than in the K5.100 groups. Selection is stronger against these infant mortality mutations in the SG.K5.50 because when an infant dies the family recaptures only half of the food that would have fed this child in the future. The other half would have come from the common pool, and it is mostly lost to the family.

From age 25–29 to 45–49 mortality for M.100, K5.100 and SG.K5.50 is very similar. At 50–54 the SG.K5.50 mortality is much lower than the others, probably due to a greater likelihood of surviving related offspring to care for. Thereafter it is higher, perhaps because of the greater availability of other surviving elders to care for the dependent offspring, so the survival of any particular individual elder is less important.

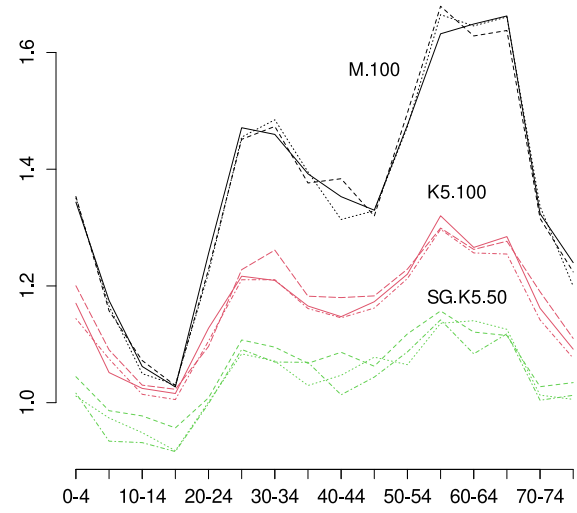
The final social arrangement in Figure 2 is SG.K5.50.D, in which the K5 components of a larger sharing group are reassigned to these sharing groups every five years. This would only matter if the unshuffled groups were more highly interrelated than the shuffled ones, and we see in Figure 2 that this is indeed the case. The shuffled ones have much lower infant mortality because a far smaller portion of the resources released by an infant's death is recaptured by the kin group with shuffling. There are also differences above age 45–49, but the explanation is not clear.

Social Sharing and the Evolution of Other Aspects of Population and Life History

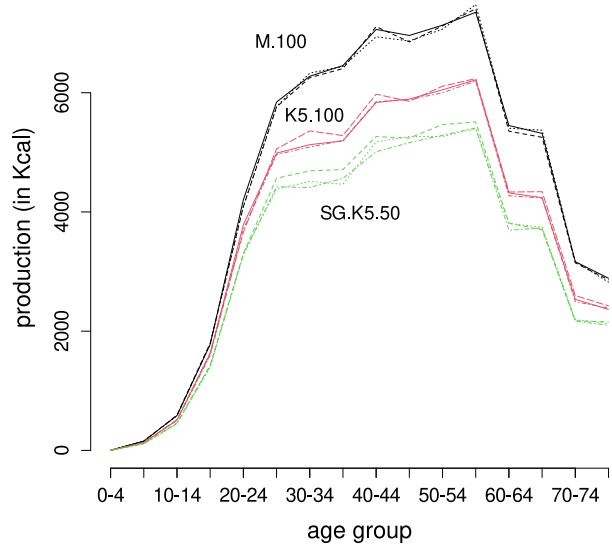
Although the genome in this simple model only directly affects age-specific mortality, it nonetheless has indirect effects that reverberate through the population and the life history. For example, when a broader sharing group bears half the costs of raising a child, then we would expect that natural selection would lead not only to lower infant/child mortality, as we saw above, but also to higher fertility, both through a kind of evolved (non-behavioral) free riding, other things equal.

Panel (a) of Figure 3 shows, for three simulations for each of M.100, K5.100, and SG.K5.50 (nine in total), the ratio of consumption at each age to the levels shown in Figure 1, after 500 years of simulation when these have stabilized. For each social arrangement the three simulations lie close together, showing that the results are consistent and systematic. In every case, average consumption is low at ages 5–9 to 15–19, when the dependency ratio (Chayanov

ratio) is unusually high, but the contrast between these younger ages and the later adult years is greatest for M.100, because in this case many adults end up with no surviving offspring with whom to share. This is much less true for the K5.100 group in which the life cycle squeeze is diluted by a larger kin group with a more diverse age distribution, and even less true for the K5.50 group with broader sharing among K5 groups.



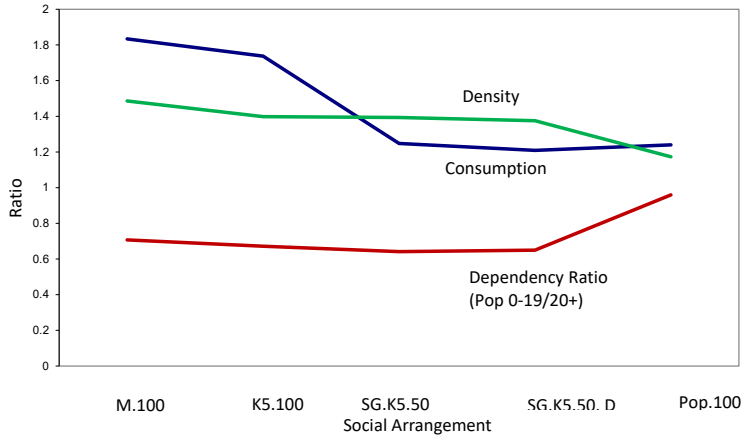
(a) Consumption



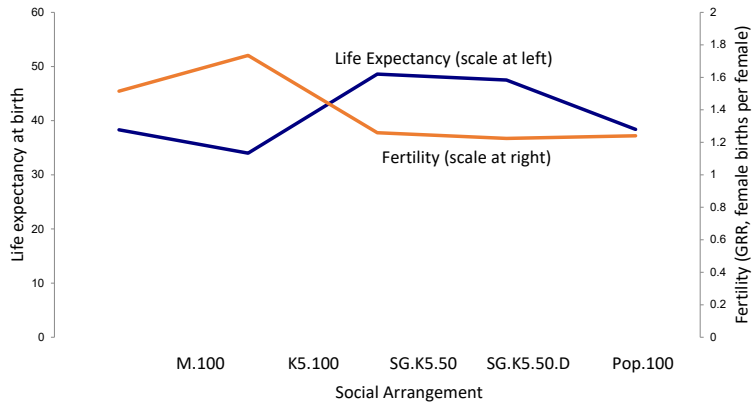
(b) Production

Fig. 3 Average age profiles of consumption and production by social arrangement. Consumption is expressed as a ratio of the age profile in Figure 1 and production is expressed in Kcal.

At the same time, it is striking that the average level of consumption is far higher in the M.100 group than the others, and the SG.K5.50 with broader sharing has the lowest consumption levels. Panel (b) of the figure shows the evolved level of production by age in the same way, which is clearly highest for the M.100 arrangement and lowest for the SG.K5.50. Why should consumption and production be highest in the M.100 group with the narrowest food sharing and closest relatedness? One possibility to consider is that the population density of this arrangement is lower, resulting in higher productivity and therefore higher consumption, a possibility we now consider.



(a) Density, Consumption and Dependency



(b) Life Expectancy and Fertility (GRR)

Fig. 4 Other outcomes by social arrangement.

Note: In Panels (a) and (b), Social Arrangements on the horizontal axis are arranged from least to most food sharing with less close kin or farther to right with non-kin, so the lines indicate the effect of food sharing on long-run equilibrium-evolved outcomes. In Panel (b), fertility is measured by the GRR or Gross Reproduction Rate which is lifetime female births per female. The simulated populations are in equilibrium with growth rates very near zero, so differences in fertility and life expectancy must be offset.

Figure 4 shows long-term equilibrium outcomes by social arrangement, with the arrangements listed on the horizontal axis in order from sharing in a narrowly defined highly interrelated group to a broader less interrelated group. Although the arrangements are categories, we plot the results as lines because with this ordering or categories it is easiest to see the association of outcome with sharing/relatedness. We see in Panel (a) that equilibrium population density is highest for the M.100 group and lowest for Pop.100. Nonetheless, consistent with Figure 3 (a), consumption is highest for the M.100 group. Clearly the explanation proposed just above is incorrect, since density is actually highest for the M.100 group, which in itself would reduce its level of consumption and production, other things equal. We also see that the dependency ratio for M.100 is slightly higher than that for K5.100 and SG.K5.50 which in itself would also lead to lower consumption.

In Panel (b) we see that fertility is highest for K5.100, but is also quite high for M.100 in contrast to broader sharing arrangements. Life expectancy varies inversely with fertility because in equilibrium the population growth rate is zero, and the Net Reproduction Rate must be 1.0. The outcomes shown here are interesting but they do not explain the patterns observed in Figure 3, so we now consider a different and unexpected possibility.

Recall that the simulation model assumed that the childhood consumption level had a positive effect on later-life productivity, through the well-established effects of early childhood wellbeing on later-life outcomes. Similarly, adult consumption levels affect adult productivity in the next cycle. These are features of the model for all social arrangements, but they turn out to favour the M.100 group precisely because its narrower sharing base makes consumption outcomes more variable. Sometimes a child's mother dies, and then it consumes little or nothing and dies too. But other times a child has a surviving mother and a surviving grandmother, and everyone in the family eats exceptionally well. In this case the child grows up to become a strong producer who can feed her own children better and survive better, and this efficient production continues with positive feedback since her children are consequently better fed. The broader social sharing in K5.100 and even more so in SG.K5.50 reduces this variability in consumption and thereby reduces the chance of entering the positive feedback loop. Variance can be good! And stable mediocrity can be bad.

Figure 5 presents box plots displaying the dispersion of consumption by social arrangement, assessed in the standard group selection simulations (which will be explained below in reference to Figure 6 (a)) after 2,500 years (500 cycles) of simulation. Although we started with M.100, K5.100 and SG.K5.50, by 2,500 years the three SG.K5.50 groups were extinct so the figure only compares the M.100 and K5.100. It shows box plots for six simulations, three for each social arrangement. The horizontal line gives the median, the box indicates the interquartile range (from 25th to 75th percentiles), the top and bottom lines indicate the 75th and 25th quartiles plus and minus 1.5 times the interquartile range, and the circles indicate outcomes outside that range. We see that there are many more circles outside that range for M.100 consumption than for K5.100, reflecting the greater smoothing of consumption variation in the K5.100 arrangement.

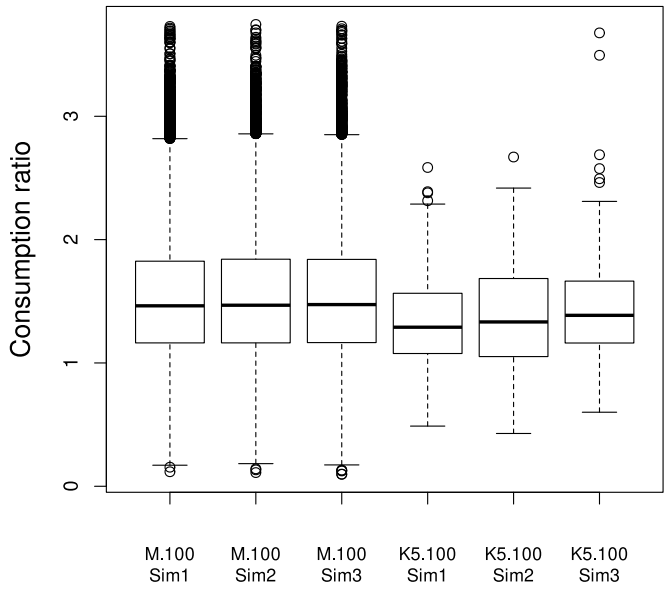


Fig. 5 Dispersion of individual child consumption outcomes after 2,500 years (500 cycles) of simulation according to social food sharing arrangement, M.100, K5.100 and SG.K5.50. However, by 500 cycles all three SG.K5.50 simulated populations had gone extinct, so are not shown here.

Note: Child consumption is measured as a ratio to the level of consumption in Figure 1 within a family unit (M or K5). The box shows the interquartile range (Q2 to Q3) and the horizontal line in the box is the median outcome. The vertical lines outside the boxes extend to 1.5 times the interquartile range above and below the upper and lower edges of the box. The circles fall outside that range. The boxes for M.100 are considerably longer than those for K5.100, and many more points fall outside the range of the vertical lines, showing that the distribution of child consumption in M.100 is much wider than for K5.100.

We will pause here to consider the plausibility of this unforeseen process. Among actual hunter-gatherers there is a great deal of heterogeneity in ability, and some of this might result from childhood conditions. A study found that among Ache hunters at the age of peak productivity (around age 40), the worst hunters got zero on 70% of days they hunted and the best hunters on only 30% of their hunting days (Koster and McElreath, 2014:117). There is a vast literature on the effects of childhood environment on later health, education, and wages. Perhaps most relevant and most persuasive are studies of long-term outcomes based on earlier randomized childhood interventions. One such study (Parker and Vogl, 2018) of the PROGRESA contingent cash-transfer program in Mexico found that wages were 50% higher for girls in households that had earlier received the randomized cash transfer, while the effect for males was the same size but was not statistically significant. Another study (Hoddinott et al, 2008) found that a randomized nutritional intervention program in Guatemala boosted men's wages by 46% decades later. These studies make clear that improved childhood conditions can lead to big increases in later productivity. However, it is not clear how to translate such findings into the context of hunter-gatherer children raised with higher levels of consumption. Nor is it clear what upper limits there may be to the size of such effects, but evidently a 50% increase is possible, and that is large. The simulated mean differences in productivity levels in Figure 3 (b) are certainly less than 50%, but those also reflect other factors such as population density.

Natural Selection at the Level of the Individual and the Group

The idea that natural selection operates simultaneously at many different levels, including group selection, has gained wider acceptance in recent years (Boyd and Richerson, 1990; Richerson and Boyd, 2004; Nowak, 2006; Okasha, 2009). Cultural evolution has also increasingly been recognized as an important process in humans and other animals (Cavalli-Sforza and Feldman, 1981; Whiten et al, 2017). Here we will ask which of the social arrangements we have described would prevail if they coexisted and foraged in the same environment, subject to the same overall population density effect on their foraging productivity, but without any other form of interaction. Assuming that somehow these sharing arrangements came into existence through cultural or biological evolution, which would be most successful?

The social arrangements we have considered differ by the size and stability of the group within which food is shared and by the closeness of kin with whom food is shared. Such different arrangements could either reflect genes or cultures. In head-to-head competition through their shared population density, some groups will do better than others. Under stable environmental conditions, the winning social arrangement would be able to equilibrate at a higher density than the other groups which would then have negative growth rates and eventually die out.

We implemented this idea by running nine concurrent simulations, three each for populations with M.100 social arrangements, three with K5.100, and three with SG.K5.50. All nine groups are assumed to forage in the same area and therefore the productivity of foraging in each group is affected by the total population size (sum of the nine individual simulated populations) and overall density, updated at each simulation cycle. Simulating three populations for each social arrangement helps us to assess whether outcomes are systematic and not accidental. The groups able to equilibrate at the highest density will win out as the others decline toward extinction.

Figure 6 plots the log of population size against time measured in cycles for each of nine simulations. Panel (a) shows the standard model specification which includes the effect of childhood consumption on later life productivity. The outcome is opposite to our expectations, although it was foreshadowed by the densities shown in Figure 4 (a). The M.100 arrangement quickly and completely dominates K5.100, which in turn dominates SG.K5.50. This outcome reflects both evolutionary processes within each social arrangement and also group selection operating on each of the nine simulated populations as they compete through their abilities to sustain themselves at each density. Looking closely at the first cycles shown in Panel (a) we can see that M.100 does worse than the other specifications for the first 100 years or so as expected, because the other arrangements do benefit from broader sharing in a larger group and from the life insurance that protects child survival. But thereafter the M.100 populations grow.

Why does M.100 win? Recall our earlier discussion of why the M.100 arrangement achieved a greater equilibrium density than other arrangements in Figure 4 (a), due to the greater variability of childhood consumption across families and its effect on later life production. To explore this effect, we turn off the child-consumption-productivity feature of the simulation model. The new result is shown in Panel (b). Now M.100 goes quickly to extinction as originally expected, while the broadest sharing arrangement, SG.K5.50, triumphs completely. Evidently the surprising success of M.100 in group competition is indeed due to the effect of child consumption on productivity. The greater variance combined with nonlinear positive feedback help the M.100 to recoup its early losses and dominate the others.

In a series of experiments, of which two are shown in Figure 7, we limited the boost to adult production that could derive from elevated consumption in childhood. Panel (a) shows that with a limiting factor of 1.35 the M.100 arrangement continued to win out decisively. However, with a slightly lower limiting factor of 1.25, the SG.K5.50 social arrangement won out decisively. The tipping point is evidently close to 1.30. These experiments and the literature reviewed earlier suggest that variability in child wellbeing could perhaps have conferred some evolutionary advantages for hunter-gatherers in the past through non-genetic inheritance of nutritional status and productivity.

(a) Standard Simulation

(b) Simulation with child productivity effect turned off

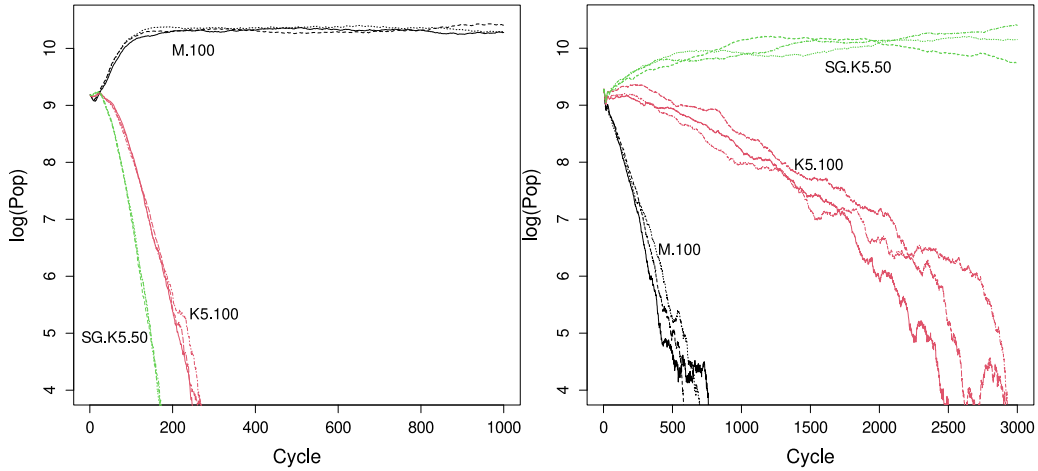


Fig. 6 Evolutionary competition among nine simulated subpopulations initially of 5,000 each, with three each of M.100, K5.50 and SG.K5.50. Plot shows the log of total population size for first 1,000 cycles (5,000 years). Initial population age distributions and mutational distributions are taken from evolved states in individual runs for each social arrangement.

Key: Black=M.100; Red=K5.100; Green=SG.K5.50. For definitions of M=Matriarchy, K5 and proportion shared (here 100% or 50%), see Note to Figure 2 or the text.

Sociality in the Modern World: The Family and the State

We have explored how different social settings alter the way that natural selection shapes the evolution of mortality and may lead to post-reproductive survival in the hunter-gatherer setting. These results depend on the shapes of the age profiles of production and consumption that are assumed in the simulations. In particular, the evolution of post-reproductive survival derives from the empirical finding by anthropological studies that on average, adults at all ages produced more than they consumed, including in old age. For hunter-gatherers, longer life and higher proportions of elderly were beneficial because elders helped with the costly task of raising the young through their long period of dependency.

(a) Child productivity effect capped at 1.35 (b) Child productivity effect capped at 1.25

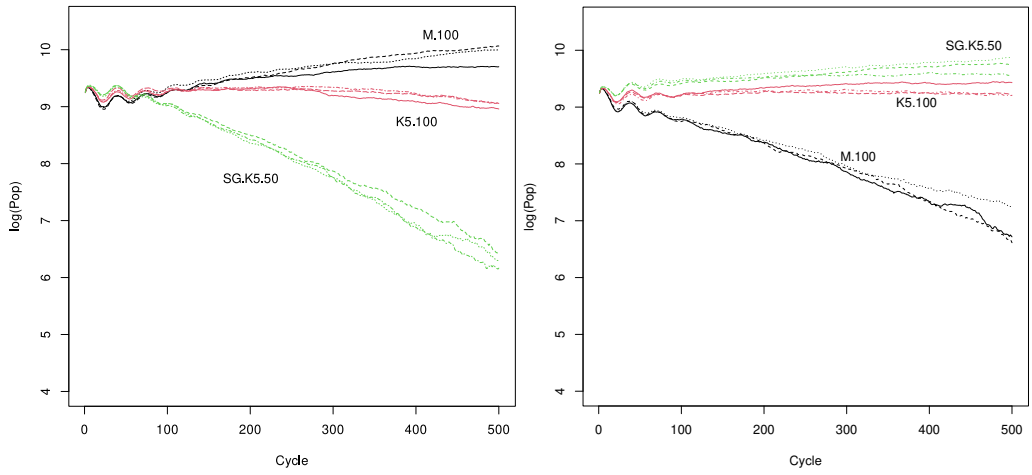


Fig. 7 Evolutionary competition among nine simulated subpopulations when effect of child consumption level on production is constrained to be no greater than a) 1.35 or b) 1.25. Populations are initially 5,000 each, with three each of M.100, K5.50 and SG.K5.50. Plot shows log of total population size for first 500 cycles (2,500 years). Initial population age distributions and mutational distributions are taken from evolved states in individual runs for each social arrangement.

As societies moved toward settled agriculture and developed property rights in land and dwellings, the elderly came to control valuable assets. Perhaps for this reason, age patterns of labour changed dramatically, altering the shape of the economic life cycle as older people reduced their hours of work and came to rely on transfers from their children to support their consumption. This is seen in the National Transfer Accounts (NTA) data (Lee and Mason et al, 2011) for low-income agricultural countries. In Figure 8, to facilitate comparison of the shapes of the age profiles across groups, each profile is divided by the average level of labour income in its group between ages 30 and 49. As shown in Figure 8, in low- and lower-middle-income countries labour income drops below the level of consumption by age 59, and thereafter older people consume far more than they produce. Nonetheless, they earn asset income as a return on the farms or other property they own, even if it is the labour of their children or other younger people that makes those assets productive. (Consumption here includes public transfers of goods and services such as health care and public education, but not public pensions because these are just income and need not be consumed.)

Figure 8 also shows the average labour income and consumption profiles for a group of rich industrial nations. Labour income is even lower in old age than in the lower income group, and consumption which had been flat or falling with age there is strongly rising in the rich countries so that the gap has greatly expanded. The rise of the welfare state, with its high and rising level of public transfers to the elderly for pensions, health care and long-term care, has played a big role in these changes, particularly in the relative increase in consumption at older ages.

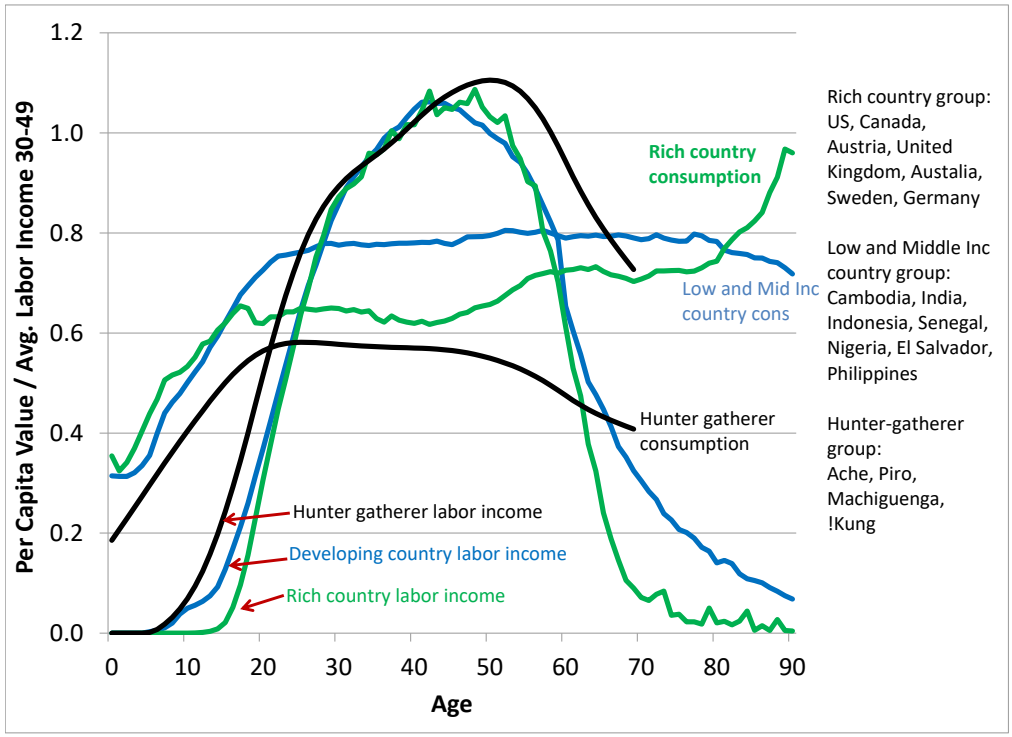


Fig. 8 The hunter-gatherer economic life cycle compared to rich and lower-income countries. Source: Hunter-gatherer age profiles are same as Figure 1. The other age profiles are calculated from National Transfer Accounts data: ntaccounts.org (see text).

Post-reproductive survival and old age most likely evolved in humans to improve the dependency ratio and help to provision and care for dependent children. It is ironic that population aging in the modern world has turned into a great cost due to altered economic behaviour of the elderly. Until the last few decades income flowed downward within virtually all societies, from older to younger ages. The average age of earning labour income was always many years greater than the average age of consuming. The public sector was small, as were pensions and publicly provided health care, and it was through the family that income was transferred to children in the form of consumption goods. This pattern persists everywhere, in that familial transfers still do flow downward in all countries of the world (Lee and Mason et al, 2011). In the rare countries where families do provide net support to the elderly, this support is dwarfed by the transfers to children. Although net familial transfers remain downward to children, public transfers have begun to flow upward in many countries, and in a growing number of countries the net direction of total transfers, public plus private, is now upward. This is a sea change, and it points to the need for very large adjustments to public policy and individual behaviour as populations age.

Discussion

Sociality and intergenerational transfers bring evolutionary advantages as we have discussed. But they also alter the forces of natural selection acting on life history traits. The matriarchal system has strong kin ties between givers and receivers of intergenerational transfers but the individual

matriarchies vary widely in their dependency ratios and life-cycle squeezes. The matriarchal arrangement leads to an evolved age schedule of mortality rather similar to the hunter-gatherer schedule and similar to historical age schedules, for example for Sweden (see Lee, 2003). Remarkably, this same general mortality pattern also evolves for older ages under social arrangements involving weaker kin ties including with sharing groups of the kind observed ethnographically, although levels of infant and child mortality differ considerably due to free riding.

The degrees of genetic relatedness within sharing groups engender different evolved life histories including levels of investment in offspring and differing evolutionary free riding. Some of these combinations of social arrangements and evolved life histories are more efficient than others, in the sense that each can attain and sustain a different maximum population density. The one able to sustain the highest density eventually wins out and is selected in inter-group competition. In general, social sharing confers benefits through life insurance and smoothing the life-cycle squeeze, but incurs costs by distorting the evolution of life histories in the direction of inefficient traits and free riding. The clearest example is the polar case of complete population level sharing in which post-reproductive survival does not evolve, and consequently the population-level dependency ratio is very high and only modest densities can be sustained.

This study has many limitations. Here are some that seem most important. 1) The models and simulations are single-sex with no mating market and no corresponding recombination of the genome at birth. This means that our kin groups are too highly interrelated. Mating would also raise the possibility of some in-migration from neighbouring populations, which would affect interrelatedness. It would be very useful to have some DNA evidence for hunter-gatherer/forager groups like the Tsimane to assess the degree of relatedness in these groups. 2) Mortality at each age is governed by genes/mutations as well as by productivity of the family and sharing groups and the dependency ratios which together determine the level of consumption at each age. However, genes only affect the level of fertility indirectly. Given the age-shape of fertility, its level is determined by consumption, which in turn depends on dependency ratios, productivity levels and sharing rules. It would be possible to introduce a mutation affecting the level of fertility but making the age shape subject to natural selection would be more complex. 3) The age shapes for fertility, consumption needs and baseline productivity are predetermined. The simulated age profiles differ from these due to differences in dependency ratios at different stages of the life cycle, levels of prior childhood consumption, population density experienced at different ages over the life cycle and so on. With our current modelling approach, it would not be possible to relax the need for baseline age profiles. This is not a perfectly general model. It is a model for humans. 4) The social group membership is taken to be given and is not governed by mutating genes governing degrees of sociality. We could instead introduce a mutation similar to mortality that raises preferences for food sharing in larger groups and assume that individuals seek out others with similar preferences to form groups. We could then see what sort of social arrangement and food sharing evolved. A more general approach might start with an optimal life history model like Chu and Lee (2013) and simulate it subject to mutations, which would permit the evolution of transfer behaviour, levels of fertility and mortality, age at maturity, menopause and so on.

This study illustrates the potential for simulation studies that incorporate mutation and natural selection in an age-structured environment with both individual and group selection. The simulations complement the more static analytic solutions of models by Hamilton (1966)

and Lee (2003) by allowing a much richer and more realistic set of assumptions, dynamic interactions of highly nonlinear processes and selection at multiple levels.

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19. Evolutionary Demography of the Great Apes

Melissa Emery Thompson and Kristin Sabbi

The living hominids share a suite of life history features that distinguishes them from other primates, including larger body size, extended juvenile growth and development, and a long lifespan. While modern humans exhibit many distinctions from their great ape relatives, these species provide an important reference by which to infer the life history characteristics of our last common ancestor. Demographic analysis of the great apes reveals specifically how life histories changed during recent human evolution and can provide perspective on inter- and intra-specific variation in life history features. In this chapter, we provide the most detailed information available on demographic characteristics of great apes, comparisons with humans, and discussion of the proximate factors that influence life history variation across the clade.

Introduction

Human life histories are extraordinary in many respects. Even in the poorest of conditions, we are capable of substantially longer lifespans compared with any other primates. Humans have slow growth rates and long periods of infant dependency, followed by prolonged juvenile and adolescent periods. Women reliably experience menopause and a prolonged post-reproductive lifespan, traits that are rare or insignificant in other primates. While weaning ages and interbirth intervals in humans are broadly consistent with allometric relationships to maternal weight across primates, they are substantially shorter than would be predicted from infant development (Harvey and Clutton-Brock 1985; Watts and Pusey 1993; Dettwyler 1995; Kennedy 2005) and much shorter than in other great apes, despite similarities in body mass and elevated costs of brain growth.

Part of our understanding of the origins of human life history requires examining our unique and recent evolutionary history, but a necessary step must come from recognizing our deeper evolutionary roots as primates and as hominids, taxa that evolved slower life history patterns than their ancestors (Charnov and Berrigan 1993). The hominid clade, comprising humans and other great apes, is arguably best defined by virtue of its life history features. A common ancestor of hominids experienced selection for a larger body, increased relative brain size and complexity, a longer lifespan, and an extended period of offspring dependency and juvenile development compared with its progenitors. These features are retained in the living great apes (hereafter, understood to refer to "non-human great apes"): 7 species comprising Sumatran orangutans (*Pongo abelii*), Bornean orangutans (*P. pygmaeus*), Tapanuli orangutans (*P. tapanuliensis*, recently identified), eastern gorillas (*Gorilla beringei*), western gorillas (*G.*

 https://doi.org/10.11647/OBP.0251.19

gorilla), common chimpanzees (*Pan troglodytes*), and bonobos (*Pan paniscus*). These species exemplify, in essence, multiple copies of a common template on which human life history evolved. This allows us to study the factors that can produce variation in life history features within and between species that have similar biology, uncovering potential pathways upon which human traits could have evolved. For example, if we want to know why our species can live for 100+ years, we must first ask how our closest relatives achieved long lifespans and what factors constrain them to 50–60 years.

A valuable feature of using great apes as a referent is that they retain the shared, derived slow life history complex despite having diverged into markedly different foraging environments and having evolved radically different social systems. All great apes, to a certain extent, use a complex diet and seem to prefer fruit — a high-quality, unevenly-distributed resource — when they can get it. All exhibit some social flexibility to minimize the costs of grouping when food is scarce, best exemplified by the fission-fusion organization of chimpanzees and bonobos. However, orangutans contend with radically fluctuating habitats, forcing them to weather periods of extreme food shortage (Knott 2005; Harrison, Morrogh-Bernard, and Chivers 2010; Vogel et al. 2012). They exhibit commensurate adaptations, such as enhanced fat storage, delayed male secondary sexual development, semi-solitary foraging behaviour, and extraordinarily slow metabolic rates (van Schaik 1999; Utami Atmoko and van Hooff 2004; Pontzer et al. 2010). Gorillas display adaptations for processing the large amounts of herbaceous material available in their habitats, effectively buffering both spatial and temporal variation in fruit availability (Remis 2003). At the extreme, mountain gorillas living in the Virunga Mountains can subsist with almost no fruit in their diet (Harrison and Marshall 2011).

While the utility of contextualizing human demographic features with primate data has long been recognized, high-quality data have been scarce, particularly for the great apes. The most prominent reference datasets on primate life history (e.g., Harvey and Clutton-Brock 1985; Smith and Jungers 1997; Leigh 2001), which continue to yield important analyses, incorporate small samples derived primarily from captivity. Captive data have a number of drawbacks, the most obvious of which is the influence of improved nutrition and medical care on features such as growth, reproductive rate, and mortality. Reproductive data are particularly fraught, due to contraceptive measures, separation of mothers and infants, and group management decisions that affect breeding opportunities. These practices are often applied intermittently (e.g., contraception only for certain individuals or at certain times), non-randomly (e.g., selection for breeding of certain individuals), and with poor documentation. Captive data are valuable for understanding the limits of variation within each species, by comparing them to wild data. Such comparisons have some less well-recognized problems. For example, all gorillas in captivity are lowland gorillas, overwhelmingly of the western species (Nsubuga et al. 2010), while data from the wild come primarily from one population of mountain gorillas, which lives at an extreme of gorilla habitat variation. Similarly, the vast majority of founder chimpanzees in captivity (along with most museum specimens) were caught in West Africa, and thus represent the subspecies *Pan troglodytes verus* (Ely et al. 2005), while the majority of wild demographic data are being generated by East African sites comprising *P.t. schweinfurthii*. To the extent that data on other subspecies are available, it is difficult to evaluate whether variation is due to true taxonomic differences or to phenotypic plasticity in response to local environments. Wild data also have their limitations. For example, ages of many individuals are estimated, particularly

for the dispersing sex. Limiting analyses to individuals with well-known ages can also impose bias, if the excluded individuals pursue specific strategies that might alter life history events (e.g., transient male orangutans). Nevertheless, great ape research in the wild has reached a point where there are a number of high-quality longitudinal demographic datasets, in many cases representing multiple populations within species. In our discussion, we focus on these wild datasets as much as possible, referring only to captive data where they are the only data available or where they can provide meaningful insights on the range of variation.

Just as the sparsity of demographic data on great apes has constrained comparative analyses, the desire to conduct such analyses has led to the overzealous use of very limited demographic datasets. Thus, this chapter is as much about how to interpret the great ape demographic data as it is about the data themselves. Are datasets comprehensive enough to make conclusions about species differences? What sources of bias occur in great ape demography, and how important are they in different datasets? How can we best apply comparisons to human demography? Our goal is to emphasize what conclusions about interspecific variability, and the sources of that variation, can reasonably be made and what further data might be needed to address outstanding questions.

Mortality

Understanding how humans came to be so long-lived necessitates considering the evolutionary backdrop of long lifespans in our sister hominids. As a group, these species have notably lower adult mortality rates than do other primates, likely an essential precondition for slow growth, large body sizes, and other aspects of a "slow" life history (Charnov and Berrigan 1993). Great ape lifespans are so long that even the most intensively studied wild populations have not been studied long enough to capture the maximum lifespan. Several chimpanzees, all females, from the Kibale Forest in Uganda are estimated to have lived into their 60s, though their exact ages are uncertain (Hill et al. 2001; Muller and Wrangham 2014; Wood et al. 2017). Two captive chimpanzees, Cheeta (of Tarzan fame) and Little Mama (Lion Country Safari Park), are estimated to have lived nearly 80 years, but their original provenances are controversial. It can generally be concluded that only a small fraction of great apes survive past the age of 50, though they appear to be capable of exceeding 60 years in exceptional circumstances. In most wild populations, life expectancy of females exceeds that of males.

A summary of mortality data from wild populations is provided in Table 1 and Figure 1. Despite their long lifespans, wild great apes do not have reduced early life mortality compared to other primate species (Bronikowski et al. 2011). Infant mortality rates during the first year of life range from less than 10% in some populations of orangutans, western gorillas, and bonobos, to 20% or higher in some populations of chimpanzees and eastern gorillas. Infanticide plays a role in this difference, as it is more common in the latter species than in the former; orangutans and bonobos have never been reported to commit infanticide in the wild. However, infanticide is an insufficient explanation as it affects about 5.5% of mountain gorilla infants born (Robbins et al. 2013) and somewhat fewer chimpanzees (Wrangham, Wilson, and Muller 2006; Wood et al. 2017) [n.b. in both species, rates are highly variable over time, even within populations]. In Grauer's gorillas at Kahuzi-Biega, rates of first-year mortality are estimated at 20% despite a rarity of infanticide (Yamagiwa et al. 2012). Mortality rates decline after the first year in all populations studied except for the Taï community of West African chimpanzees, which has

been affected by periods of high infant and juvenile mortality from leopard predation and the Ebola virus (Boesch and Boesch-Achermann 2000). Thus, while human mortality in the first year of life falls within the range of other great apes, the rate of human survival to adulthood is considerably higher. Anywhere from 25% to over 80% of great apes in wild populations die before reaching reproductive maturity. This high sensitivity of survival in early life is particularly important for population viability and of critical concern given that many of the long-term study groups that have yielded demographic data are assumed to be relatively protected from conservation threats compared to nearby groups/populations.

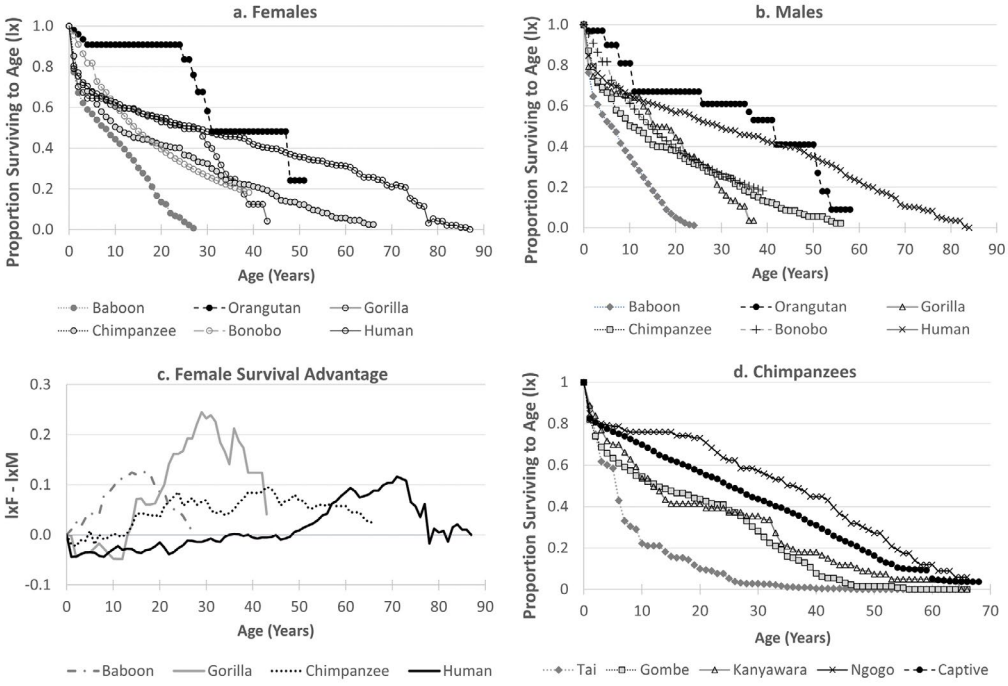

Figure 1

Fig. 1 Comparative survival data from hominid life tables, indicating proportion of individuals born surviving to age x (l_x). (a) Survivorship of females; (b) survivorship of males; (c) excess survivorship of females, calculated as female l_x − male l_x, (d) survivorship of chimpanzees (males and females combined), illustrating differences among captives and four wild populations. Baboon and human forager curves are shown for contrast. Age-specific survival/mortality were not available for bonobos past age 7 but were approximated from mortality rates by age-class and were truncated at age 40 because long-term survival is unknown. Bonobo and orangutan data excluded from sex comparison because adult mortality is based on poor samples and age estimates (see text). Human and chimpanzee curves in a, b, and c show the mean of several populations. Sources: baboons (*Papio cynocephalus*, Amboseli: Bronikowski et al. 2016a,b); orangutans (females ages 0–17, *Pongo spp*.: van Noordwijk et al. 2018; males and females aged 18+: *Pongo abelii*, Ketambe: Wich et al. 2004); mountain gorillas (*Gorilla beringei beringei*, Karisoke: Bronikowski et al. 2016a,b); wild chimpanzees (*Pan troglodytes schweinfurthii*, Gombe: Bronikowski et al. 2016a,b; Kanyawara: Muller & Wrangham 2014; Ngogo: Wood et al. 2017; *P. t. verus*, Taï: Boesch & Boesch-Achermann 2000); captive chimpanzees, averaged from United States and Europe (Dyke et al. 1995), Japan (Havercamp et al. 2019); bonobos: (Wamba: Furuichi et al. 1998); Human foragers (Ache, forest period: Hill & Hurtado 1996; Hadza: Blurton-Jones 2016; Hiwi, pre-contact: Hill et al. 2007; *n.b.* !Kung San mortality data omitted because not differentiated by sex).

Mortality in the first year of life is similar between the sexes, or slightly skewed towards females, while excess male mortality emerges in adolescence and adulthood. Prime adulthood, between the ages of approximately 15 and 30 years, is characterized by low rates of mortality, with some populations experiencing rates as low as those observed in human foraging populations (Wich et al. 2004; Muller and Wrangham 2014; Wood et al. 2017). Humans' increased longevity arises not from consistently lower mortality rates across adulthood but from a prolongation of the low mortality period. In the other great apes, mortality rates accelerate at approximately 25 years of age, while this take-off point is delayed by 20–30 years in human populations. A recent analysis of life tables in several primate species, including humans, East African chimpanzees, and mountain gorillas, concluded that there was no strong phylogenetic signal in Gompertz-derived adult mortality parameters, given that hominids fell within the continuum of other primates (Bronikowski et al. 2011). However, these data also show that hominids cluster at the low end of the continuum, with lower rates of initial mortality (i.e. mortality at onset of adulthood) and rates of aging (acceleration of age-specific mortality rate) than most other primate species.

At this point, life tables for wild apes remain sparse and may be vulnerable to stochastic variation, so caution should be used in interpreting variation among species. There are two major impediments to constructing life tables on wild great apes: age estimation and unrecognized mortality. For older individuals, who were adults when first identified by researchers, ages may be mis-estimated by 10 or more years. These oldest individuals have a relatively minor influence on the shape of survival functions, as the number of individuals whose ages are known with negligible error increases with research time. However, age estimation is a more serious problem for the dispersing sex, particularly if dispersal occurs multiple times or across a wide range of ages. Dispersal also creates the possibility for unrecognized mortality, as it may not be possible for dispersal events to be distinguished reliably from mortality events. Unrecognized mortality may also occur if rates of observation for some individuals are low. The latter is particularly a problem for identifying early infant mortality. These problems will gradually improve as researchers use genetic information to track individuals over larger study areas and monitor health and reproductive status of subjects.

Orangutans present a particular challenge, as males may experience delayed secondary sexual development, which makes it difficult to assign ages based on appearance alone (Utami et al. 2002). Sexual development is accompanied by dramatic physical changes and can occur rapidly, which also prevents researchers from recognizing an individual post-transition. Additionally, orangutan males often disperse over great distances and many remain transients for significant periods of time (van Schaik 1999; Nietlisbach et al. 2012). This creates significant uncertainty about the ages of new individuals entering the study area, as well as about the mortality status of individuals who disappear. These problems are noted by the authors of the only wild orangutan adult life table (Wich et al. 2004), which shows similar or higher survival in the wild than in captivity (Anderson et al. 2008; Wich, Shumaker, et al. 2009) or even, for early adulthood, human forager populations (Figure 1). This could be an artifact of having a small sample from a period of relative stability. Two features of the life table are cause for concern. First, the distribution of risk years exhibits distinct bulges at the author-defined age categories. For example, flanged males were assigned an age of 35 years when first encountered, thus the life table contains fourteen males aged 35 years, twice as many as were observed at age 34 and more than were observed at any age after 4 years. This could be possible if the

Ketambe study site was a particularly attractive place for flanged males to mate and feed, and thus experienced an influx, but it would then be a poor representation of the broader study population. A second concern is that Ketambe males were reported to have substantially higher survival than females. There is a general expectation for a male-bias in mortality, particularly in species experiencing strong sexual selection for male body mass and polygynous mating effort (Clutton-Brock 1991; Clinton and Le Boeuf 1993; Allman et al. 1998; Colchero et al. 2016). In captivity, male orangutans exhibit much lower survival than females (Anderson et al. 2008; Wich, Shumaker, et al. 2009). It is notable that the biases imposed by dispersal and transience are expected to be most relevant to males, thus these results should be taken with caution. Despite the limitations of this sample, it can be clearly concluded that orangutans experience low mortality rates and can achieve lifespans at least comparable to those of other great apes.

Both sexes of gorillas disperse and may occupy multiple groups over their lifetimes (Tutin 1996; Yamagiwa and Kahekwa 2001; Harcourt and Stewart 2007), thus ages can be difficult to estimate unless a large number of groups can be monitored. Since starting with three groups of mountain gorillas in the 1960s, research at Karisoke has expanded to include twenty groups, and their work provides the only published wild gorilla life table (Bronikowski et al. 2016a, 2016b). While the research centre can only study the groups of mountain gorillas ranging on the Rwandan side of the Virunga Mountains, these groups range within a fairly restricted area and are geographically isolated from most other gorilla groups (Gray et al. 2013), thus the study population comprises the majority of groups that individuals could move between, and demographic data carry a high degree of confidence. Other study sites are more constrained. For example, the Mbeli Bai site monitoring western lowland gorillas affords the opportunity to observe a large number of unhabituated groups (>60) but is limited to making long-distance observations from a platform overlooking a 12.9 ha swampy clearing (Breuer et al. 2009). Thus, little is known about the variation in mortality across gorilla populations or species.

In chimpanzees, males remain in their community for life and females disperse and reproduce within a fairly narrow age range, so age estimation is a fairly minor problem after a couple of decades of research. Accordingly, several life tables exist for chimpanzees. All but one of these life tables comprises the East African subspecies. As noted above, the West African population at Taï has had a particularly devastating recent history, reflected in mortality rates that are not viable over the long-term (Boesch and Boesch-Achermann 2000; Köndgen et al. 2008). By contrast, a recent report from East African chimpanzees in the Ngogo community describes remarkable survivorship, exceeding that of captive chimpanzees (Wood et al. 2017). This study concluded that under favourable ecological circumstances, including high food availability and relative protection from human disturbance, wild chimpanzees may experience mortality rates as low or lower than those of human hunter-gatherers until the age of about 40. There are good reasons to believe that the Ngogo community would have reduced mortality compared to other chimpanzee communities. They are relatively isolated from human settlements that can be a source of disease, and they are not hunted by humans or other predators. The Ngogo chimpanzees have access to more fruit (Potts, Watts, and Wrangham 2011) and maintain higher energy balance than the nearby Kanyawara community (Emery Thompson et al. 2009), which itself exhibits relatively high adult survival (Muller and Wrangham 2014). They may indeed exemplify the best-case scenario for wild chimpanzees, but one that would appear to generate growth rates too high to be stable over the long-term. However, these specific survival estimates

should be treated with caution due to peculiarities in the life table. For example, the survival function predicts that 20% of females ever born should live past the age of 60 years, while only 4 (in a community numbering 115 females) ever have. Unrecognized mortality is a distinct and unavoidable possibility in this sample due to the unusually large size of the community. This may occur because it takes many years to habituate and positively identify all individuals (particularly females), during which time deaths may go unrecognized. Additionally, a large number of female migration events may obscure death in early adulthood. Given the relative rarity of mortality events recorded, even a small number of unrecorded deaths could substantially alter the survival functions.

Wild bonobo life table statistics are only available for a small number of juveniles, though overall mortality rates are provided for adults and for adolescent males (Furuichi et al. 1998). These were 0.040 and 0.046 deaths/year, respectively, over an entire 20-year study, which included periods of intensive poaching. During a stable period, mortality rates dropped to approximately 2%. This initial data on adult mortality falls within the range of variation reported for other great apes, but bonobos may experience a relatively low infant mortality rate.

The range of variation in mortality among wild great apes, and even just among chimpanzees (Figure 1D), is very large, and given that long-term studies have been conducted primarily in protected areas, may represent only a fraction of the variation seen in nature. Available mortality data do not reveal a strong species signal, and instead suggest the influence of localized ecological conditions, including habitat disturbance, predation, natural disasters, poaching, and epidemic diseases. These stochastic factors, along with temporal variation in risk of infanticide (e.g., periodic male takeovers in gorillas) and intraspecific violence (e.g., intercommunity conflict in chimpanzees), mean that rates of mortality can vary dramatically even within a population over time. For example, the Taï chimpanzees experienced a 15-fold decrease in survival between earlier and later periods of study (Boesch and Boesch-Achermann 2000). Given the tremendous influence of human activities on ape populations, it may not be possible to reconstruct the patterns of mortality characteristic of these species during their evolutionary histories. This is perhaps not so different from the demographic examinations of modern human foragers (Gurven and Kaplan 2007).

After decades of research on wild great apes, there is relatively little understanding of cause-specific mortality. Even in well-monitored groups, a large number of individuals simply disappear without ever being recovered. In rainforests, decomposition and scavenging occur so rapidly that full necropsies cannot be performed even a couple of days after death. Thus, we know primarily about causes of death that have been preceded by obvious signs. For example, respiratory diseases occur in distinct outbreaks and cause readily observable signs, so individuals that disappear after coughing and sneezing can safely be assumed to have died from the disease.

Predation: As large-bodied primates, great apes are less vulnerable to predation than are other species, and large predators have in many cases been extirpated from their habitats. While rare, predation does occur and is likely to have been a low but consistent source of mortality for all great apes. Leopards are the principal non-human predator for gorillas, chimpanzees, and bonobos (Boesch and Boesch-Achermann 2000; Henschel, Abernethy, and White 2005; D'Amour, Hohmann, and Fruth 2006; Harcourt and Stewart 2007; Klailova et al. 2012). During one 4-year period, leopard predation was the primary source of mortality for West African

chimpanzees at Taï, accounting for 39% of deaths (Boesch and Boesch-Achermann 2000). Older infants and juveniles appear to be targeted most frequently (Boesch and Boesch-Achermann 2000), but adults are also attacked and consumed (Henschel, Abernethy, and White 2005). Lions have killed chimpanzees during temporary incursions into the forest (Tsukahara 1993) and likely pose a threat for chimpanzees ranging in savannah habitats, though no such attacks have been observed (Stewart and Pruetz 2013). Both tigers and clouded leopards appear to prey upon wild orangutans (Rijksen 1978), and orangutans exhibit prolonged alarm responses to predators (Lameira et al. 2013), but tigers are absent from Borneo and the high degree of arboreality in orangutans likely provides them with protection.

Poaching: Predation by humans is a persistent threat to all great ape species. Commercial poaching for bushmeat consumption is most common in western and central Africa, where it can have catastrophic impacts on great ape populations (Walsh et al. 2003; Stiles et al. 2016). Even small-scale hunting, such as for local consumption or for traditional medicine, practices that have likely persisted for much longer, can have important effects on great ape populations (Meijaard et al. 2011). For example, densities of Bornean orangutans are better predicted by distance to the nearest hunting village than by logging intensity or ecological measures of habitat quality (Marshall et al. 2006). Even where great apes are not targeted for hunting, they are vulnerable to snares and traps set for other animals (Quiatt, Reynolds, and Stokes 2002), and they are killed when perceived to be a threat to crops or the safety of local villagers.

Trauma: Non-poaching trauma accounts for a considerable number of deaths. Infanticide is common in gorillas (Robbins et al. 2007a; Breuer et al. 2010; Hassell et al. 2017) and chimpanzees (Wilson and Wrangham 2003; Townsend et al. 2007). Chimpanzees also kill mature individuals during intergroup conflicts and even within their own communities, comprising approximately 1.4 deaths/100 individuals/year (Wrangham, Wilson, and Muller 2006). One-on-one conflicts among silverback gorillas and flanged male orangutans result in severe injury and death (Meder 1994; Knott 1998b). Falls from trees can also be fatal (Boesch and Boesch-Achermann 2000).

Infectious disease: Respiratory disease, frequently of human origin, is a persistent cause of mortality in African ape populations (Leendertz et al. 2006; Williams et al. 2008; Sakamaki, Mulavwa, and Furuich 2009; Humle 2011; Spelman et al. 2013). Outbreaks spread rapidly within groups, often producing signs in nearly 100% of individuals. Single outbreaks have been known to kill 10–20% of chimpanzees in a community within a matter of weeks (Kaur et al. 2008; Köndgen et al. 2008; Negrey et al. 2019). While old chimpanzees are most likely to exhibit respiratory signs, significant mortality occurs among all age classes except juveniles (Emery Thompson et al. 2018). Multiple outbreaks of Ebola haemorrhagic fever have caused catastrophic mortality among gorillas and chimpanzees in West Africa over the past 35 years (Leroy et al. 2004; Ryan and Walsh 2011). African apes have also experienced smaller-scale outbreaks of anthrax, polio, scabies, and measles (Leendertz et al. 2006; Williams et al. 2008; Hoffmann et al. 2017). A number of viruses (e.g., simian foamy virus) and intestinal parasites (e.g., *Oesophagostomum*, *Strongyloides*), along with malaria parasites, occur persistently in wild apes, but their long-term impact on health is not yet understood (Hasegawa, Kano, and Mulavwa 1983; Landsoud-Soukate, Tutin, and Fernandez 1995; Dupain et al. 2002; Lilly, Mehlman, and Doran 2002; Liu et al. 2008; Gillespie et al. 2010; Prugnolle et al. 2010). Chronic infections may play a role in the "wasting" illness observed in some older chimpanzees (Goodall 1986; Nishida et al. 2003; Terio et al. 2011). Simian immunodeficiency virus (SIV), a retrovirus

related to HIV, occurs in some populations of chimpanzees and gorillas, but appears absent in bonobos (Santiago et al. 2002; Van Dooren et al. 2002; Van Heuverswyn et al. 2006). Among Gombe chimpanzees, the effects of SIV are not easily observed, but infected individuals have significantly reduced long-term survivorship (Keele et al. 2009). While wild orangutans are known to be infected by many of the pathogens impacting other apes (Kilbourn et al. 2003), no information is available on cause-specific mortality.

Degenerative Disease: Degenerative diseases, such as cancer, heart disease, and kidney disease, are expected to be major sources of adult mortality in long-lived species. A large number of unexplained deaths due to "old age" might suggest to be the case for great apes, but evidence of these processes has proved elusive. Heart disease is the largest cause of mortality among captive great apes (Lowenstine, McManamon, and Terio 2015; Strong et al. 2016; Laurence et al. 2017). However, atherosclerosis, arteriosclerosis, and associated coronary artery disease, the processes underlying most aging-associated cardiovascular deaths in humans, are very rare in great apes. Instead, captive great apes suffer from aortic dissections and myocardial fibrosis (Lammey et al. 2008; Varki et al. 2009; Lowenstine, McManamon, and Terio 2015). Evidence from a limited number of necropsies suggests that these or other cardiovascular processes are not significant sources of mortality in wild chimpanzees (Terio et al. 2011) or mountain gorillas (Nutter et al. 2005; Cooper 2017). Type II diabetes is rare even in well-fed captive apes (Kuhar, Bettinger, and Laudenslager 2005; Lowenstine, McManamon, and Terio 2015). Renal disease occurs in captive great apes, but its presence in the wild is unconfirmed (Lowenstine, McManamon, and Terio 2015; Strong et al. 2016). Malignant neoplasms occur but appear relatively rare in both wild and captive apes compared to humans (Lowenstine, McManamon, and Terio 2015; Laurence et al. 2017). Perhaps these would be found if more necropsies were done, but genetic evidence suggests that great apes have an increased capacity for cell apoptosis compared to humans (Arora, Mezencev, and McDonald 2012). Amyloid plaques and neuron loss have been identified in the brains of older captive great apes, but the full suite of signs of Alzheimer's disease have not (Finch and Austad 2015, Edler et al. 2020).

Growth and Development

Postnatal Growth

A key feature of primate life histories is relatively slow growth compared with other mammals. Allometric comparisons across taxa suggest that primates as a group are constrained by a rate of production — energy available for growth and/or reproduction — of about 42% that of other mammals (Charnov and Berrigan 1993). The net result is prolonged infant dependence, a slow rate of reproduction, and a delay until attainment of full adult size and reproductive maturity. Slow growth may be advantageous, allowing primates to learn complex foraging (Kaplan et al. 2000) or social skills (Joffe 1997) before they enter the adult world, or it may simply be less risky for juveniles than to assume the costs of rapid growth in competitive or unstable environments (Janson and van Schaik 1993). Alternatively, somatic growth rates may be constrained by the costs of building a large brain (Foley et al. 1991; Ross and Jones 1999). The latter has convincing, direct support for humans, who experience an unusually slow rate of early growth. Growth rates are slowest when the metabolic demands of brain growth are highest, and the subsequent adolescent growth "spurt" is delayed until the costs of the brain decline (Kuzawa et al. 2014).

Comparisons across primates suggest that variation in growth is not simply a product of how long it takes to reach a particular body size, but as in humans, manifests in complex variation in the timing and speed of successive growth stages (Leigh 2001).

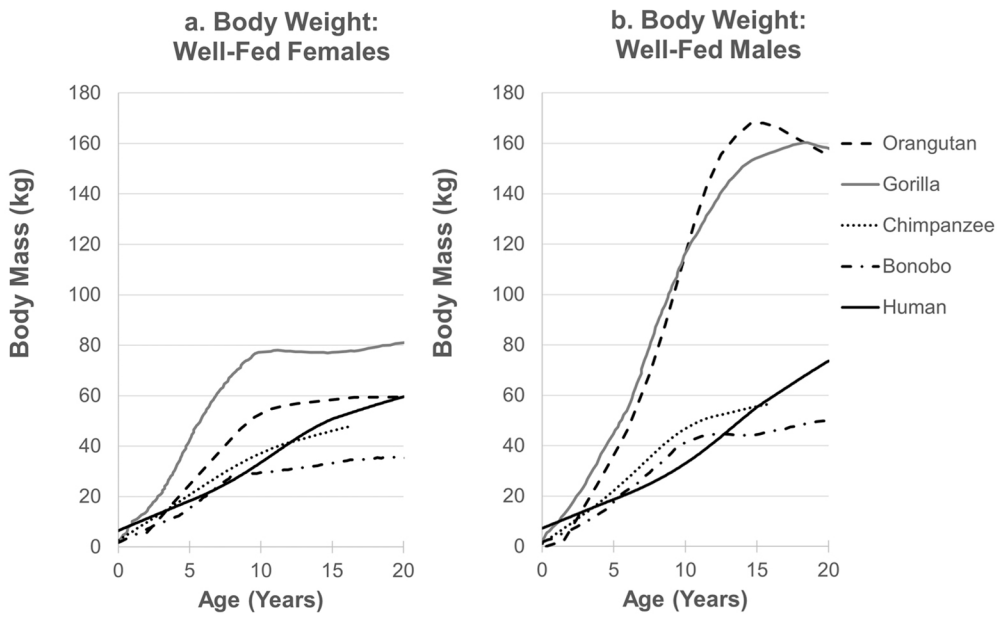

Figure 2.

Fig. 2 Comparative hominid growth. Growth in body mass among male (a) and female (b) hominids in well-fed populations, loess-smoothed data. Data sources: orangutans (*Pongo spp*., N = 24, developmentally arrested males excluded; Fooden and Izor 1983); gorillas (*Gorilla gorilla*, N = 72F, 64M: Leigh and Shea 1996); chimpanzees (*Pan troglodytes*, N = 56F, 49M: Hamada and Udono 2002); bonobos (*P. paniscus*, N = 13F, 23M: Leigh and Shea 1996); humans (US, CDC, N >500,000: Kuczmarski et al. 2002). Where necessary, raw data acquired via WebPlotDigitizer (v. 3.9, Ankit Rohatgi).

Comparative data on growth in great apes, as in other primates, has primarily been generated from captive populations. This is important because the stable nutritional base in captivity accelerates both growth and sexual maturation and may do so differently depending on the natural diet. Captive data have other disadvantages. For example, increased fat deposition in sedentary captive colonies can lead to less robust correlations between body mass and growth in stature, and reproductive interventions can obscure the trade-off between reproduction and growth. As a general rule, differences in adult body mass among great ape species are due to differences in the rate of early growth, whereas sexual dimorphism within species results primarily from differences in the duration of growth and the magnitude of the growth spurt (Figure 2, Fooden and Izor 1983; Shea 1983; Leigh and Shea 1996). Gorilla growth rates start higher and accelerate more rapidly than either species of *Pan*, reaching peak velocities more than twice as great (Table 2). Chimpanzees grow slightly faster than bonobos during the early growth period, but chimpanzees grow for longer before reaching peak velocity. Sex differences in growth rates are minimal until adolescence, when male rates accelerate. Whereas male apes exhibit distinct adolescent growth spurts in weight, female growth spurts are notable in gorillas

and bonobos but indistinct in orangutans and chimpanzees (Leigh 1996). Growth in mass for most apes reaches an asymptote by approximately 10–15 years, except for male gorillas and orangutans who may grow for substantially longer. All of the great ape species grow faster during the early growth period than do humans and reach an earlier peak velocity. Thus, humans are exceptional both in how slowly and for how long they grow, and these characteristics are not readily explained by adult body mass (Leigh 2001).

Growth data from wild apes are sparse and differences in analytical approaches makes them difficult to compare directly to captives. Most estimates of wild body mass and length come from animals shot for museum collections, which may be a biased sample and may have errors in weight estimation. Based on the available data, the effect of captivity on adult body mass varies substantially. Orangutans are about 60% heavier, while gorillas and bonobos are of similar mass in both contexts (Table 2). Comparative growth data on living animals are available only for chimpanzees; wild chimpanzees at Gombe National Park were weighed by baiting hanging scales with food (Pusey et al. 2005). Differences in growth between Gombe and captive chimpanzees are apparent from infancy and result in an approximate 1/3 reduction in body mass (n.b., Gombe chimpanzees are small compared with other wild chimpanzees: Emery Thompson and Wrangham 2013), a difference that parallels that between human foragers and those in developed countries (Figure 3). However, wild individuals reach their full adult mass at approximately the same age as do captive individuals. Sexual dimorphism is not conspicuously enhanced with captivity for any of the great apes.

Brain growth is not well characterized in great apes. The brains of captive chimpanzees grow at a substantially lower rate than humans, and the marked difference in size is near fully realized in the first year (Leigh 2004). Growth rates subsequently decelerate rapidly such that chimpanzees complete their brain growth at about the same age, or slightly earlier (4–7 years), than do humans (5–7 years) (Herndon et al. 1999; Leigh 2004; Robson and Wood 2008; Coqueugniot and Hublin 2012; Neubauer et al. 2012). Wild mountain gorillas reach adult brain size by only age 3–4 years (McFarlin et al. 2013) The significance of adult brain mass differences among great apes is unclear. Early efforts to compare brain size across species emphasized encephalization quotients that account for body size (chimpanzees > orangutans > gorillas, Marino 1998), while recent studies suggest that absolute size (gorillas > chimpanzees = orangutans > bonobos, Table 2) is most relevant to cognition (Reader and Laland 2002). Phylogenetic analyses indicate a dissociation between brain size and body size during primate evolution (Montgomery et al. 2010). New analyses using neuronal cell counts indicate that brains of primates, including all hominids, follow a common isometric scaling pattern (Herculano-Houzel and Kaas 2011). An interesting conclusion of this work is that while human brains are extraordinary in their absolute size, sophistication, and cost, they do not actually have larger brains than expected for primates of their size (Azevedo et al. 2009; Herculano-Houzel 2012). Instead, it is the other great apes that are outliers, with the evolution of large body mass outpacing selection on the brain (Herculano-Houzel and Kaas 2011).

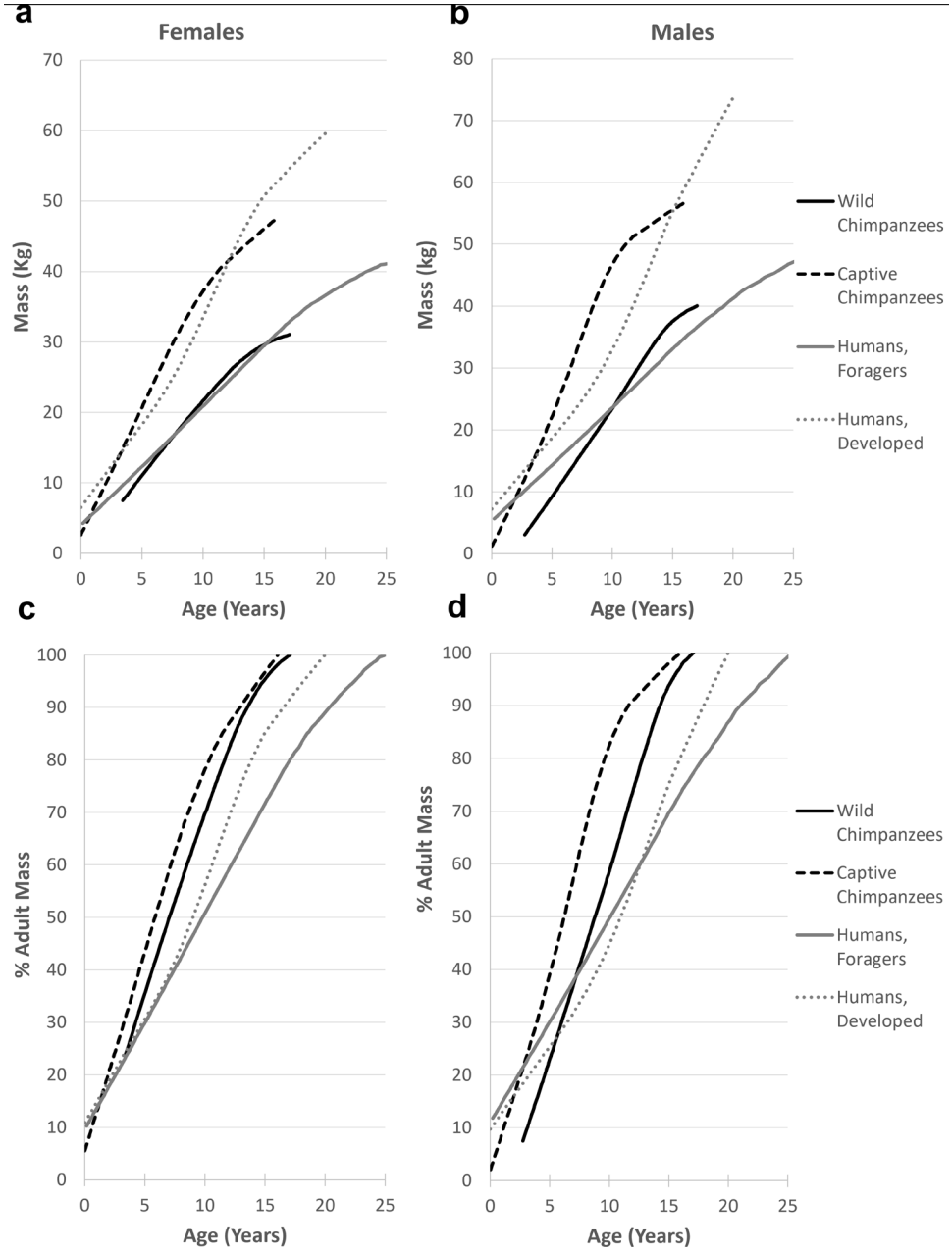

Figure 3.

Fig. 3 Growth in body mass among chimpanzees and humans under subsistence and well-fed conditions, loess smoothed data: (a,c) females, (b,d) males. Panels a, b depict absolute growth in body mass, emphasizing the intraspecific effect of energy availability. Panels c, d depict growth relative to adult body mass, emphasizing interspecific effects. Data sources: wild chimpanzees, *Pan troglodytes schweinfurthii* (Gombe, N = 26F, 31M: Pusey et al. 2005); captive chimpanzees, *P. t. verus* (Sanwa-Kagaku Kumamoto Primate Park, N = 56F, 49M: Hamada and Udono 2002); human foragers (Dobe !Kung: Howell 2010); humans, developed nation (U.S., CDC, >50,000: Kuczmarski et al. 2002). CDC data available to age 20, captive chimpanzees to age 16, !Kung and Gombe data truncated at ~95% adult weight (ages 25, 17 respectively). Where necessary, raw data acquired via WebPlotDigitizer (v. 3.9, Ankit Rohatgi).

Weaning and Nutritional Independence

While age of weaning is considered a critical developmental milestone across species, it is clear that its significance has changed in humans due to the availability of cooked or processed weaning foods and contributions to infant provisioning by fathers and alloparents. Humans consequently wean infants at a relatively young age compared to other hominids despite longer periods of nutritional dependence and maturation (Kennedy 2005). Observations from the other great apes indicate that the weaning ages correspond more clearly with the expectation of later weaning associated with slower developmental trajectories (Table 3). That is, all of the great apes nurse their infants for longer periods in the wild than do other primates. Differences among the apes correspond to later features of maturation: weaning is earliest in the Karisoke population of mountain gorillas (3.3 years) and latest in orangutans (6–8 years).

There are two major problems with defining and comparing ages of weaning in wild hominids. The first is the reliability of measurement. Nipple contact is difficult to observe and quantify, as a lot of nursing may occur out of view while high in trees or during the night. It is also not clear how well nipple contact times approximate actual milk ingestion. A second issue emerging from recent studies is that age of last nipple contact, the classic definition of weaning age, appears to be a poor indicator of relative infant development or maternal nutritional support (van Noordwijk, Kuzawa, and van Schaik 2013; Bădescu et al. 2017). In many smaller primates, the transition from exclusive maternal nutritional support to completely independent infant foraging occurs relatively rapidly at a stage of growth at which the infant's nutritional needs outstrip the mother's ability to accommodate them (Lee, Majluf, and Gordon 1991; Lee 1996). In these species, weaning is marked by overt maternal rejection and mother-infant conflict, and reliably precedes the resumption of maternal cycling, often corresponding to seasonal patterns of resource availability. For hominids, the increased energetic requirements of larger bodies and brains necessitates multiple years of lactation, including a substantial period where both mothers and infants need to contribute to meet the costs of growth (van Noordwijk, Kuzawa, and van Schaik 2013; van Noordwijk et al. 2013).

Studies of developmental processes in apes have just begun to gain traction, and these studies indicate that the multi-year lactation pattern is fundamentally different from the "classic" primate model (van Noordwijk et al. 2013). Hominid lactation comprises 3 major phases. The period of exclusive lactation lasts only a few months, with first ingestion of solid food occurring at similar ages as in other primates. Subsequently, infants continue to nurse intensively, while using supplemental foods, until their second or third year of life, at which time they undergo a marked nutritional transition to independent feeding. This transition comprises steep increases in infant time spent independently foraging (van Noordwijk et al. 2013; Matsumoto 2017; Bray et al. 2018) and decreases in time spent suckling (Clark 1977; Stewart 1988; Nowell and Fletcher 2007). The energetic relevance of these transitions has recently been confirmed using stable isotope ratios, which comprise signatures of milk ingestion or of dietary convergence by mother and infant (Reitsema 2012). For example, mother and infant chimpanzees have high divergence of carbon and nitrogen isotopes in their faeces until the infant reaches approximately 18 months and begin to converge thereafter (Bădescu et al. 2017). Dental nitrogen isotopes from deceased juvenile chimpanzees tell a similar story (Fahy et al. 2014). Urinary C-peptide of insulin levels have been used to track changes in the energetic costs of lactation for chimpanzee mothers, and these profiles also indicate a transition to a reduced load at approximately 2

years postpartum (Emery Thompson, Muller, and Wrangham 2012). This transition is most functionally equivalent to weaning in other primates, though it occurs later. However, most hominids do not fully wean until they are between 3 and 8 years old, suggesting a third stage wherein infants can provide most of their own calories but continue to use breastmilk for supplemental nutrition. It is unclear how consistently infants use breastmilk after this point, or how reliant they are on milk for survival. Dental barium isotopes from the teeth of orangutans, which have the latest weaning ages of any mammal, indicate that the pattern of milk ingestion fluctuates dramatically, with significant peaks in milk consumption during periods of food scarcity (Smith et al. 2017). Isotopic signatures of nursing disappear by about 4 years in wild chimpanzees, though infants are observed nursing for 5–6 years (Bădescu et al. 2017). Some wild chimpanzees can survive, albeit at a disadvantage, if they are weaned or even orphaned in their third year (Boesch et al. 2009; Emery Thompson et al. 2016), even though most continue to suckle for 2–4 years longer. While mountain gorilla infants can be weaned anywhere between ~2–5 years of age, final weaning age has had little impact on survival (Eckardt, Fawcett, and Fletcher 2016). Thus, for infants, whose costs of growth and competitive disadvantage make them particularly vulnerable to fluctuations in food supply (Janson and van Schaik 1993), mother's milk may not be a consistent need but an important buffer. This may help explain why mountain gorillas complete weaning substantially younger than other great apes. The low-quality herbaceous vegetation that comprises the mountain gorilla diet might seem less than ideal for infants, but it is consistently and abundantly available and requires relatively little learning of selection or processing techniques. Bamboo shoots, in particular, are soft and high in energy yield and seem to be ideal weaning foods (Grueter et al. 2014; Eckardt, Fawcett, and Fletcher 2016). On the other hand, orangutans experience the most drastic fluctuations in food supply (Conklin-Brittain, Knott, and Wrangham 2006) and continue to use supplemental nursing for the longest of any of the hominids.

The classic model of primate reproduction emphasizes the role of nursing, and consequently of weaning, as an adaptive regulator of birth spacing. As mothers are predicted to reproduce again only when their infants are sufficiently developed and capable of foraging independently, nursing itself is thought to suppress maternal reproduction. There is even a well-known mechanism for this. The hormone prolactin, produced by the mother's pituitary in response to the suckling stimulus, directly acts to suppress ovarian cycling. Under this model, infants exert considerable influence over maternal reproductive schedules, though mothers can push back by rejecting infants from the nipple at an earlier age than they might like. However, in humans it has become clear that even very intensive nursing practices may not suppress maternal cycling if the mother has abundant energy available to her (Ellison and Valeggia 2003; Valeggia and Ellison 2004; Valeggia and Ellison 2009), chiefly because the prolactin response to suckling is blunted in well-nourished mothers (Lunn et al. 1984). Instead, reproductive timing is determined by the ability of a mother to recover the energetic losses of pregnancy and lactation. While intensive breastfeeding detracts from maternal energy balance, breastfeeding alone, particularly subsequent to the introduction of supplemental foods, may not comprise a sufficient "metabolic load" to prevent the resumption of cycling (Ellison and Valeggia 2003; Valeggia and Ellison 2004). It is thus observed that many women resume cycling or even conceive while still nursing their previous infant (Gioiosa 1955; Merchant, Martorell, and Haas 1990; Moscone and Moore 1993). Similarly, weaning is not a prerequisite for the resumption

of cycling in great apes, and it appears that infants habitually suckle into their mothers' next pregnancy (orangutans: van Noordwijk et al. 2013; gorillas: Eckardt, Fawcett, and Fletcher 2016; Robbins and Robbins 2021; chimpanzees: personal observation). The final age of weaning, therefore, is determined by the mother's reproductive pattern rather than the infant's age or relative development. For example, wild chimpanzees who were forced to wean early due to a mother's pregnancy had a significant growth disadvantage relative to those whose mothers reproduced slowly (Emery Thompson et al. 2016). Thus, species variation in final weaning ages make sense in the scheme of other life history variation, but individual weaning ages may be poor indicators of development. Weaned apes are often well past the age at which they can feed themselves but well before the age when benefits of mother's milk are exhausted.

In both chimpanzees (Lonsdorf et al. 2020) and mountain gorillas (Robbins and Robbins 2021), later weaning ages have been reported male infants than for female infants. Maternal rank did not affect weaning age in the mountain gorillas. Among Gombe chimpanzees, high ranking mothers had both the earliest and latest ages of weaning (Lonsdorf et al. 2020). This pattern likely reflects two processes, one where improved condition drives faster reproduction among high-ranking females (forcing earlier weaning), and another where rank is increasing with age as fertility declines (high terminal investment).

Behavioural Development

Great apes experience many years of further growth and development after becoming nutritionally independent from their mothers. This juvenile period, defined as the interval between weaning and sexual maturity (Pagel and Harvey 1993), is greatly extended in primates compared to many other species, even after accounting for differences in adult body size (Charnov and Berrigan 1993). Skeletal, cognitive, endocrine, and sexual maturation proceed across this period. As in humans, great ape juvenility is a time of continued close proximity to mothers, frequent play, and development of foraging expertise and social relationships. The great apes also exhibit a distinct and lengthy period of adolescence, the interval between the first signs of puberty and the attainment of full adult size and adult social and breeding status. This period comprises increasing independence from the mother, initiation of sexual behaviour, slowly increasing fecundity, elaboration of secondary sexual characteristics, participation in status competition, and (depending on sex) dispersal into new groups.

Orangutans cling to their mothers almost exclusively for the first 2 years of life, and by 4 years they spend little time in direct contact with their mothers (van Noordwijk et al. 2009). They continue to spend almost all of their time within 10m of their mothers until approximately 6–8 years, and rarely associate with their mothers after age 9–10 years (van Noordwijk et al. 2009; van Adrichem et al. 2006). In Western gorillas, infants are in direct contact with mothers for 1–1.5 years and cling very rarely after approximately age 3.5 years (Nowell and Fletcher 2007). By that age, they are more apt to wander away from their mothers than are orangutans, but they continue to spend at least 50% of their time within 10m of their mothers until about age 6 years (Nowell and Fletcher 2007). Mountain gorillas reduce spatial proximity with their mothers more rapidly. They are in contact almost exclusively for the first 6 months of life, with very low rates of contact after 2 years (Fletcher 2001). Individuals begin wandering more than 5m away by age 1 and spent less than half their time within 5m by 2.5 years (Fletcher 2001). Chimpanzee infants are in contact almost exclusively until about 7–9 months (van de Rijt-Plooij and Plooij

1987). Males begin traveling in different subgroups from their mothers at about 7–8 years of age, and by ages 12–14 spend most of their time with adult males, though there is considerable variation in this (Pusey 1983, 1990a; Machanda et al. 2014). Females, who typically disperse and will not form adult relationships with other members of their natal group, continue to associate in the same subgroup as their mothers at high rates until they emigrate (Machanda et al. 2014). When individuals of either sex are in the same subgroup with their mothers, they tended to remain in close proximity (<15 m) with her until at least 10 years of age (Pusey 1983).

Though individual differences in attainment of behavioural milestones may reflect differences in growth or maturation, the difference between species likely has more to do with socioecological factors. Given their high risk of infanticide from outside males, it is somewhat surprising that mountain gorillas leave the safety of their mothers relatively early and often compared with other hominids. However, even in the first year of life, immature gorillas seek proximity with silverback males, who offer the most effective protection (Stewart 2001; Rosenbaum, Silk, and Stoinski 2011).Differences in mother-offspring distance across species covary with group cohesion — where groups as a whole exhibit tighter proximity, as in mountain gorillas, infants and juveniles spend less time in close proximity with their mothers. These differences also covary with dietary complexity, perhaps suggesting differences in the importance of staying near mothers for observational learning of foraging. In gorillas, where low-quality forage is easily acquired, infants may have less need to observe their mothers' food choices and processing behaviour. Orangutans, on the other hand, rely on a wide range of foods to buffer periods of food scarcity, and infants carefully observe their mothers in foraging contexts (Schuppli, Forss, et al. 2016; Schuppli, Meulman, et al. 2016).

Dispersal

In great apes, as in most species, one or both sexes tend to disperse from their natal groups around the time of sexual maturity, but dispersal patterns vary considerably. Dispersal is thought to serve a primary function in reducing the potential for inbreeding (Pusey 1987, 1990b). While individuals also use mate choice behaviours to avoid mating with close kin (Pusey 1980), these mechanisms can fail if a male relative is sexually coercive or if paternal kin are not well recognized (Walker et al. 2017). The diversity of dispersal patterns among great apes suggests that emigration serves additional or alternative functions, such as mitigating competition among relatives or providing access to better breeding opportunities.

Both bonobos and chimpanzees exhibit clear female-biased dispersal whereby most, but not all, females leave their natal groups around reproductive maturity, while males remain in their natal groups for life (Nishida 1979; Furuichi 1989; Eriksson et al. 2006). Female chimpanzees almost always emigrate after starting their sexual swelling cycles and having mated with males in their natal communities (Pusey 1980; Stumpf et al. 2009), and the swelling itself may serve as a kind of "passport" to facilitate acceptance by males in a new group (Nishida 1979). Most chimpanzee females emigrate between ages 10 and 15 (Emery Thompson 2013b; Wittig and Boesch 2019), while bonobos emigrate between ages 6 and 10 (Furuichi et al. 2012). Females may emigrate immediately after starting their swelling cycles or may be delayed for as much as 2 years. Dispersal timing in female chimpanzees in Kibale National Park was not predicted by gynaecological age or elevations in glucocorticoid levels, but tended to occur during periods of high diet quality, suggesting that females emigrated when they had energy to spare (Stumpf

et al. 2009). The majority of females transfer only once and do so permanently. However, female chimpanzees and bonobos may visit several communities before settling or leave their natal group temporarily and return to breed later (Pusey 1980; Goodall 1986; Furuichi et al. 2012), and rare secondary dispersal by adult female chimpanzees occurs (Nishida, Takasaki, and Takahata 1990; Emery Thompson, Newton-Fisher, and Reynolds 2006; Walker et al. 2017). In both chimpanzees and bonobos, male dispersal is rare due to hostile intergroup relations, but isolated (some temporary) male immigration events have been observed (Sugiyama 1999; Hohmann 2001; Sugiyama 2004; Furuichi et al. 2012).

Gorillas exhibit bisexual dispersal: either sex may disperse or may be philopatric (Douadi et al. 2007; Harcourt and Stewart 2007; Robbins et al. 2009). Dispersal of males appears to be dependent on breeding opportunities. Mountain gorilla males generally leave their natal groups between ages 13 and 21, after the saddles on their backs turn grey and they have reached, or nearly reached, their full adult size (Stoinski et al. 2009). They often range temporarily in bachelor groups until they are able to acquire females to establish their own groups. Some mountain gorillas remain in their natal groups by inheriting the dominant silverback position after the former silverback's death or by maturing into a position as a secondary male (Watts 2000; Robbins and Robbins 2005). In Eastern lowland gorillas, which have uni-male groups, males emigrate relatively early at 10–14 years (Yamagiwa and Kahekwa 2001). Female gorillas mature faster and leave their natal groups earlier than males: between 8–10 years in the Western species (Robbins et al. 2004), 7.5 to 15.3 years in eastern lowland gorillas (Yamagiwa and Kahekwa 2001), and approximately 8 years in mountain gorillas (Harcourt and Stewart 1978). Unlike chimpanzees, gorilla females may exhibit secondary or even tertiary dispersal, apparently to manage the risk of infanticide (Harcourt and Greenberg 2001; Stokes, Parnell, and Olejniczak 2003; Harcourt and Stewart 2007). Females may maintain kin associations by dispersing with female relatives or into groups with related individuals (Bradley, Doran-Sheehy, and Vigilant 2007).

Adult orangutans establish individual ranges, thus both sexes effectively disperse. However female orangutans can be viewed as the more philopatric sex because at least some females establish home ranges adjacent to or overlapping their mothers' and maintain friendly, albeit rare, interactions with maternal relatives (van Noordwijk et al. 2012; Ashbury et al. 2020). Males emigrate substantially farther than females (Nietlisbach et al. 2012; van Noordwijk et al. 2012), and some may continue to shift ranges throughout life (van Schaik 1999). Genetic evidence suggests that developmental arrest does not impact dispersal: even sexually-mature unflanged males are not found near related individuals (van Noordwijk et al. 2012).

Despite variation within and among great apes, as a group they depart fundamentally from the predominant pattern of male dispersal and strong female kin associations observed in cercopithecine primates. Available genetic and ethnographic evidence paint an uncertain view of sex-biased dispersal during human evolutionary history, with some studies supporting a history of female migration and others suggesting a considerably more complicated pattern (Seielstad, Minch, and Cavalli-Sforza 1998; Hammer et al. 2001; Marlowe 2004; Wilder et al. 2004; Langergraber et al. 2007). This likely emerges from high flexibility in residence patterns across human populations, including multilocality, and perhaps from different patterns of local versus long-range migration. While agriculture appears to be responsible for a recent strong

tendency for patrilocality (Marlowe 2004), the great ape data parsimoniously indicate that some degree of female dispersal would also have occurred in early hominin ancestors.

Sexual Maturation

Menstruation occurs in all great apes, but it is lighter than in humans and cannot be reliably detected in the wild. Among captive apes, menarche occurs at approximately age 8 years in orangutans, bonobos, and chimpanzees, and 6–7 in Western gorillas (Knott 2001). In female chimpanzees and bonobos, large oestrogen-dependent swellings of the anogenital region provide a conspicuous indication that ovarian cycles have begun (Dahl, Nadler, and Collins 1991). Fully tumescent swellings begin at approximately age 10–12 years in wild East African chimpanzees, after months or even years of small, sporadic swellings (Wallis 1997; Nishida et al. 2003; Stumpf et al. 2009; Walker et al. 2018). Earlier ages (8–9 years) are reported for small samples of West African chimpanzees at Bossou (Sugiyama 2004) and bonobos at Wamba (Kuroda 1989). Gorillas exhibit much smaller labial swellings that first emerge at approximately 6–8 years in wild mountain gorillas (Harcourt et al. 1980; Harcourt and Stewart 2007), but possibly not until 9–10 years in Western gorillas (Breuer et al. 2009). First cycling likely begins later in orangutans, which have no external indicators of ovulatory state, as first sexual behaviour does not occur until 10–14 years (Knott, Emery Thompson, and Wich 2009).

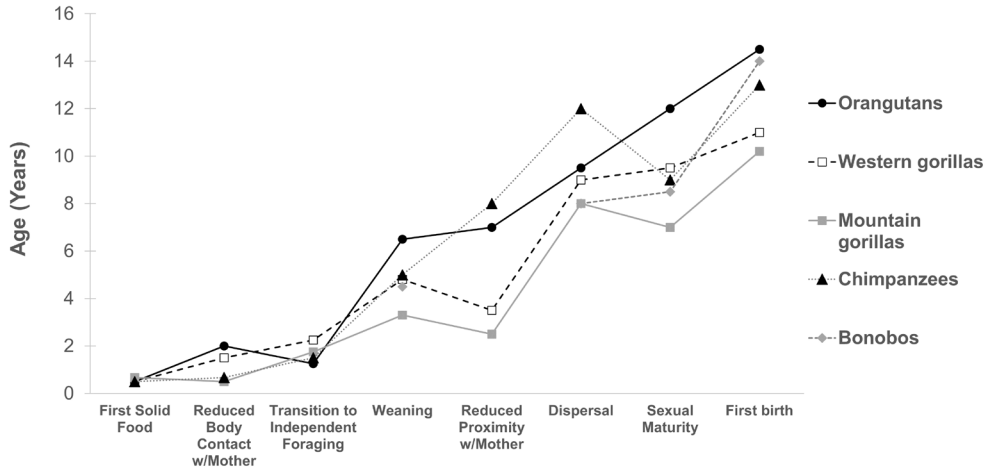

Figure 4.

Fig. 4 Maturation in hominids. Relative timing of foraging, social, and sexual development in wild great apes, showing approximate species averages for each milestone (data: see Tables 3 and 4). Note that data are missing for some events in bonobos. Mountain gorilla data shown are for Karisoke, where all milestones are available, though it should be noted that the second mountain gorilla population at Bwindi has later ages of weaning (Robbins and Robbins 2021).

Like humans, great ape females undergo substantial periods of adolescent subfecundity, defined as the interval between onset of sexual cycling and first conception (Montagu 1979). These data are difficult to acquire for individuals, as dispersal often interrupts the subfecund interval. As a result, the reports of true adolescent subfecundity based on natal individuals (approximately 1–1.5 years in mountain gorillas, 2.5 years in chimpanzees, and 4–5 years in orangutans, Knott 2001) underestimate the difference between mean age of menarche and first birth. In chimpanzees, for example, natal individuals give birth 1–2 years earlier than do immigrants (Nishida et al. 2003; Walker et al. 2018). This suggests that dispersing females may suffer a longer period of adolescent subfecundity.

Testicular enlargement occurs in wild chimpanzees at approximately 8–10 years (Goodall 1986; Pusey 1990a). First ejaculatory copulations occur at approximately age 9 years in the wild (Goodall 1986; Pusey 1990a; Sugiyama et al. 1993), compared with approximately 7 years in captivity (Marson et al. 1991). Like females, males undergo a long period of subfecundity, as sperm counts and viability increase (Marson et al. 1991). Comparable data are not available for bonobos, but puberty-associated increases in testosterone occur at the same age in captive chimpanzees and bonobos (Behringer et al. 2014). The testes of male gorillas are too small to be reliably evaluated in the wild (Harcourt 1995). The development of other adult characteristics (e.g., sagittal crest, silver hair) takes place between approximately 10 and 16 years in wild gorillas (Watts and Pusey 1993; Breuer et al. 2009). Male reproduction is typically constrained by the ability to become the dominant silverback, which occurs at a minimum age of 14 and average age of 17 (Breuer et al. 2009; Stoinski et al. 2009). Orangutan males exhibit two sexually-mature morphs (Rijksen 1978; Schürmann and van Hooff 1986; Utami et al. 2002). Some males may maintain an undeveloped ("unflanged") state, difficult to distinguish from the subadult phenotype, for up to 20 years after reaching sexual maturity. Fully "flanged" males exhibit pronounced cheek flanges, enlarged throat pouches, ropy hair, and increased body mass, and display pronounced differences in behaviour, such as in long-calling rates, activity budgets, and interactions with females (Utami Atmoko and van Hooff 2004; Knott et al. 2010). While flanged males produce higher levels of testosterone than unflanged males (Kingsley 1982; Maggioncalda, Sapolsky, and Czekala 1999), particularly if they developed early (Emery Thompson, Zhou, and Knott 2012), unflanged males are physiologically capable of siring offspring (Utami et al. 2002; Goossens et al. 2006). Given relatively late ages of first reproduction among captive orangutans compared with other great apes, it is reasonable to assume that wild orangutan males reach reproductive maturity late relative to their wild counterparts. As a general rule, great apes are similar to humans in that females exhibit outward signs of sexual maturity (e.g., sexual swellings) early in the pubertal transition and before the development of full fecundity, whereas males do not develop the physical appearance of adults until well after they are capable of siring offspring.

Figure 4 depicts the timing of behavioural development and sexual maturation across great apes. While these data indicate some consistent species differences in developmental rate, especially after weaning, they also indicate that species do not follow a consistent trajectory across modalities. For example, there is very little variation in when great apes ingest their first solid food or when they draw down their reliance on breastmilk, but greater variation in when offspring are finally weaned. While orangutans and mountain gorillas tend to fall at the upper

and lower extremes, respectively, at each time point chimpanzees deviate from the expected pattern by maintaining social cohesion with their mothers and natal groups for longer.

Female Reproduction

Fertility

First births to wild females occur at average ages of 10–12 years in gorillas, 10–14 years in chimpanzees and bonobos, and 14–16 years in orangutans (Table 4). These differences reflect an additive effect of interspecific differences in age of maturity and duration of subfecundity. In general, reproductive rates correlate with the interspecific and interpopulation variation in age of first birth (Figure 5, $r = 0.696$, $N = 14$, $p = 0.006$ for wild populations), with mountain gorillas exhibiting the fastest reproduction and orangutans the slowest. However, compared to the other species, most populations of chimpanzees and bonobos have faster reproductive rates than their late age of first reproduction should predict. This could, in part, reflect the costs of dispersal on delaying first reproduction in these species (Walker et al. 2018). The Bossou chimpanzee study group, where first births are known only for non-dispersing females, is an outlier for chimpanzees, but conforms to the predicted pattern for the other apes (Sugiyama and Fujita 2011).

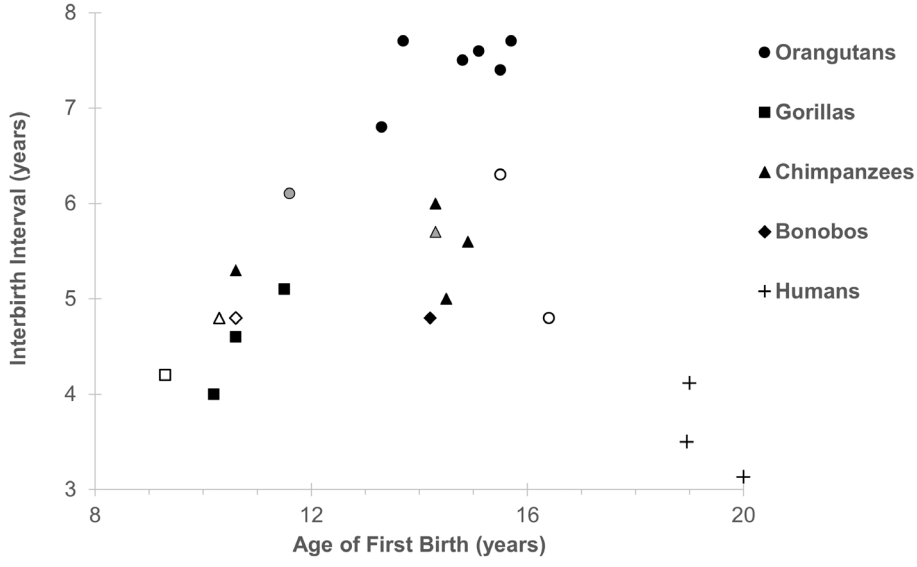

Figure 5.

Fig. 5 Relationship between the length of interbirth intervals and age of first birth in great apes, using data from Table 4. Symbol shapes according to species. Closed symbols depict wild populations, open symbols captive populations, and gray symbols free-ranging provisioned populations. Among wild populations, $r = 0.696$, $N = 14$, $p = 0.006$. Human data (+) shown for comparison.

Fertility data for chimpanzees is the most comprehensive and shows remarkably little interpopulation variation: for seven populations of two subspecies, average intervals between births range from 5 to 6 years. Intrapopulation variation greatly exceeds interpopulation variation (Emery Thompson 2013b), and shows broad overlap with interbirth intervals of human populations (Emery Thompson and Ellison 2017). Birth rates of wild chimpanzees peak between ages 15–25 years, an earlier and less pronounced peak than is exhibited in human forager populations (Emery Thompson, Jones, et al. 2007).

The interval between births consists of a prolonged period of postpartum amenorrhea, a shorter period of cycling to conception, and gestation. Most of the variation in interbirth intervals within and between species occurs during postpartum amenorrhea, which typically comprises 60–80% of the interbirth interval. Thus, interbirth intervals following the deaths of unweaned offspring are substantially shorter than those when the previous offspring survives (Harcourt et al. 1980; Furuichi et al. 1998; Emery Thompson, Jones, et al. 2007; Wich, de Vries, et al. 2009). However, bonobos exhibit unusually early resumption of cycling, followed by an unusually long period of cycling (Kano 1992; Furuichi and Hashimoto 2002). This pattern also has been observed in West African chimpanzees at Taï (Deschner and Boesch 2007). Since data on West African chimpanzees are available only for this one population, it is not yet clear whether this is a subspecies-typical pattern, or if they exhibit an extreme of interpopulation variation across chimpanzees. The functional significance of this difference is yet unclear, but it has been hypothesized that a longer period of cycling may reduce sexual coercion or increase the benefits of promiscuous mating (Wrangham 2002; Deschner and Boesch 2007).

Variation in fertility among chimpanzees has been attributed to temporal and interindividual variation in dietary quality and/or female energy balance (Emery Thompson 2013b), which affects occurrence of sexual cycles (Anderson, Nordheim, and Boesch 2006), ovarian hormone production (Emery Thompson, Wrangham, and Reynolds 2006; Emery Thompson, Kahlenberg, et al. 2007; Emery Thompson and Wrangham 2008; Emery Thompson, Muller, and Wrangham 2014), waiting time to conception (Emery Thompson and Wrangham 2008), and duration of postpartum amenorrhea (Emery Thompson, Muller, and Wrangham 2012). Similar effects are suggested for at least one population of wild orangutans, where ovarian hormones and conceptions peak during peaks in food availability (Knott 1999; Knott 2001; Knott, Emery Thompson, and Wich 2009). The influences of energy availability are reflected in markedly accelerated reproductive rates in captive versus wild great apes (Table 4). Interspecific variation in interbirth intervals is still evident among well-fed captive populations, so reproductive rates are likely to have a strong genetic component. However, these differences may still be long-term adaptations to feeding ecology, promoting more conservative reproductive effort in response to chronic resource instability. Mountain gorillas have the most stable food base and the shortest birth intervals, while orangutans have the most unstable food base and the longest birth intervals. Western gorillas pursue a more chimpanzee-like diet than do mountain gorillas at Karisoke, and they have correspondingly longer birth intervals. Bonobos pursue a more gorilla-like diet than do chimpanzees and have correspondingly shorter birth intervals. Early reports from orangutans proposed the reverse: longer birth intervals in Sumatran versus Bornean orangutans were suggested to result from more productive Sumatran forests, perhaps as a transition to a slower overall life history (Marshall et al. 2009; Wich et al. 2011; Skinner 2014). However, newer comprehensive analyses indicate that the birth intervals of the two species

are statistically indistinguishable (van Noordwijk et al. 2018). Captive orangutans exhibit a small but significant species difference in the opposite direction, with longer birth intervals in captive-born individuals from the Bornean species (Anderson et al. 2008). Neither captive nor wild studies detect significant differences in other demographic variables between Bornean and Sumatran orangutans, supporting a general conclusion that these species have not experienced divergence in life history evolution (Anderson et al. 2008; Knott, Emery Thompson, and Wich 2009; Wich, de Vries, et al. 2009; van Noordwijk et al. 2018).

Comparative reproduction in human populations is often estimated using completed fertility, which is the sum of all age-specific fertility rates. This statistic assumes survival to the end of the reproductive period. By this metric, completed fertility in wild chimpanzees and mountain gorillas would be approximately 8 infants per female (calculated from: Karisoke gorillas, Robbins et al. 2006; multi-site chimpanzee sample, Emery Thompson, Jones, et al. 2007), as high as many high-fertility human populations. This is clearly misleading because most wild great apes do not live through their potential fertility. If mortality is considered, each chimpanzee female born in a multi-site sample (Emery Thompson, Jones, et al. 2007) would be expected to produce 2.53 infants (a net reproductive rate of 1.27 daughters), while the net reproductive rate for the largest chimpanzee sample at Gombe is only 0.745 daughters per female (Bronikowski et al. 2016b). The net reproductive rate of mountain gorilla females at Karisoke is estimated at 1.918 daughters per female (Bronikowski et al. 2016b). Those chimpanzee and gorilla females who live long enough to reproduce at all may be expected to produce approximately 3–5 surviving offspring (Yamagiwa and Kahekwa 2001; Robbins et al. 2007a; Emery Thompson 2013b). Given that a large proportion of females do not live to reproduce at all, most great apes hover dangerously close to the replacement levels of fertility necessary for population viability.

Humans and the other great apes share derived characteristics of the reproductive system that distinguish them from many other primates (Emery Thompson 2013a, 2013b; Emery Thompson and Ellison 2017). These differences include not only the physiological systems governing the reproductive process (e.g., ovulation, pregnancy), but a generally cautious pace of reproduction in which fecundity is highly sensitive to energy availability. The unexpectedly faster rate of human reproduction may not have required major innovations in reproductive physiology but can have instead resulted largely by adaptation to enhanced energy availability for reproductive females, which is in turn a product of cognitive and social adaptations including cooking, tool use and cooperative foraging and hunting, division of labour, and food sharing (Hawkes et al. 1998; Kaplan et al. 2000; Kramer 2005; Wrangham and Carmody 2010; Reiches et al. 2009), as well as metabolic adaptations for higher energy throughput (Pontzer et al. 2016). For example, whereas male foragers increase their workload in order to provision offspring and directly supplement the energy budgets of reproductive women (Marlowe 2001), male great apes provide food for neither. Male chimpanzees have a net negative effect on the energetic status of their female associates (Emery Thompson, Muller, and Wrangham 2014), while association with males raises glucocorticoid levels of female orangutans (Kunz et al. 2020). While male gorillas protect and play with infants (Rosenbaum et al. 2018), and may even adopt orphans, infanticidal behaviour has extreme negative consequences for female reproductive success (Robbins et al. 2007b).

Female Aging and Fertility

The question of whether extended post-reproductive lifespans occur in the great apes is of central importance to evaluating hypotheses for the evolution of this feature in humans. Nevertheless, it continues to be debated whether a significant post-reproductive lifespan is a distinctly human trait (Pavelka and Fedigan 1991; Hawkes 2003; Alberts et al. 2013) or an extension of a trait common in other primates (Cohen 2004; Walker and Herndon 2008). Some of the debate hinges on the choice and definition of terms, such as "menopause", "reproductive cessation", "reproductive senescence", "post-reproductive lifespan", or "post-fertile viability". There is also some fundamental disagreement on the appropriate question. Evolutionary anthropologists are largely concerned with the occurrence and length of the post-reproductive life stage and whether it specifically evolved due to its adaptive benefits. Gerontologists are concerned with mechanistic and evolutionary processes shaping fertility decline, regardless of whether this results in reproductive cessation. Theoretical biologists have focused on whether it is a logical expectation for somatic and reproductive lifespans to be tightly linked, or if post-reproductive lifespans are and should be the norm and subject to allometric scaling (Cohen 2004). To a certain extent, these perspectives can exist simultaneously, provided that the evidence, and their interpretations are accurately understood.

A variety of processes contribute to reductions in fertility with age, but the critical constraint on reproductive lifespan in humans is the exhaustion of the lifetime supply of ovarian follicles, the specific phenomenon that results in menopause. This process is not easily detected, thus various proxies are used, including presence of menstruation (also difficult to detect in wild apes), presence of sexual swellings (only readily observed in *Pan*), and demonstrated fertility. On the one hand, evidence of continued fertility, such as from population-level analysis of age-specific fertility rates, is difficult to misconstrue. However, many analyses, particularly for cross-species comparisons, rely on inference from the interval between last reproduction and death to calculate the proportion of individuals experiencing a post-reproductive lifespan and/or the proportion of the lifespan individuals spend in the post-reproductive stage. These criteria are applied inconsistently and introduce statistical bias (Levitis and Lackey 2011). Moreover, observations of amenorrhea or infertility in a small number of older apes cannot necessarily be attributed to the menopausal process, as little is known about other sources of secondary infertility, such as foetal loss, endometriosis, hormonal imbalances, or infections in the reproductive tract. A second hurdle comes from distinguishing menopause from the natural process of reproductive senescence, which leads to longer intervals between births. For example, in the Kanyawara community of wild chimpanzees, a female estimated to be in her early 60s had experienced over 8 years of amenorrhea following her last infant but began exhibiting sexual swelling cycles in the months before her death (personal observation). The influence of ill health on fertility is also underappreciated. A cross-population study of wild chimpanzees indicated that, even in their 30s, females who were close to death exhibited reduced fertility compared with healthy females (Emery Thompson, Jones, et al. 2007).

Signs of reproductive senescence in gorillas and chimpanzees are clear and consistent with reproductive aging in humans. Cycles gradually increase in length throughout adulthood and become more irregular, ovarian hormone levels decline, and birth rates decrease (Caro et al. 1995; Atsalis et al. 2004; Atsalis and Margulis 2006; Robbins et al. 2006; Emery Thompson, Jones, et al. 2007; Lacreuse et al. 2008). Some captive apes exhibit long acyclic periods at older ages

(Atsalis and Margulis 2006). These data have been interpreted as data for perimenopause and menopause occurring prior to age 40 years in captive apes (Atsalis and Margulis 2006; Videan et al. 2006; Atsalis and Videan 2009). However, other captive facilities have reported continued menstrual cycling in nearly all individuals, suggesting the opposite — menopause is very rare (Lacreuse et al. 2008; Herndon and Lacreuse 2009). In one study of captive chimpanzees, only one of 20 females over the age of 39 years ceased cycling for longer than 12 months: she was 56 years old (Lacreuse et al. 2008).

Direct data on ovarian follicle depletion are available only for captive chimpanzees and one bonobo. In an early study of captive chimpanzees, six deceased females aged 35–48 had histological evidence of continued reproductive ability: primary follicles, recent luteal activity, and/or active endometria (Graham 1979). Two more recently histological analyses used larger samples. Based on different sample sizes and slightly different modelling approaches, one concluded that rates of follicular depletion in humans and chimpanzees were nearly indistinguishable (Jones et al. 2007), while the latter reported a faster rate of decline after age 35 years in humans, albeit with broadly overlapping confidence intervals (Cloutier, Coxworth, and Hawkes 2015). These specific conclusions should be treated with caution not only because the sample of relatively old chimpanzees is sparse but because chimpanzee follicle counts resulted from ovarian cross-sections, while human follicle counts were from whole ovaries. However, two essential points are clear. The ovaries of most (15 of 18) chimpanzee females over age 35 years (up to 49 years) had non-negligible counts of primordial follicles, suggesting continuing fertility to late ages. And, both chimpanzees and humans converge on an approximate age of follicular exhaustion near 50 years.

In humans, menopausal status can be detected by sustained, elevated levels of the gonadotropins LH and FSH and a decreased response to GnRH challenge (Wood 1994). One aged captive bonobo (the only examined, precise age unknown) exhibited menopausal ovaries and had both elevated gonadotropin levels and weak response to GnRH (Gould, Flint, and Graham 1981). The 47- and 48-year old chimpanzees in the same study responded normally to GnRH challenge and exhibited cyclic gonadotropin peaks, though they were less defined than in younger chimpanzees. A larger study of captive chimpanzees found declining LH and FSH levels among older females (Videan et al. 2006), but the significance of this pattern is unclear as it is opposite to that found in aging women. Notably, *in vivo* and post-mortem examinations of captive apes have sometimes shown structural abnormalities of the ovaries and reproductive tract that might interfere with reproductive function of some individuals independently of menopausal processes (Graham 1979; Loskutoff et al. 1991).

Relevant data from the wild are limited to observed births. Among Sumatran orangutans at Suaq Balimbing, interbirth intervals (N = 14) did not increase with age, and the three oldest females had recent births (at ages 38, 38, and 41–50 years: Wich et al. 2004). An analysis of 38 years of demographic data from the mountain gorilla population in the Virungas found that birth rates declined moderately in older age groups, but births to females in their late 30s and early 40s were observed (Robbins et al. 2006). As many as 25% females had terminal birth intervals that exceeded expectation, but the post-reproductive lifespan was trivial, approximately 1–3% of the total lifespan. Birth rates aggregated across six populations of chimpanzees also exhibited a clear pattern of reproductive senescence; interbirth intervals increased with age, but there was evidence for continued reproductive ability in females in their late 40s and even

early 50s (Emery Thompson, Jones, et al. 2007). Birth rates declined to zero at approximately the same age that survivorship reached zero. Taken together, these data suggest that great apes experience a senescent process that can culminate in menopause in some individuals during the 5th or 6th decade of life, but that even in favourable conditions, few individuals live to see this transition. Post-reproductive viability in the great apes would appear to be too rare and too brief to have been a target for selection.

Comparisons across mammals indicates that as maximum lifespan increases, the duration of fertility fails to keep pace, suggesting that the "shelf-life" of oocytes is constrained (Huber and Fieder 2018). In accordance, the great ape data is consistent with the conclusion that the age of last reproduction has been largely conserved during human evolution, such that menopause is primarily the result of extended human lifespan (Hawkes 2003).

The great ape data suggest that the attraction of human men to young women (Buss 1989; Kenrick and Keefe 1992) is a derived feature linked to long-term pair bonding. Whereas men seeking marriage partners may be concerned with the years of fertility remaining, great ape males can be expected to allocate reproductive effort according to a female's likelihood of bearing offspring in the immediate future, as well as her probability of successfully rearing them. In the absence of a long post-reproductive period, aging chimpanzee females not only retain their attractiveness, they actually become more attractive to males, earning more solicitations and inciting more vigorous male competition than younger females (Muller, Emery Thompson, and Wrangham 2006). In most nonhuman primates, young females are less attractive mates (Anderson 1986). Lengthy sub-fecund periods of nulliparous females may deter males from expending mating effort on them. In both chimpanzees and gorillas, females also experience a particularly long interval between first and second births (Jones et al. 2010; Robbins et al. 2006), perhaps because early reproductive effort competes with the last years their own growth. Additionally, older mothers are expected to produce higher quality offspring due to prior maternal experience, increased access to resources, and/or stronger social ties. Like many other primates, first-born mountain gorilla infants have significantly higher mortality than later-born infants (Robbins et al. 2006). This is not true of chimpanzees, despite the low rank of primiparous mothers, perhaps because primiparous mothers are more solicitous of their infants (Stanton et al. 2014). Because birth rates decline at later ages, chimpanzees born to older mothers receive longer periods of investment and experience less sibling competition, positively impacting their growth (Emery Thompson et al. 2016). On the other hand, without a prolonged post-reproductive period, older ape mothers pose a higher risk of orphaning dependent infants.

Male Reproduction

In the wild, chimpanzee males are capable of siring offspring by the age of 9 years (Langergraber et al. 2012). According to genetic paternity analyses, males achieve a peak in paternity between ages 16 and 25 years (Newton-Fisher 2004; Boesch et al. 2006; Wroblewski et al. 2009; Muller et al. 2020) and appear to be capable of successful reproduction into at least their mid-forties or early fifties, though the ages of some of the oldest fathers may be overestimated. As rank influences paternity in all populations studied, the age distribution of paternities is influenced in part by the ages that males can achieve high rank. However, males who were unusually young or old when siring their offspring did not do so because they were high-ranking, but in

spite of being low-ranking (Wroblewski et al. 2009). Individual males are known to have sired up to 7 offspring, and some have sired offspring over periods of at least 16 years (Wroblewski et al. 2009). In addition to dominance rank, male reproductive success can be influenced by the formation of coalitions with other males (Gilby et al. 2013) and the use of coercive aggression against females (Feldblum et al. 2014).

Mountain gorilla males are known to have sired offspring between the ages of approximately 9 and 25 years, with a mean paternal age of 19 years at Karisoke (Bradley et al. 2005; Nsubuga et al. 2008). With the assumption that they sired all offspring born during their tenures, dominant silverbacks (N=22) sired an average of 7.2 (range 0–27) offspring that survived to weaning (Robbins et al. 2014). This is a slight overestimate, as dominant silverbacks actually sire only 71–85% of those offspring born in multimale groups (Bradley et al. 2005; Nsubuga et al. 2008). Due to higher infant mortality, shorter tenure lengths, and smaller harems, silverback harem leaders in the western gorilla population at Kahuzi-Biega produce only approximately 2 infants that survive to weaning (Breuer et al. 2010). The fertility rate of an average male should be lower if not all males are able to become dominant. Male body size and competitive strength can impact reproductive success by increasing harem size and tenure length (Caillaud et al. 2008; Breuer et al. 2010). In multimale groups, mountain gorilla males are reported to improve their paternity success by behaving affiliatively with infants, even those they have not sired (Rosenbaum et al. 2018).

The occurrence of male bimaturism, and the dispersal of males over long distances, hinders estimates of fertility parameters for wild male orangutans. Males succeed at siring offspring when both flanged and unflanged (Utami et al. 2002; Goossens et al. 2006). One successful male is known to have sired at least 3 offspring over the course of 19 years (Utami et al. 2002).

Data on the ages of sires are not available for bonobos. A recent study indicates high reproductive skew compared with chimpanzees, with one bonobo male siring at least 9 offspring over a 12-year period (Surbeck et al. 2017). In addition to dominance rank, the presence of a male's mother improves reproductive success (Surbeck et al. 2019). Mothers provide agonistic support to males and facilitate their proximity to fecundable females (Surbeck, Mundry, and Hohmann 2011).

The most obvious feature that distinguishes human male fertility from that of great apes is the extent to which men are constrained by marriage in most societies. Depending on marital practices within a population, men may experience delays to the age of first reproduction, economic or cultural constraints on polygyny, and fertility rates that are interdependent with their wives'. Additionally, the ways in which males compete differs between the great apes, who rely largely on aggressive competition, and humans for whom economic resources and social prestige may be better predictors of reproductive success (Gurven, Kaplan, and Gutierrez 2006; von Rueden, Gurven, and Kaplan 2010). As the latter peak relatively later in life than physical prowess, human males can be predicted to experience later ages of paternity than their great ape counterparts (Muller et al. 2020). Nevertheless, recent evidence suggests that male chimpanzees in some communities can leverage coalitionary support to maintain high rank long past their physical prime (Watts 2018), potentially extending reproductive tenures.

Conclusions

Demographic data from the great apes provide an essential comparison for understanding the nature of human life history evolution. Historically, these comparisons have relied on small numbers of individuals in captivity or rough estimates from the wild. As this review reveals, there are still many gaps where we lack comprehensive, ecologically-valid data on great ape life history parameters. This is not for lack of effort. Great ape researchers face enormous hurdles, some owing to the elusive habits of the species themselves, and some to chronic problems of civil unrest, epidemic disease, and other conservation threats in the areas they inhabit. Yet, these problems place a premium on high-quality demographic data that can be used to evaluate the impacts of human disturbance on long-term population sustainability.

Mountain gorilla research at Karisoke and chimpanzee research at Gombe, Kibale, and other younger field sites have generated high-quality datasets on fertility and mortality and will yield more detailed and precise data as their study durations approach and surpass the lifespans of the animals they study. An exciting new wave of research has produced increasingly detailed analysis of behavioural development in infants and juveniles. New and newly-reopened field sites are beginning to build datasets for poorly-studied species, such as western lowland gorillas and bonobos. Researchers are now in the process of using modern techniques of parallel-laser photogrammetry, genetic analysis, endocrinology, immunology, and stable isotope analysis to generate detailed data on body growth, infant nutrition, health, and population structure. The next ten to twenty years will be a critical period of coalescence for great ape life history studies.

By combining what we do know about great apes from the wild and captivity, some broad conclusions are possible. In general, orangutans exhibit characteristics of a conspicuously slower life history than the other great apes, characterized by a later age of sexual maturity, later ages of weaning, longer interbirth intervals, and higher survivorship. Mountain gorillas, at least in one of the two populations, exhibit the inverse suite of characteristics indicative of a faster life history schedule. The other species fall in the middle, with bonobos and western gorillas exhibiting reproductive characteristics at the faster end of the range of variation exhibited in chimpanzees. Considerable intraspecific variation is evident but poorly estimated as some species are well-studied in only one locality.

The pace of reproductive events is notable because it does not indicate a strong correspondence to body mass within this clade. Classic models of life history predict, with the support of ample comparative evidence, that larger body mass will correspond with a slow life history because it takes longer to grow larger, and body mass confers a survival advantage (Harvey and Clutton-Brock 1985; Read and Harvey 1989; Charnov 1991). Gorillas would then be predicted to be the slowest, and chimpanzees and bonobos the fastest. Furthermore, body growth studies suggest that among the great apes, larger body mass (except for the males of highly dimorphic species) is not associated with a longer growth period. We suggest that while large body size is essential to understanding the evolution of slow life histories in the hominids as a clade, variation within this group of closely-related species may stem from more specific adaptations to feeding ecology. Importantly, we may expect selection on body mass and selection on life history strategies to move along different axes in response to different features of the environment. Generally, growing too large is disadvantageous for apes due to the costs of travel, particularly when patchily-distributed fruit resources require ranging over large areas (Isbell et al. 1999; Key and Ross 1999). Indeed, flanged male orangutans appear to suffer

high costs of travel, and modify their behaviour (and perhaps their developmental strategies) accordingly (Mitani 1989; Utami Atmoko and van Hooff 2004; Knott and Emery Thompson 2013). However, large body mass in gorillas confers a nutritional advantage for processing large amounts of herbaceous material (Gaulin 1979; Watts 1996; Müller et al. 2013). These resources pose low ecological risk to mothers and infants, as they are widespread and continuously available (Watts 1998). Orangutans, by contrast, experience the highest ecological risk, posed by dramatic and unpredictable fluctuations in fruit availability (Knott 1998a; Harrison, Morrogh-Bernard, and Chivers 2010; Vogel et al. 2012). Such conditions certainly constrain fertility in the short term, but may have additionally selected for a highly risk-adverse reproductive schedule with extended infant dependence on maternal nutritional support (van Noordwijk and van Schaik 2005; Knott, Emery Thompson, and Wich 2009; Smith et al. 2017). Thus, when we consider humans alongside the apes, we have no reason to invoke strong allometric constraints and can expect that the dramatic differences in resource quality and stability effected by technological and social adaptations (Hawkes et al. 1998; Kaplan et al. 2000; Carmody and Wrangham 2009) will have been critical to shaping our life histories (Emery Thompson and Ellison 2017). However, the reduction of ecological constraints has been replaced by strong internal constraints of a greatly enlarged brain (Aiello and Wheeler 1995; Kuzawa et al. 2014).

Over and above the progress on generating demographic statistics on great apes, researchers are gaining important insights into the dynamics of life history variation. This includes close study of the role of energy availability in interindividual and interpopulation variation in fertility, and perhaps mortality. Additionally, rapid progress has been made in understanding the dynamics of infant nutritional development and birth spacing. These types of examinations have revealed important differences in life history processes between hominids and other primates that have the potential to transform our approach to human life history evolution (Knott 2001; Emery Thompson 2013b; van Noordwijk, Kuzawa, and van Schaik 2013; van Noordwijk et al. 2013; Emery Thompson and Ellison 2017). With this in mind, there is a critical need for detailed data on the natural causes of mortality in all of the great apes to better understand the factors that constrain lifespan. Finally, as datasets improve, an essential future goal will be to model the influence of variance in particular life history parameters on reproductive success. Together, these approaches may allow us to assess the vulnerability of different life history features to selection during the evolution of the human species.

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Tables 1–4 (Comparative mortality and survival data for wild great apes, Comparative growth data for great apes, Weaning and nutritional development in wild great apes, and Reproductive parameters of wild and captive great apes) are available online at https://doi.org/10.11647/OBP.0251#resources

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20. Did Grandmothers Enhance Reproductive Success in Historic Populations?: Testing Evolutionary Theories on Historical Demographic Data in Scandinavia and North America

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Human reproductive success requires both producing children and making investments in the development of offspring. To a large extent these investments are made by the parents of the child, but researchers are now looking beyond the nuclear family to understand how extended kin, notably grandmothers, enhance reproductive success by making transfers to progeny of different kinds. The extent to which kin influence fertility and mortality outcomes may vary across different socio-economic and geographic contexts; as a result, an international comparative framework is used here to sharpen our understanding of the role of kin in reproduction. This chapter assesses the role of grandmothers in fertility outcomes in a comparative historical demographic study based on data from Scandinavia and North America. The individual-level data used are all longitudinal and multigenerational, allowing us to address the impact of maternal and paternal grandmothers on the fertility of their daughters and daughters-in-law. Attending to heterogeneous effects across space and time as well as within-family differences via the use of fixed effects models, we discover broader associations of the paternal grandmother with higher fertility across the four regions. We also find a general fertility advantage associated with the post-reproductive availability or recent death of the maternal grandmother in the four populations. Important variations across regions nevertheless exist in terms of the strength of the association and the importance of the grandmother's proximity. Our interpretation is that grandmothers were generally associated with high-fertility outcomes, but that the mechanism for this association was co-determined by family configurations, resource allocation and the advent of fertility control.

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Introduction

In the evolutionary theory of aging, menopause and especially the extended period of post-reproductive lifespan enjoyed by humans has long been considered a puzzle (e.g. Rogers 1993; Peccei 2001; Ladhenperä et al. 2004), and much research effort has been devoted to explaining this phenomenon both theoretically and empirically (Williams 1957; Lee 2003). While not unique in experiencing menopause (e.g. Paul 2005), human females are alone in having relatively low post-reproductive mortality (e.g. Hill & Hurtado 1991; Voland et al. 2005; Hawkes et al. 1998, 2000; Kaplan et al. 2000). Menopause may be seen as an adaptation which increases fitness: aging mothers with declining age-related fecundity or whose reproduction concluded in midlife could instead assist their own daughters in their reproduction (Williams 1957). While researchers concur that the long post-reproductive life span among humans is not just an artifact of the aging process, but a result of evolutionary processes, there is an extensive literature questioning some of the theoretical mechanisms of why and when menopause developed. (Hawkes et al. 1998; Hill and Hurtado 1991; Peccei 2001, 2005; Rogers 1993). Reproductive success is a key to evolution, and it is determined by both the number of offspring (fertility) and the survival of offspring and their parents (mortality). In this way human reproduction requires both producing children and making investments in the development of offspring, activities which take place over a considerable amount of time. To a large extent these investments are made by the mother of the child, but it is also widely recognized that other individuals make investments in children either directly through caregiving, or indirectly by helping their mothers. Lee (2003) highlights the role of such transfers in explaining variations in human aging. While fathers also play an important role in child development (Mace & Sear 2005), researchers have looked beyond the nuclear family to understand how extended kin affect reproductive success of mothers by making transfers of different kinds. The benevolent role of maternal grandmothers has been central to this argument (Voland et al. 2005; Hawkes et al. 1998). By helping their daughters in different ways, women in post-reproductive ages could increase the number and quality of their grandchildren, thereby promoting fitness.

The extent to which kin influence fertility and mortality outcomes may vary by socio-economic and geographic contexts; as a result, an international comparative framework is needed to sharpen our understanding of the role of kin in reproduction. The aim of this chapter is to assess the role of grandmothers for fertility among their daughters in a comparative historical perspective. We use historical demographic data from Scandinavia and North America to assess the role of kin in fertility before the fertility transition. The individual-level data used are all longitudinal and multigenerational, allowing us to address the association between the presence and proximity of grandmothers and the fertility of their daughters or daughters-in-law. We compare the impact of paternal and maternal grandmothers, as well as analyze heterogeneous effects across space and time. To distinguish women belonging to different generations, we identify grandmothers as the generation F0, mothers (our subjects) as the generation F1 and children (those who are born to our subjects) as generation F2.

Background

We draw upon two analytic frameworks featured in evolutionary anthropological studies: the helpful grandmother hypothesis (e.g. Hawkes et al. 1998, 2000; Hawkes & Coxworth 2013;

Voland et al. 2005), which describes the positive influence of post-reproductive women (F0) on grandchildren's survival, and the concept co-operative breeders, a "relatively unusual childrearing system in which mothers receive help from many other individuals in raising offspring…". (Beise 2004; Mace & Sear 2005; Sear & Coall 2011; Sear 2015; Hawkes & Smith 2009). Both analytic frameworks emphasize the important role played by kin in fertility, survival and longevity. These hypotheses have been applied in historical demographic studies of several different contexts. For example, in a study of a parish in Northwest Germany, Voland and Beise (2002) found maternal grandmothers to reduce infant mortality substantially, while they found opposite effects of paternal grandmothers (Voland and Beise 2002; see also Beise 2004). They interpret this negative impact of paternal grandmothers as a result of conflict between the maternal grandmother and the paternal grandmother. Fertility-enhancing effects of grandmothers have been reported in pioneer-era Utah (Hawkes and Smith 2009), yet studies which have addressed the inter-generational transmission of fertility have obtained divergent results (Brunet & Vézina 2015; Kolk 2013), as have studies of the transmission of longevity from parents to children (Houde et al. 2008; Brunet and Vézina 2015; van den Berg et. al 2017). In historic Quebec, Desjardins et al. (1991) report a "very weak and not significant" intergenerational transmission of fertility; an additional 2001 article by Gagnon and Heyer found the association of mother's and daughter's fertility to be "almost null" (Gagnon & Heyer 2001). The latter study, however, was more concerned with the consequences of the intergenerational transmission of fertility for the evolution of a population's gene pool and did not adjust for potential confounders. A new study directly addressing the grandmother effect in historic Quebec concluded that living maternal grandmothers enabled daughters to increase the number of children born by 2.1, and that this effect was stronger among maternal grandmothers living in proximity (Engelhardt et. al. 2019). A recent study of late-nineteenth-century Utah distinguished women whose mothers manifested high or low fertility relative to the fertility of the mother's own cohort; women whose mother had relatively high fertility demonstrated, in turn, higher parity progression ratios (Jennings, Sullivan & Hacker 2012; Anderton et al. 1987). A broader study of recent net marital fertility (children < 5) in 1880 United States found a modest positive effect of paternal grandmothers: women with potential mothers-in-law living in adjacent households had about 2% more children than women with no potential mothers-in-law living nearby (Hacker and Roberts, 2017).

Sear and Coall (2011) review the literature on the impact of grandmothers on fertility in different contexts, before and after the fertility transition. Most studies seem to identify some kind of grandparent influence on fertility, but both the direction of the influence and which grandparent is most important differs widely across studies. Nonetheless, a conclusion from the review of pre-transitional societies is that paternal grandmothers seem to promote fertility, while maternal grandmothers if anything seem to reduce fertility. However, as these reported patterns are inconsistent it is difficult to reach a consensus about the role of grandmothers on offspring fertility. Indeed, Sear and Coall call for more data to shed more light on this issue. We suggest that some of the variability in the literature is related to different family systems in different contexts, which have implications for co-residence and proximity of grandmothers.

There are a number of potential proximate explanations responsible for a fertility-enhancing grandmother effect. First, while the presence of a grandmother may have helped new mothers initiate breastfeeding, it also is possible that the presence of a grandmother stimulates earlier

weaning, allowing the mother to engage in other productive activities and leaving more child care to the grandmother. At the same time the shorter period of lactation would enhance subsequent fertility, and hence shorten birth intervals for mothers with a grandmother present (Gauthier 1991; Hawkes et al. 2000). Secondly, the presence of a grandmother might promote mothers' access to resources in the form of better nutrition, and possibly a lower workload, two conditions which would promote higher fertility in pre-transitional contexts (Sear and Coall 2011). Thirdly, grandmothers could exercise social pressure on their daughters to reproduce in order to maximize the number of grandchildren, although such an effect is likely to vary by context. Some have argued that the social pressure to bear more children would be greatest from paternal grandmothers who prioritize grandchildren as successors or as workers for the family patrimony and who may be less concerned about the physical costs of childbearing for the mother (Mace and Sear 2005; Sear and Coall 2011). Maternal grandmothers, on the other hand, may have encouraged their own daughters to limit or at least space births out of a concern for the hazards of repeated childbearing (Sear and Coall 2011).

We address our research questions with four robust datasets drawn from one Scandinavian and three North American populations, applying a common set of analyses of birth intervals. Our internationally comparative approach resembles that used in prior research on fertility and longevity involving these data (Smith, Gagnon et al. 2009; Gagnon et al. 2009; Dribe et al. 2017). While previous analyses of demographic behavior during this period have explored genetic predispositions, early-life conditions and socio-economic status, the mediating influence of kin on these processes remains largely underexplored. In particular, we highlight the role of proximity and availability (via vital status) of grandmothers, exploring whether the hypothesized positive influence of grandmothers on fertility varied by their residential proximity to their children and grandchildren. Finally, we consider differences between maternal and paternal grandmothers, controlling for proximity to the daughters/daughters-in-law.

Context

The Scandinavian population included in this trans-Atlantic comparative study are persons residing in five rural parishes (Halmstad, Hög, Kågeröd, Kävlinge, and Sireköpinge) in Scania, located in southern Sweden.[1] The period is defined as births occurring from 1766 to 1899 (Table 1). These parishes had a total of 3,900 inhabitants in 1830. By the end of 1900, this figure had increased to 5,500, suggesting approximately the same growth rate as Sweden as a whole. The selected parishes are close in geographical location, showing the variations that could occur in a community regarding size, topography, and socioeconomic conditions. Both life expectancy at birth and fertility was somewhat higher than for Sweden as a whole in the nineteenth century, but closely followed the same development over time (Quaranta 2013: 53; Bengtsson and Dribe 2010). Mortality started to decline in the late eighteenth century, when infant mortality began to fall, closely followed by child mortality. Infant mortality in the area fell from around 250 per thousand in the 1760s to around 100 per thousand in 1900 (Johansson 2004), which is similar to the development for Sweden as a whole (Hofsten and Lundström 1976, Table 46). From about the mid-nineteenth century, adult mortality started to decline as well. Life expectancy at birth increased from about 40 years in the beginning of the nineteenth

1 http://www.ed.lu.se/databases/sedd

century to around 50 years in 1900. Over the same period life expectancy at age 20 increased from 37 years to 46 years (Statistics Sweden 1999: Table 5.4). Fertility in Sweden, as well as in the region we are looking at, started to decline around 1880 and followed a quite typical pattern where industrialization, urbanization and previous mortality decline all contributed to the decline; total fertility in the country declined from 4.2 in 1880 to 1.8 in 1930 (Dribe 2009; Bengtsson and Dribe 2014). Mean age at first marriage was 25.4 for women and 27.9 for men in the five parishes in 1815–1894. About 12 percent of men and 20 percent of women in the same period were never married at age 45, a proportion which increased substantially over the nineteenth century (Dribe and Lundh 2014: 222–23).

Table 1: Summary of periods, demographic indicators and case counts Scania (Sweden), St. Lawrence Valley (Quebec), Saguenay (Quebec), and Utah

	Scania	Q-SL	Q-Sag	Utah
Period under study				
Births of children (F2)	1758–1883*	1666–1791	1843–1963	
Births of mothers (F1)	1784–1899	1650–1750*	1807–1914**	1847–1919*
Demography				
Infant Mortality Rate	100/1000 in 1900	240.9/1000	1861: 144/1000	72–87/1000
Life Expectancy at birth	1800: 40; 1900: 50	35.5	1861–1931: 48–54	
Life Expectancy at age 20	1800: 37; 1900: 46	53.9		
Total Fertility Rate	4.2 in 1880; 1.8 in 1930	11	up to the 1930s: 10–11	8–11
Timing of fertility decline	1880	20th century	1930–1960	1880
Mean age at marriage - men	28	26	1850–1890: 25	25
Mean age at marriage - women	25	22	1850–1890: 22	21
% never married at age 45/50	men: 12%; women: 20%	6.5–10%	1850–1890: 3–5%	
Population in analysis				
Population	3,900 in 1830; 6,300 in 1939	70,000 in 1760	5,241 in 1851; 190,142 in 1951	200,000 in 1890
Number of F1 mothers	927	9,921	18,547	182,069
Number of F2 children	2,865	71,166	143,365	
Number of parishes/counties	5	135	122	

Q-SL = Quebec - St. Lawrence Valley
Q-Sag = Quebec Saguenay Lac St-Jean region
*Criteria used for data selection
** Selection was based on marriage: women who married in the region from the beginning of settlement (first marriage recorded in 1842) to 1929 were selected (first marriages only).
Sources:
Scania: Quaranta 2013; Bengtsson and Dribe 2010; Johansson 2004; Hofsten and Lundström 1976; Dribe 2009; Dribe and Lundh 2014
Q-SL: Charbonneau et. al. 2000; Amorevieta-Gentil 2010; Ouellette et al. 2012; Dillon 2010;
Q-Sag: Bouchard 1996; Pouyez et Lavoie 1983
Utah: Bean et al. 1990

Crossing the Atlantic Ocean, our earliest North American population studied is the colonial population of the St. Lawrence Valley in Quebec, encompassing reproducing women born from 1650 to 1750 and their births occurring from 1666 to 1791 (Table 1). From an initial group of 6,500 founders, the colony grew via natural reproduction to over 70,000 by 1760 and 180,000 by the end of the eighteenth century (Desjardins 2008: 78; Charbonneau et. al. 2000: 104,106). This population was marked by high marriage intensity, an early age at marriage, high fertility and high infant mortality. In clonial Quebec, infant mortality was higher than that observed in Scania; about 241 per 1,000 from 1640–1779 overall but rising from 171 per 1,000 for children born before 1680 to 225–350 per 1,000 during and after the British conquest (1750 to 1779) (Charbonneau et. al. 2000: 124; Amorevieta-Gentil 2010: 131). Life expectancy at birth for the whole population born from 1608 to 1760 was 35.5 years, while those who lived to at least age 20 could expect to live to 54 years; adult longevity increased by 2 to 3 years during the latter part of the eighteenth century (Charbonneau et. al. 2000: 126; Ouellette et. al. 2012: 588–89). While the late-nineteenth-century Scanian population studied showed signs of fertility control, the Catholic population of Quebec practiced natural fertility and bore large families: the total fertility rate of the Quebec population born 1680 to 1760 was 11, while families had on average 7.3 children (Gagnon et. al. 2009; Dribe et. al. 2017; Charbonneau et. al. 2000: 123). The mean age at marriage for Quebec women and men was 2–3 years younger than observed in Scania, 22 for women and 26 for men. A larger proportion of colonial Quebec women were ever-married compared to their counterparts in Scania: just 6–10% of Quebec persons aged 50+ had never married (Dillon 2010: 153; Charbonneau et. al. 2000: 113).

Many demographic and economic patterns evident in the seventeenth- and eighteenth-century Quebec population of the St. Lawrence valley can also be observed within the nineteenth- and twentieth-century Saguenay region, the second Quebec population studied. Our analysis of the Saguenay population includes reproducing women who married in the region between 1842 and 1929. These women were born between 1807 and 1914, and their children between 1843 and 1963 (Table 1). The Saguenay region, located approximately 200 kilometers north of Quebec City, was characterized by its relative geographical isolation and cultural uniformity. The colonization of the region by French Canadians began in the 1830s and the population grew rapidly from 5,241 in 1851 to 190,142 in 1951 (Pouyez et al. 1983). Nuptiality and fertility characteristics until the first decades of the twentieth century were similar to colonial Quebec. Age at marriage was low — 22 on average for women and 25 for men — and the proportion ever married was high with only 3 to 5% of the population aged 50+ never married. Fertility was also high with an average of about 10 to 11 children in complete families up until the 1930s (Gagon et. al. 2009; Dribe et al. 2017; Bouchard 1996: 179) as the fertility transition occurred later in the Saguenay region than in the rest of Quebec and other parts of Canada (Gauvreau et al. 2007). However, life expectancy at birth was higher than in colonial Quebec at 48 years in 1861 and reaching 53.5 in 1931 while the infant mortality rate was much lower at 144 per 1,000 in 1861 (Pouyez et al. 1983).

Our fourth historic population is located in Utah, in the western United States, and examines women born from 1850 to 1919 (Table 1). Utah was characterized by rapid settlement which began in 1847 primarily by members of the Church of Jesus Christ of Latter-day Saints (LDS). LDS immigration into Utah was also accompanied by non-LDS immigration, though initially these non-LDS migrants represented a smaller proportion of this migration stream. Individuals

and families who joined the LDS church and who immigrated were generally from the eastern seaboard United States and from Northern and Western Europe. According to data from the U.S. Census Bureau, the resident population of the state grew from just over 11,000 in 1850 to over 200,000 by 1890 and then to over 500,000 by 1930. This rapid rate of growth reflected both high natural increase and substantial immigration. Fertility rates in Utah during this period were the highest in the United States, certainly owing in part to the pro-natalist doctrine of the LDS faith, as well as economic forces promoting increased fertility in the rural and agriculturally dominated West relative to other parts of the United States. The total fertility rate of married women born 1860 to 1864 was 10.6, nearly as high as that observed in colonial Quebec and in the Saguenay region (Bean et al. 1990: 130). However, as seen in the Scanian population, substantial fertility decline was evident by the 1880s (Bean et al. 1990:135–6). At 72–87 per 1,000 children, Utah's infant mortality rates during this time were lower than those observed in the earlier colonial Quebec population and in the Saguenay region for the same period but not as low as those observed in Scania. Women's mean age at marriage in Utah was 21 while that for men was 25, averages which are younger than those observed in Scania and similar to those seen in colonial Quebec and in Saguenay.

The four populations studied vary in terms of socio-economic setting. The Scanian population studied is almost entirely rural. One of the parishes developed into a small town by the end of the nineteenth century following the construction of the main railroad on the west coast. Even though Sweden allowed partible inheritance, and from 1845 onwards equal inheritance for sons and daughters, farms were normally transferred to one of the children while the others were compensated in different ways. It was more common to transfer to a son than to a daughter (or son-in-law), but the latter happened frequently as well (Dribe & Lundh 2005a). As a result, women more often moved to their husbands' place of origin, but it was not uncommon for the husbands to move to the wives' place of origin. Among nineteenth-century farmers, freeholders as well as tenants, grandparents usually co-resided with the son or daughter who took over the farm, while intergenerational co-residence seems to have been much less common among the non-landed groups (Dribe & Lundh 2005b; Lundh & Olsson 2002). Hence, rather than making assumptions based on normative practices, it is vital to control for proximity when analyzing the impact of grandparents.

The Quebec St. Lawrence valley population was also largely agricultural, with most families residing on small farms. The colonial Quebec population included only three urban areas with a mix of trades, artisans, merchants, military and small number of social elites such as government officials. The nineteenth- and early twentieth-century Saguenay population also consisted mainly of farmers, with many who combined farm work with logging the forests during the winter. In contrast to the colonial Quebec population, however, industrialization had begun with the implantation of pulp industries at the turn of the twentieth century and progressed much faster with the arrival and expansion of important aluminum and hydro-electric plants before World War II (Igartua and de Fréminville, 1983; Bouchard, 1996). Land transmission both in colonial Quebec and in Saguenay was gendered, as it usually passed to one or more sons. The transmission practices were generally "pluri-établissement" rather than strict primogeniture, with efforts made to settle all surviving sons, ideally on land in proximity to the family patrimony, though migration could be used, either by the whole family or by brothers as a strategy to perpetuate the agricultural mode of life (Dechêne 1974: 244, 248; Greer 1985:

74; Dépatie 1990: 177, 189; Lavallée 1992: 212; Bouchard 1996: 212, 333; Dillon 2010: 144–45; Beauregard et al. 1986: 399). Daughters often moved to the parish of residence of the husband. In the St. Lawrence valley, up to two-thirds of women moved between their own parish of baptism and the parish of marriage/baptism of the first child (Dillon 2016: Table 3; Beauregard et al. 1986: 402), while in the Saguenay region, about one-third of marriages involved spouses from different parishes (Bouchard 1996: 266). As a result, a gendered intergenerational transmission of migration propensity may be observed (Gagnon et al. 2006), as men settled around their male kin. As a result, women may have been more likely to reside in proximity to the paternal grandmothers than the maternal grandmothers.

Like the Saguenay population, the Utah population featured an increasingly mixed economy, with settlers chiefly engaged in farming. The non-LDS populations, on the other hand, were disproportionately engaged in mining and the railroads during the development of the American West. This development was made manifest by the connection of two railroad systems linking the eastern and western portions of the United States and culminating in northern Utah in 1869, a transition symbolized by the driving of the Golden Spike in Promontory Utah.

Drawing upon the substantial information regarding these four populations, we offer five fundamental hypotheses. First, we hypothesize that living paternal and maternal grandmothers will be positively associated with shorter waiting times to next birth of their reproductive-age daughters or daughters-in-law. The presence of grandmothers may have increased fertility via several mechanisms: grandmothers may have stimulated earlier weaning by liberating the mother for other activities; they may have promoted mothers' health via improved nutrition and a lowered physical workload; and they may have exerted a social pressure on mothers to bear more children. Second, we hypothesize a special role played by grandmothers living close by, whose proximity may have entailed nutritional and/or labour-saving benefits. A grandmother's physical presence could also signal access to a broader circle of extended kin and their physical resources. Owing to those benefits, we hypothesize that fertility-enhancing effects will be stronger among grandmothers living in proximity, and that some of the effects may persist among adult daughters whose grandmothers had died recently. Third, we propose that the availability of a grandmother at higher birth orders may wield a stronger relative effect, as mothers at this point in their life course may have been more busy and in greater need of help. While women at this stage of the life course may have benefitted from the presence of older daughters who provided child care for younger siblings, access to their mother or mother-in-law may have also posed a clear advantage in women's ability to continue bearing children. Fourth, since paternal grandmothers may have been concerned with the need to produce young workers for the family patrimony while maternal grandmothers may have been more concerned with their daughter's health, we hypothesize stronger fertility-enhancing effects with respect to paternal grandmothers. Furthermore, since across these contexts many women moved to their husband's place of residence, possibly even their husband's family patrimony, paternal grandmothers may have been more often physically present and able to contribute to their daughter-in-law's fertility outcomes. Fifth and finally, we expect to see stronger size effects in Utah, a population with high fertility yet with more population heterogeneity (LDS versus non-LDS) and which was beginning to undergo the fertility transition, potentially leading to greater differentiation of fertility outcomes.

Microdata Sources

We draw upon four data sets encompassing three regions: the Scanian Economic-Demographic Database (SEDD), representing southern Sweden, the Registre de la population du Québec ancien (RPQA), representing the seventeenth- and eighteenth-century Quebec colony of the St. Lawrence Valley, the BALSAC database, representing the nineteenth- and twentieth-century Saguenay Lac St.-Jean region of Quebec, and the Utah Population Database (UPDB), representing the settler population of the state of Utah during its frontier era and its subsequent development into the early portion of the twentieth century. What is central to the analysis are the three generations that are fundamental to test our hypotheses. As stated in our introduction, we adopt the following notation to describe the three generations:

F0: Grandmothers

F1: Mothers (the subject in fertility analyses)

F2: Children (the births of children represent the fertility outcomes)

The Scanian Economic Demographic Database (SEDD) is based on family reconstitutions and local population registers, which include information on demographic events and migration for all household members and families in households (Bengtsson et al. 2017). Vital events were checked against birth and death registers to adjust for possible under-recording of events in the population registers. In this study, we use data from 1766 to 1899. Between 1766 and 1814 the data are based on family reconstitutions and linked annual information at the family level on place of residence, land holdings and occupation. From about 1815 onwards data are based on population registers with individual information on migration to and from households, vital events, etc. The resulting database contains all individuals (men and women) born in the different parishes or migrating to them. Instead of sampling particular cohorts, every individual is followed from birth or time of arrival in the parishes to death or migration out of the parishes. The dataset for analysis was constructed using the programs developed in Quaranta (2015).

Data on the population of the St. Lawrence Valley, Québec, a population spanning both the French and English regimes, are drawn from the *Registre de la population du Québec ancien* (RPQA), a parish register-based family reconstitution of the Québec Catholic population from 1621 to 1799 (Dillon et al. 2017). The RPQA data are mainly based on linked baptismal, marriage and burial acts, with some supplementary information deriving from complementary sources such as marriage contracts. The database includes all identified Catholic individuals who were born, married or died in the parishes of the St. Lawrence Valley. These data feature complete information on dense kin networks: in the context of Québec's natural fertility regime, individuals could have as many as 9 siblings, the age spread of siblings could be 20 years, and younger siblings could be the same age as the children born to their eldest siblings (Dillon et al. 2017: 7).

The Saguenay data are drawn from the BALSAC population database which includes church and civil records for an almost exclusively Catholic population. All births, marriages, and deaths that occurred in Saguenay from the onset of colonization to 1971 have been transcribed and linked using family reconstitution methods to form the BALSAC database (BALSAC 2019). Individuals in this database are followed until they die or migrated out of the Saguenay region.

The Utah microdata come from the Utah Population Database (UPDB) (Pedigree and Population Resource 20122). The core of the historic portion of the settler population and their

descendants within the UPDB are based upon information from over 185,000 three-generation family documents provided by the Genealogical Society of Utah. These genealogical records provide data on migrants to Utah and their descendants born from the early 1800s to the mid-1970s (Smith, Mineau et al 2009). These data have been supplemented with vital records that further describe the numbers, dates, and locations of births and deaths for individuals and their family members represented in the UPDB.

Selection, Methods and Operationalization of Variables

Selection

In terms of time period, our data are selected from the earliest year feasible for each data set. The Scanian analysis selected women under observation from January 1, 1766, to December 31, 1899, while the three North American studies selected reproducing women born over specified seventeenth to early twentieth-century periods (see Table 1). Our general aim is to study pre-transition populations, and as such the mothers (hereafter called F1) are selected for a pre-transition period in each of the four datasets. However, in the case of Scania and Utah, some of our intervals move into the period when changes associated with the fertility transition are beginning to happen. The Scanian data cover the period 1766–1900, and include 927 mothers (F1) and 2,865 children (F2). The Quebec RPQA database provides data from the earliest period, analyzing 9,921 mothers (F1) born between 1650 and 1750; the births of their 71,166 children (hereafter called F2) extend from 1666 to 1791. The Saguenay data encompass 18,547 mothers who married in the region between the beginning of settlement (first marriage recorded in 1842) up to 1929. These women were born from 1807 up to 1914, and the births of their 143,365 children cover the years 1843 to 1963. The UPDB sample used in the analysis reported in this paper include 182,069 women born in Utah between 1850 and 1919 who are observed living in Utah after age 15.

In general, mothers with known dates and places of birth and death or outmigration, who married at least once and have had at least one child are selected. These selections were made in order to study a population of women (and their husbands) who are not sterile, and to ensure data quality and completeness. We also excluded a modest number of observations for reasons of data-consistency and date and link quality. We analyze the fertility of women in their first marriage, and include women with both full and curtailed reproductive periods; women who married yet died before menopause or who lost their husband before menopause (here defined as age 50) were also included. Since our analyses concern inter-birth intervals, the time at risk of the mother begins with the previous birth. Furthermore, the time at risk of the mother is right censored at age 50 or if the mother or her husband died before the mother reaches age 50. Inter-birth intervals are typically longer than first-birth intervals due to the delayed return to fecundability after a period of amenorrhea associated with breastfeeding. If the inter-birth interval was more than 5 years long, an intervening birth may have been missed. In these cases, we censored the interval at 5 years, with subsequent intervals retained for analysis.

The RPQA analysis includes only subjects and husbands born in Quebec, includes all parish-to-parish migrants, and excludes only a small number of women or children who emigrated from Quebec. The maternal and paternal grandmothers are examined in separate models, and for each model, we select the grandmothers whose death date is known or whose date

of outmigration is known (in the case of Scania). Since the number of maternal and paternal grandmothers whose death date is known varies, the total number of observations per dataset varies somewhat per model.

Method

We used Cox proportional hazard models for all analyses. We employ models without and with family fixed effects (based upon observations grouped by sibship), stratifying on the grandmother (F0). Results derived from models which do not incorporate fixed effects reflect differences across all families, potentially indicating healthful or detrimental behaviors on the part of particular families. Our fixed effects models, on the other hand, control for inter-familial variations and thus focus on differences within groups of sisters or sisters-in-law. To better compare our fixed effects results and non-fixed effects results, all regression analyses have constrained the denominator for all models to mothers with at least one sister or sister-in-law. We present results for all birth intervals together (birth intervals 2+), as well as results specifically for the 2nd and 3rd birth intervals, birth intervals 4 and higher, and birth intervals 9 and higher (for Quebec and Utah only; the number of observations in the Scania data do not allow for analyses of birth intervals 9 and higher).

Variable Operationalization

The dependent variable of prime interest is the time to the next birth, with all inter-birth intervals considered together or stratified by birth order. Our independent variables of interest are the vital status and proximity of the maternal and paternal grandmothers. These are time-varying variables, since the vital status and proximity of the grandmother will change in the course of her daughter or daughter-in-law's reproductive life and in the course of her grandchildren's youth. Since the Scania data encompass five parishes, grandmothers who migrated out of the 5-parish region have unknown destinies, and thus are given a unique 5th value "outmigrated". The Utah database distinguishes between grandmothers who were alive and living in the same county from those who were alive and living in a different county. The location of the grandmother is determined by comparing the time and place of their death to the birth parish of the last grandchild born prior to the grandmother's death (Quebec) or the closest county among all the F1's births (Utah). In Scania the vital and proximity status of the grandmother is determined by considering whether she resided in the same parish as the mother and her date of death (if the death occurred in the 5-parish region). The grandmother variables thus include the following values:

i. Grandmother alive & in same parish
ii. Grandmother alive & in different parish/county (Quebec & Utah only)
iii. Grandmother died 0–4 years ago (Quebec & Utah; Scania: died in same 5-parish region)
iv. Outmigrated from the parish & status unknown (Scania only)
v. Grandmother died more than 5 years ago (Quebec & Utah; Scania: died in same 5-parish region) (This value serves as the reference group)

Since grandmothers' attention to their daughters and grandchildren was potentially diffused across a variety of children and grandchildren (brothers and sisters of the F1 and cousins of the F2), we also control for the size of ego's or her husband's sibship (time-varying). These controls are applied in models that did not use fixed effects. When we use a fixed effects specification this variable drops out and is accounted for by the fixed effects. When analyzing the role of maternal grandmothers, we control for the size of ego's sibship, and when analyzing the role of paternal grandmothers, we control for the size of ego's husband's sibship. When using fixed effects models, we do not need to control for the size of ego's or her husband's sibship since these do not vary for a given grandmother.

We include in our models a range of control variables: the age of the mother at the previous birth (continuous), and the age of the mother at previous birth squared (continuous), the birth rank of the mother (continuous), the birth rank of the previous child (continuous), whether the previously-born child died before age 1 (time-varying and continuous), current year or mother's year of birth to represent the historical period (continuous and time-varying), and urban versus rural place of birth/marriage of the mother (Quebec and Utah only). In the Quebec St. Lawrence Valley data, death dates are not known for many children (F2) born toward the end of our study period, requiring an adjustment to the "previously-born child died before age 1" variable. In the absence of a death date, we used date of marriage to identify children who had not died in infancy. Children for whom neither a death nor a marriage is recorded are classified in a separate value "destiny unknown". We omit ego's mother's and father's ages at death, as this information is unknown in the SEDD database. The Utah analyses also control for membership in the Church of Jesus Christ of Latter-day Saints. Since the Catholic populations included in both Quebec databases are generally homogeneous with respect to high fertility behavior, there is no religion variable included in the Quebec data. Similarly, religion is not included for Scania, as almost the entire population belonged to the Lutheran state church (Bengtsson and Dribe 2014).

Description of Results

Descriptive Statistics

The four regions represented in our study encompass a variety of historic settings, periods and conditions. Accordingly, both similarities and differences across our populations are evident in the means and percentage distributions for the demographic and social characteristics included in our analyses (Tables 2 and 3). These descriptive statistics are weighted by the person-years under observation, and stratified by birth intervals, and encompass all mothers (F1) in analysis, whether or not they had a sister or sister-in-law. Whereas the Scanian data include entirely rural parishes (save for the last decades of the nineteenth century when one of the parishes grew into a small town), both Quebec files include a modest proportion of urban dwellers (up to a quarter in the case of the Saguenay region). On the other hand, about 40% of Utah residents lived within the Wasatch Front, four counties that would evolve into today's urban corridor. Another distinction of the Utah population is that nearly two-thirds of this population were members of the Church of Jesus Christ of Latter-day Saints; active members of this church generally have high fertility, do not smoke or consume alcohol, are socially integrated via their church activities, and they often fast one day a month, all behaviors that promote health and enhance fertility.

Table 2: Descriptive statistics (means and percentages), Maternal Grandmother, by region and birth intervals Scania, Quebec-St. Lawrence, Quebec-Saguenay Lac St. Jean and Utah, 1650–1900

	Birth Intervals 2+				Birth Intervals 2 & 3				Birth Intervals 4+				Birth Intervals 9+		
	Scania	Q-SL	Q-Sag	Utah	Scania	Q-SL	Q-Sag	Utah	Scania	Q-SL	Q-Sag	Utah	Q-SL	Q-Sag	Utah
Maternal grandmother status (%)															
Alive & living in same parish/county	50.81	27.64	28.90	50.72	63.51	35.77	35.37	56.53	44.15	25.36	27.36	47.59	20.73	23.25	38.54
Alive & living in different parish/county		27.83	33.13	23.44		32.00	38.93	24.92		26.66	31.75	22.64	22.75	27.13	20.48
Dead 0–4 years	11.21	10.55	9.52	6.05	9.39	8.71	8.13	4.55	12.15	11.06	9.85	6.86	12.53	11.41	9.06
Dead 5+ years	33.03	33.99	28.44	19.79	21.84	23.52	17.57	14.00	38.90	36.92	31.04	22.91	43.99	38.21	31.92
Outmigrated	4.95				5.26				4.79						
Previously-born child died before age 1 (%)	9.18	20.46	22.73	6.89	9.65	17.95	22.13	6.43	8.93	21.17	22.87	7.14	26.58	26.26	8.90
Previously-born child - destiny unknown (%)		8.58				7.59				8.86			9.01		
Mother's age at previous birth	32.62	30.41	30.59	28.61	27.66	23.66	22.95	23.63	35.22	32.31	32.42	31.11	36.68	36.76	36.95
Number of children ever born	3.95	6.83	7.21	5.45	1.49	2.51	2.51	2.52	5.24	8.08	8.33	6.64	10.61	11.32	10.39
Mother’s sibship size	5.59	9.43	9.70	7.91	5.55	9.36	9.58	7.80	5.60	9.55	9.73	7.96	9.70	9.87	8.14
Mother’s birth rank	3.47	5.06	4.63	4.68	3.46	5.05	4.60	4.74	3.47	5.10	4.64	4.65	5.10	4.71	4.56
Year (Utah: Mother's year of birth)	1849	1753	1913	1910	1847	1747	1905	1907	1850	1754	1914	1912	1758	1919	1915
Urban residence (Utah: = Wasatch) (%)		16.99	23.21	39.48		18.00	18.75	42.21		16.70	24.27	38.01	16.62	26.66	34.25
Member of Latter-day Saints Church (%)				37.33				60.17				64.01			64.76
Number of births	2,865	71,166	42,604	125,750	1,203	18,642	10,101	46,254	1,662	52,524	32,503	79,496	17,625	11,913	13,715
Number of mothers	927	9,921	5,169	26,362	886	6,250	5,169	25,609	673	3,671	4,655	20,792	672	3,069	5,877

Q-SL = Quebec - St. Lawrence Valley
Q-Sag = Quebec Saguenay Lac St-Jean region
Blank cells = information not available or not applicable
Scania: "same parish" = 5-parish region (very local)
Sources: Scania: SEDD 2017; Q-SL: PRDH - RPQA 2014; Q-Sag: BALSAC 2017; Utah: UPDB 2014

Table 3: Descriptive statistics (means and percentages), Paternal Grandmother, by region and birth intervals Scania, Quebec-St. Lawrence, Quebec-Saguenay Lac St. Jean and Utah, 1650–1900

	Birth Intervals 2+				Birth Intervals 2 & 3				Birth Intervals 4+				Birth Intervals 9+		
	Scania	Q-SL	Q-Sag	Utah	Scania	Q-SL	Q-Sag	Utah	Scania	Q-SL	Q-Sag	Utah	Q-SL	Q-Sag	Utah
Alive															
Alive & living in same parish/county	40.36	21.29	31.90	45.53	55.80	28.29	42.30	52.20	32.39	19.19	29.50	41.94	14.01	22.40	31.650
Alive & living in different parish/county		24.37	23.50	21.71		32.41	29.10	24.18		22.16	22.20	8.10	15.99	18.70	16.780
Dead 0–4 years	11.20	12.37	11.00	7.17	11.34	10.39	8.60	5.44	11.13	12.91	11.60	29.58	13.94	13.40	10.420
Dead 5+ years	43.13	41.97	33.50	25.59	27.63	28.29	20.00	18.18	51.14	45.74	36.70	20.38	56.06	45.60	41.150
Outmigrated	5.30				5.22				5.34						
Previously-born child died before age 1 (%)	8.60	20.19	22.70	6.88	8.82	17.48	21.90	6.39	8.48	20.93	22.90	7.15	26.36	26.00	8.940
Previously-born child - destiny unknown (%)		8.35				7.16				8.68			8.96		
Mother's age at previous birth	32.63	30.48	30.60	28.62	27.63	23.62	22.80	23.63	35.21	32.37	32.40	31.12	36.73	36.80	36.960
Number of children ever born	3.97	6.87	7.25	5.48	1.51	2.51	2.51	2.52	5.24	8.08	8.35	6.65	11.29	11.34	10.390
Father’s sibship size	5.61	9.48	9.40	7.42	5.47	9.38	9.30	7.31	5.68	9.51	9.40	7.47	9.61	9.51	7.580
Year (Utah: Mother's year of birth)	1854	1754	1913	1910	1851	1749	1905	1907	1855	1756	1914	1912	1760	1919	1915
Urban residence (Utah: = Wasatch) (%)		13.63	23.00	39.34		14.45	18.30	42.00		13.40	24.10	37.91	13.72	26.43	34.820
Member of Latter-day Saints Church (%)				63.21				60.81				64.50			65.180
Number of births	2,313	62,050	39,963	122,910	969	16,158	9,384	45,163	1,344	45,892	30,579	77,747	15,474	11,253	13,376
Number of mothers	761	8,508	4,795	25,713	714	5,311	4,777	24,981	545	3,197	4,356	20,331	590	2,880	5,743

Q-SL = Quebec - St. Lawrence Valley
Q-Sag = Quebec Saguenay Lac St-Jean region
Blank cells = information not available or not applicable
Scania: "same parish" = 5-parish region (very local)
Sources: Scania: SEDD 2017; Q-SL: PRDH - RPQA 2014; Q-Sag: BALSAC 2017; Utah: UPDB 2014

A comparison of demographic indicators across our four populations reveal one important difference: higher infant mortality in both Quebec populations, measured as the percent of previously-born children who died before the age of 1 year. In the case of the Quebec St. Lawrence Valley population, considering the percent of children known to have died before age 1 together with those whose destiny is unknown (but which may include some infant deaths), from 20% to 29% of children born in the previous birth interval died in infancy, and this percentage rose to a minimum of 26% and a maximum of 36% for birth intervals 9+ (Tables 2 & 3, Q-SL). This high level of infant mortality in colonial Quebec is indicated in a 2010 study of Quebec infant mortality from 1640 to 1779 (Amorevieta-Gentil 2010: 131) and is a sharp contrast to the experience in Utah (Bean et al 2002). Amorevieta-Gentil identified steadily rising infant mortality rates in Quebec across the eighteenth century, with frequent mortality spikes resulting mostly from epidemic diseases such as smallpox, typhus, or measles, as well as social and health crises resulting from the British Conquest (1756-1763) (Amorevieta-Gentil 2010; Bruckner et al. 2018). Infant mortality rates between 1765 and 1779 ranged from 250 to 350 per thousand (Amorevieta-Gentil 2010: 131). The levels of infant mortality were somewhat lower in the Quebec Saguenay region (23%), since the data for this region extend into the mid-twentieth century, when fertility was still high but public health measures had begun to lower the infant death rate (Gaumer and Authier 1996). Infant mortality in the Quebec Saguenay region was nevertheless higher than that exhibited in either Scania or Utah. In these two regions, the percentage of previously-born children who died before age 1 was about 9% and 7% respectively, a result which may reflect improved health practices or conditions. Another notable difference across our populations concerns the number of children ever born and included in analysis, which is about 7 children in the case of the two Quebec populations (birth intervals 2+), 5.5 children in the case of Utah and 4 children in the case of Scania. Accordingly, mother's and father's sibship size is generally higher in the two Quebec populations (about 9 to 10, Tables 2 & 3) and in Utah (9) but lower in Scania (6 for both mothers and fathers). Mother's age at previous birth is slightly lower in the two Quebec populations (30-31 years) compared to the Utah and Scanian populations (about 33).

Finally, we present the percent distribution of maternal and paternal grandmothers' vital status and proximity across birth intervals. For the data concerning birth intervals 2+, maternal grandmothers in Utah are distinguished by greater survival: 75% of maternal grandmothers in Utah were alive, compared to just 55% of maternal grandmothers in Quebec-St. Lawrence Valley. Maternal grandmothers in the Quebec Saguenay region, on the other hand, almost matched their Utah counterparts in terms of survival (62% were alive for birth intervals 2+). Similar regional differences across North America prevailed for the paternal grandmothers (Table 3), at lower percentages across the board (e.g. 67% in Utah, 55% in Quebec Saguenay and 46% in Quebec St. Lawrence Valley). Since the Scania data identifies maternal and paternal grandmothers as alive only if they were also living in one of the five rural parishes, we cannot make exactly the same survival comparison that we did with the Quebec and Utah grandmothers, but we do see that 40–50% of paternal and maternal grandmothers in Scania were both alive and living in proximity to their daughters or daughters-in-law (F1). About 5% of maternal and paternal grandmothers in Scania are indicated as having outmigrated from the area of study. Half of the grandmothers in Utah were not only alive but also living in the same county as their daughter. In the two Quebec regions, on the other hand, just over a quarter of grandmothers were both alive and living within the same parish as their own daughter. In the Saguenay region, nearly a third of paternal

grandmothers were both alive and living in the same parish as their daughters-in-law, but the same was true for only 21% of St. Lawrence Valley paternal grandmothers. Notwithstanding the tendency of Quebec families to transmit property through the male line, somewhat larger proportions of Quebec St. Lawrence Valley women resided in proximity to their own mother (26%) than to their mother-in-law (21%). This distinction was in part related to the larger percentages of paternal grandmothers who had already died (56% compared to 46% of maternal grandmothers). In all cases, at higher birth intervals, larger percentages of grandmothers were distributed in the "Dead 0–4 years" and "Dead 5+ years" categories, representing how the passage of time would gradually deprive mothers of their potential helpers.

Multivariate Analysis

Across the regions included in our study, we observe significant and positive influences of both maternal and paternal grandmothers on fertility outcomes of their daughters. The strength and consistency of these effects vary, but it is noteworthy that the grandmother effect is observable across such diverse contexts. In most cases, we show non-fixed effects and then fixed effects versions of our models; applying fixed effects often increases the strength of our positive hazard ratios for fertility, but not always. These distinctions are important, as significant positive hazard ratios in fixed-effects models indicate differences across sisters, whereas significant positive hazard ratios in non-fixed effects models indicate differences across families. Tables 4 and 5 present abridged results, focusing on the variables of interest "Maternal Grandmother Status" and "Paternal Grandmother Status"; appendices A and B present the full results for Tables 4 and 5, including all control variables.

Our internationally comparative analysis has demonstrated a positive association of grandmothers' availability and presence with daughters' fertility (shorter birth intervals); nevertheless, the context and timing of this effect differed across our populations. In Scania, living grandmothers promoted higher-order fertility (4+). This effect was similar for maternal and paternal grandmothers, but was evident in non-fixed effects models only, suggesting differences across families were more important in the Scanian context. Among Scanian women bearing children of birth rank 4 and higher, those whose mother or mother-in-law was alive and living in the same parish experienced a 1.123 to 1.162 times higher hazard of next birth compared to those whose mother or mother-in-law had died more than five years earlier (Table 4, model 17 and Table 5, model 47). This effect was manifested more broadly in the case of Scanian paternal grandmothers, for all birth intervals (2+): we observe that women whose mother-in-law was alive and living in the same parish had a 14% higher hazard of next birth compared to women whose mother-in-law died more than five years earlier (Table 5, model 31). Paternal grandmothers in Scania were also associated with fertility-enhancing effects if they had outmigrated or if they had died recently rather than 5+ years ago. In fact, the hazard of next birth was higher if the paternal grandmother had died recently or had outmigrated than if she was alive (Table 5 models 31, 39 and 47). Our Scanian analyses pertaining to maternal grandmothers manifest somewhat different patterns. We observe a fertility-enhancing effect of available maternal grandmothers only in the case of women giving birth to children of birth rank 4+, once again in a non-fixed effect model (Table 4, model 17). More generally, maternal grandmothers in Scania who had outmigrated were associated with longer birth intervals (Table 4, models 5 and 21); this was also the case of Scanian maternal grandmothers who had died recently (Table 4 model 5). Thus, we observe in Scania a reasonably consistent fertility-enhancing effect of living paternal grandmothers juxtaposed with a fertility-diminishing effect of deceased or outmigrated maternal grandmothers.

Table 4: Women's risk of next birth, by maternal grandmother status Scania, Quebec and Utah, 1650–1900, Abridged Table

Maternal Grandmother status (ref: dead 5+ years)	Scania		Q-SL		Q-Sag		Utah	
	H.R.	P>z	H.R.	P>z	H.R.	P>z	H.R.	P>z
Birth Intervals 2+, No Fixed Effects	1*		2		3		4	
Alive & in same parish / county	1.071	0.159	1.052	0.000	1.022	0.216	1.012	0
Alive & in different parish / county			1.065	0.000	1.028	0.107	0.997	0.468
Dead 0–4 years	1.037	0.586	1.040	0.016	1.013	0.577	1.016	0
Outmigrated (Sweden only)	0.971	0.665						
Birth Intervals 2+, Fixed Effects	5		6		7		8	
Alive & in same parish / county	0.885	0.299	1.111	0.000	1.081	0.032	1.025	0.035
Alive & in different parish / county			1.131	0.000	1.094	0.014	1.002	0.842
Dead 0–4 years	0.821	0.039	1.083	0.000	1.062	0.049	1.025	0.062
Outmigrated	0.501	0.000						
Birth Intervals 2 & 3, No Fixed Effects	9		10		11		12	
Alive & in same parish / county	0.997	0.973	0.992	0.738	1.025	0.540	0.998	0.67
Alive & in different parish / county			1.036	0.176	1.039	0.338	0.976	0.01
Dead 0–4 years	0.974	0.825	1.004	0.918	1.141	0.017	1.006	0.368
Outmigrated	0.981	0.860						
Birth Intervals 2 & 3, Fixed Effects	13		14		15		16	
Alive & in same parish / county	1.142	0.641	1.163	0.063	1.065	0.592	1.028	0.326
Alive & in different parish / county			1.288	0.000	1.141	0.268	1.001	0.978
Dead 0–4 years	1.084	0.731	1.107	0.135	1.176	0.102	1.03	0.335
Outmigrated	0.703	0.314						
Birth Intervals 4+, No Fixed Effects	17		18		19		20	
Alive & in same parish / county	1.123	0.057	1.064	0.000	1.019	0.330	1.015	0
Alive & in different parish / county			1.069	0.000	1.025	0.195	1.002	0.668
Dead 0–4 years	1.071	0.399	1.046	0.010	0.983	0.510	1.019	0
Outmigrated	0.974	0.764						
Birth Intervals 4+, Fixed Effects	21		22		23		24	
Alive & in same parish / county	0.950	0.758	1.104	0.000	1.108	0.017	1.03	0.054
Alive & in different parish / county			1.098	0.002	1.097	0.033	1.004	0.782
Dead 0–4 years	0.820	0.117	1.083	0.000	1.037	0.314	1.028	0.108
Outmigrated	0.636	0.043						
Birth Intervals 9+, No Fixed Effects			25		26		27	
Alive & in same parish / county			1.069	0.007	1.045	0.157	1.024	0.013
Alive & in different parish / county			1.088	0.000	1.021	0.491	1.025	0.079
Dead 0–4 years			1.056	0.056	1.008	0.831	1.037	0.004

*Numbers in abridged table refer to models in Appendix A
Q-SL = Quebec - St. Lawrence Valley
Q-Sag = Quebec Saguenay Lac St-Jean region
Blank cells = information not available or not applicable
Scania: "same parish" = 5-parish region (very local)
Sources: Scania: SEDD 2017; Q-SL: PRDH - RPQA 2014; Q-Sag: BALSAC 2017; Utah: UPDB 2014

Table 5: Women's risk of next birth, by paternal grandmother status
Scania, Quebec and Utah, 1650–1900, Abridged Table

Paternal Grandmother status (ref: dead 5+ years)	Scania		Q-SL		Q-Sag		Utah	
	H.R.	P>z	H.R.	P>z	H.R.	P>z	H.R.	P>z
Birth Intervals 2+, No Fixed Effects	31*		32		33		34	
Alive & in same parish / county	1.142	0.014	1.092	0	1.116	0.000	1.019	0
Alive & in different parish / county			1.092	0	1.094	0.000	1.014	0.001
Dead 0–4 years	1.200	0.015	1.067	0	1.052	0.027	1.021	0
Outmigrated	1.052	0.704						
Birth Intervals 2+, Fixed Effects	35		36		37		38	
Alive & in same parish / county	0.975	0.847	1.061	0.041	1.019	0.587	1.034	0.004
Alive & in different parish / county			1.091	0.003	1.040	0.304	1.024	0.034
Dead 0–4 years	1.037	0.736	1.081	0.000	1.025	0.407	1.035	0.01
Outmigrated	0.526	0.488						
Birth Intervals 2 & 3, No Fixed Effects	39		40		41		42	
Alive & in same parish / county	1.107	0.245	1.062	0.025	1.081	0.042	1.011	0.066
Alive & in different parish / county			1.043	0.105	1.064	0.129	1.019	0.051
Dead 0–4 years	1.157	0.243	1.076	0.041	1.072	0.199	1.009	0.16
Outmigrated	1.438	0.095						
Birth Intervals 2 & 3, Fixed Effects	43		44		45		46	
Alive & in same parish / county	0.862	0.621	1.125	0.177	0.941	0.593	1.045	0.124
Alive & in different parish / county			1.128	0.168	0.978	0.858	1.037	0.186
Dead 0–4 years	0.932	0.780	1.165	0.030	1.096	0.347	1.043	0.18
Outmigrated	3.00E+12	1.000						
Birth Intervals 4+, No Fixed Effects	47		48		49		50	
Alive & in same parish / county	1.162	0.031	1.095	0.000	1.125	0.000	1.02	0
Alive & in different parish / county			1.107	0.000	1.099	0.000	1.012	0.015
Dead 0–4 years	1.268	0.012	1.064	0.000	1.041	0.109	1.025	0
Outmigrated	0.908	0.572						
Birth Intervals 4+, Fixed Effects	51		52		53		54	
Alive & in same parish / county	0.830	0.334	1.008	0.816	1.023	0.591	1.039	0.012
Alive & in different parish / county			1.069	0.058	1.028	0.532	1.022	0.116
Dead 0–4 years	1.091	0.557	1.061	0.024	1.002	0.964	1.043	0.017
Outmigrated	0.000	1.000						
Birth Intervals 9+, No Fixed Effects			55		56		57	
Alive & in same parish / county			1.073	0.013	1.225	0.000	1.026	0.007
Alive & in different parish / county			1.151	0.000	1.133	0.000	1.022	0.128
Dead 0–4 years			1.099	0.001	1.032	0.403	1.009	0.469

*Numbers in abridged table refer to models in Appendix B
Q-SL = Quebec - St. Lawrence Valley
Q-Sag = Quebec Saguenay Lac St-Jean region
Blank cells = information not available or not applicable
Scania: "same parish" = 5-parish region (very local)
Sources: Scania: SEDD 2017; Q-SL: PRDH - RPQA 2014; Q-Sag: BALSAC 2017; Utah: UPDB 2014

When we cross the Atlantic Ocean to the seventeenth- and eighteenth-century Quebec St. Lawrence Valley settlement, we observe stronger and more consistent associations between the presence of a grandmother and the risk of next birth. Maternal and paternal grandmothers in the Quebec St. Lawrence Valley region who were alive and living in the same or a different parish were almost always associated with a higher hazard of next birth, for most birth intervals. In the analyses focusing on maternal grandmothers, effect sizes were strengthened in fixed effect models, suggesting that maternal grandmother availability differentiated fertility outcomes across sets of sisters more than it did across families. In the fixed effects model for all birth ranks 2+, Quebec St. Lawrence Valley women whose own mother was alive and living in the same or different parish had a 11% and 13% higher hazard of next birth compared to women whose mother had died more than five years ago (Table 4, model 6). The highest hazard ratios observed in the maternal grandmother analyses concern birth intervals 2 & 3 in the fixed effects models (hazard ratios of 1.163 and 1.288 for alive & in same or different parish respectively, although the former hazard ratio has a significance level of .063, just above the .05 threshold; Table 4 model 14). Quebec St. Lawrence Valley women whose mothers-in-law were alive also manifested higher fertility. For example, women giving birth to children of birth ranks 4+ had a 10–11% higher hazard of next birth if their mother-in-law was alive, regardless of her proximity status. The fact that the hazard ratios in the paternal grandmother analyses demonstrate slightly higher effect sizes, contrary to the maternal grandmother analyses, suggest that paternal grandmother availability made more of a difference across families. Even women whose mother or mother-in-law had recently died manifested modestly higher hazards of next birth compared to women whose mother or mother-in-law had died more than five years earlier. Overall, we view fertility-enhancing effects of both maternal and paternal grandmothers in the Quebec St. Lawrence Valley population, with somewhat greater effect sizes observed with respect to maternal grandmothers.

Moving forward in time to the nineteenth- and twentieth-century Saguenay, Quebec, region, we once again observe positive and statistically significant associations between grandmothers' availability and women's time to next birth. In the case of this population, the "grandmother effect" is more evident with respect to paternal grandmothers and in non-fixed effects models rather than fixed effects models. Women giving birth to children of any birth rank 2+ had a higher hazard of next birth if their mother-in-law was alive and living in proximity (hazard ratio 1.116) or if their mother-in-law was alive but living in a different parish (hazard ratio 1.094). These women also had a slightly higher hazard of next birth if their mother-in-law had only recently died (hazard ratio 1.052, Table 5, model 33, non-fixed effect model). The same associations are demonstrated in the non-fixed effects models for birth intervals 4+ and 9+, with the highest effect sizes demonstrated for the highest birth intervals of 9+: in this latter case, Saguenay women had a 23% and 13% higher hazard of next birth if their mother-in-law was alive and living in the same parish or in a different parish (Table 5, model 56). Maternal grandmothers in the nineteenth-century Saguenay region were also associated with fertility-enhancing effects, but less consistently, and more often in fixed effect models. For example, women giving birth to children of any birth rank (2+) had a 8% higher hazard of next birth if their own mother was alive and living in the same parish, and a 9% higher hazard of next birth if their mother was alive and living in a different parish (Table 4, fixed effects model 7). Maternal grandmothers were also associated with a faster time to next birth for birth intervals

4+ (Table 4, fixed effects model 23). Despite the existence of a positive grandmother effect for both maternal and paternal grandmothers in the Saguenay region, we observe generally larger effect sizes in the cases of paternal grandmothers. As observed in the Quebec St. Lawrence Valley population, non-fixed effect models yielded more important results with respect to paternal grandmothers in the Saguenay region, whereas significant maternal grandmother effects in the Saguenay emerge in the fixed effect models.

In late-nineteenth- and early twentieth-century Utah, we find fertility-enhancing effects mainly among paternal grandmothers; effects which are more modest in terms of effect size than those observed in the two Quebec regions and in the Scania region. The general trends observed among paternal grandmothers in Utah are nevertheless similar to those observed in the Quebec populations: women giving birth to children of birth ranks 2 or more had a marginally higher (hazard ratios of 1.02 and 1.01, respectively) hazard of next birth if their mother-in-law was alive and living in the same or in a different parish (Table 5, model 34). These results were true in both fixed effects and non-fixed effects models (Table 5, models 34 and 38). When examining specific birth intervals, we observe positive paternal grandmother effects in Utah with respect to birth intervals 4+ and 9+ (Table 5, models 50, 54 and 57). Maternal grandmothers in Utah were likewise associated with modest, though statistically-significant, fertility-enhancing effects. We observe a small positive association between the presence and proximity of a maternal grandmother and fertility generally for birth ranks 2+ (hazard ratios of 1.01 or 1.02, either fixed effects or non-fixed effects models, Table 4, models 7 and 8), for birth intervals 4+ and for birth intervals 9+ (non-fixed effects in both cases, Table 4, models 20 and 27).

Our multivariate analysis included a range of demographic control variables (mother's age at previous birth, mother's age at previous birth squared, mother's or father's sibship size, mother's birth rank, the birth rank of the previously-born child and whether the previously-born child had died before age 1) as well as several contextual variables (year, religious affiliation for Utah and urban-rural status for Quebec and Utah) (Appendices A and B). The hazard of next birth was influenced in predictable ways by our demographic control variables, and we present here the main contrasts across our four populations. Across all variables, including our variable of interest, the strongest positive hazard of next birth was associated with the death of the previously-born child. Infant mortality interrupted breast-feeding, prompting an early return of menstruation and risk of conception. In Scania, women whose previous child died before age 1 had as much as five times the hazard of next birth compared to women whose previous child survived (Appendix A, maternal grandmother analysis, birth intervals 2–3, model 13). The same variable in the Quebec and Utah models also manifested a strong positive association with the hazard of next birth, though the size of the hazard ratios for each North American region was smaller, reaching no more than 2.126 in the case of Quebec St. Lawrence Valley (Appendix A, maternal grandmother, model 14). Mother's age at previous birth was also strongly and positively associated with the hazard of next birth. The age association is U-shaped, with highest hazard ratios observed in the early twenties; thus this continuous variable, entered into models which also control for the birth rank of the previous child, indicates that women's waiting time to next birth was shorter as they aged from the late teenage years into their twenties. Once again, we see somewhat higher hazard ratios in the case of Scania than in Quebec and Utah (see, for example, hazard ratios of 1.399 in Scania versus the hazard ratios 1.203, 1.133 and .979 in Quebec St. Lawrence, Quebec Saguenay and Utah respectively, Appendix A, maternal grandmother

analysis, birth intervals 2+, models 5, 6, 7 and 8). In the case of the two Quebec populations, higher birth ranks of the previously-born child were associated with lower hazards of next birth, particularly for birth intervals 2 & 3 in the fixed effects models (hazard ratios of .617 to .633 Appendices A and B, models 14, 15, 44 and 45). In the non-fixed effects models, we included mother's or father's sibship size to see if the grandmother effect was diluted for mothers with many siblings or siblings-in-law. The hazard ratios for this variable, though almost always above 1 and statistically significant, were very modest, ranging from 1 in Utah (Appendix A, model 27, maternal grandmother, birth intervals 9+ and Appendix B model 42, paternal grandmother, birth intervals 2 and 3) to 1.030 in the Saguenay region (Appendix A, model 11, maternal grandmother, birth intervals 2 & 3; exceptionally, one hazard ratio below the value of one, .682, is evident in the Saguenay population, Appendix B, model 41, paternal grandmother, birth intervals 2 and 3).

Our contextual control variables did not yield many notable results in terms of the size of the hazard ratio. For all four regions, the association of period (year as a time-varying continuous variable) with the hazard of next birth was often statistically significant but the hazard ratios were usually just under or just over 1. The hazard ratios for period were more often statistically significant in the fixed effects models, and tended to decrease when fixed effects were applied. For example, in the case of the maternal grandmother analyses, birth ranks 2+, non-fixed effects models, the hazard of next birth either increased or decreased each year by a factor of 1.000, .999, 1 and .998 in the case of Scania, Quebec St. Lawrence, Quebec Saguenay and Utah respectively (Appendix A, models 1, 2, 3 and 4; only the results for models 2 and 4, Quebec St. Lawrence and Utah, were statistically significant). The same model with fixed effects applied demonstrate a modest shift to hazard ratios consistently below one: .983, .988, .990 and .994 for these four regions (Appendix A, models 5, 6, 7 and 8). Residence in urban areas in the Quebec St. Lawrence population was generally associated with a very small increase in the hazard of next birth, almost always in non-fixed effects models. For example, in the maternal and paternal grandmother analyses concerning all birth ranks 2+, Quebec St. Lawrence women had a 1.032 and 1.024 higher hazard of next birth if they lived in an urban area (Appendix A, model 2 and Appendix B, model 32). The Quebec-Saguenay population, covering women born in a later period but living in a largely rural area, manifests instead the more usual negative association of urban or semi-urban residence with fertility; however, these results were statistically significant only in analyses concerning the highest birth orders, 9+. In the maternal grandmother analyses, Saguenay women giving birth to children of rank 9+ had a .923 and .987 hazard of next birth if they lived in a semi-urban or urban place of residence compared to their rural-dwelling counterparts (Appendix A, model 29, fixed effects; a similar result, .927, prevails in the case of urban-dwelling women, birth rank 9+ in the paternal grandmother analyses, Appendix B, model 56, non-fixed effects). In Utah, women who lived in the urban district of Wasatch had longer waiting times to next birth, particularly with respect to birth intervals 2 and 3, where the hazard ratio of next birth was .986 (maternal grandmother analysis) and .983 (paternal grandmother analysis) (Appendices A and B, fixed effects models 12 and 42). Conversely, in Utah active Mormon women consistently had a lower hazard of next birth. These hazards are modest and generally lower in the fixed effects models, with the lowest hazard ratio observed for birth ranks 2+, fixed effect model: .985 in the maternal grandmother analysis and .982 in the paternal grandmother analysis (Appendix A, model 8 and Appendix B, model 38).

Discussion and Conclusion

In this chapter we have taken the first steps towards a comparative analysis of the impact of grandmothers on the reproductive outcomes of their children. We have studied four different contexts in North America and Scandinavia for which we have longitudinal individual-level data spanning three generations over long periods of time. Several interesting findings have emerged. Taken together, our results point to important effects of kin on reproductive success of families, much in line with predictions from anthropological theories on cooperative breeding. Paternal grandmothers consistently promote fertility across all four regions, particularly in Scania, with the maternal grandmother marginally more important in the Quebec St. Lawrence region. Women whose mothers-in-law were alive had children at a faster pace than other women. The availability or recent death of the paternal grandmother was associated with a higher hazard of next birth in 4 out of 8 models for Scania, 6 out of 8 models for Quebec-St. Lawrence, 4 out of 8 models for Quebec-Saguenay, and 5 out of 8 models for the state of Utah. We also find a general fertility advantage associated with the availability or recent death of the maternal grandmother in the four populations, in 3 out of 8 models in Scania, 6 out of 8 models in Quebec-St. Lawrence, 5 out of 8 models for Utah and 2 out of 8 models for the Quebec-Saguenay region. These findings are consistent with theories on cooperative breeding which argue that the help women receive from grandmothers, and possibly also other kin, facilitates childbearing and induces couples to have more children. We nevertheless observe notable variations across these regions.

In Scania, paternal grandmothers were associated with fertility-enhancing effects if they were either present, or outmigrated or recently died; Scanian maternal grandmothers were associated with higher fertility outcomes for birth ranks 4 or higher. Maternal grandmothers did not show the same consistent impact on fertility as the paternal grandmothers across all regions, but they were important in colonial-era Quebec. The Quebec St. Lawrence Valley population demonstrated the most consistent maternal and paternal grandmother associations. In addition, in several models for this seventeenth- and eighteenth-century Quebec population, the hazard ratio is modestly stronger if the grandmother is not proximate rather than if she is. Both maternal and paternal grandmothers in the Saguenay region were associated with higher fertility, but the largest hazard ratios in the Saguenay models are evident for birth ranks 9+, rather than across different birth ranks as in the case of the Quebec St. Lawrence Valley. While maternal grandmothers showed fertility-enhancing effects in Utah, the effect sizes for Utah's maternal grandmothers were slightly smaller or not statistically significant compared to the results shown in the Utah paternal grandmother analysis.

Differences across these populations also emerge in terms of the relevance of using fixed effects models, which compare fertility outcomes across sets of sisters or sisters-in-law, or non-fixed effects models, which compare fertility outcomes across families. The fixed effect models accounted for genetic and environmental effects on both longevity and fertility which were potentially shared within families. The distinct relevance of fixed and non-fixed effect models across Scania, Quebec and Utah is important, as it may signal possible differences in the underlying mechanism of the grandmother effect in each of the four regions, as well as the relative importance of inter-familial heterogeneity in each country in terms of health, environment, social class and other factors. In Scania, the positive association of the paternal and maternal grandmother with fertility generally emerged in non-fixed effects models,

suggesting that fertility outcomes in this Swedish region were differentiated across families. Paternal grandmothers were associated with higher fertility even if they had recently died or had outmigrated. In this instance, the higher fertility of daughters-in-law may result from a positive selection in terms of the resources of the extended family which led the grandparents to move at an advanced age. At the same time, two non-fixed effects models for Scania indicate sisters' fertility outcomes diverged when the maternal grandmother was absent: mothers' fertility was lower if the maternal grandmother was recently dead or had outmigrated.

In the two Quebec populations, on the other hand, the use of stratified and non-stratified models illuminated important differences between maternal and paternal grandmothers. The highest hazards of next birth with respect to living maternal grandmothers were generally observed in fixed effects models, whereas the highest hazards of next birth when the paternal grandmother was alive were more often seen in the non-fixed effects models. The maternal grandmother analysis suggests greater differences across sisters within the same family than across the colony as a whole, and indicates as well that the fertility-enhancing effects of maternal grandmothers in historic Quebec were highly life course-dependent. At the beginning of their reproductive careers, women (F1) experienced higher fertility when they had access to their own mothers, the maternal grandmothers, and it did not much matter if their own mother lived in proximity. Perhaps maternal grandmothers in historic Quebec were willing to travel to help each successive daughter bear her first few children or to lend a hand during difficult periods. The maternal grandmother will likely want all of her daughters to succeed, and will make the accommodations and compensations necessary to distribute her assistance across her own daughters. However, the pertinence of the fixed effects models for the maternal grandmother analysis suggests that sisters who began their childbearing career while their own mother was still alive benefitted disproportionately from her assistance, relative to sisters who commenced childbearing in the absence of their mother. The assistance of the paternal grandmother, on the other hand, was not differentiated across her daughters-in-law, perhaps because her focus was indeed on lending assistance to her own daughters. The greater pertinence of non-fixed effects models in the paternal grandmother analysis suggests that her availability (even in a different parish or recently-dead) functioned as a proxy for support from a broader kinship network or from a family endowed with greater access to food or labour resources, such as a more-developed farm, or a farm located in an agriculturally-productive frontier location.

In Utah, fertility-enhancing grandmother effects were evident among both maternal and paternal grandmothers, in non-fixed and fixed effects models and at varying parity levels, with slightly higher size effects in the paternal grandmother analyses. The effect sizes observed in the Utah hazard ratios were notably smaller than those observed in the two Quebec populations as well as the Scania population. The Utah population encompasses nineteenth- as well as twentieth-century births, and as such portrays fertility patterns in a largely rural context which is nevertheless beginning to experience the demographic transition. The modest effect sizes shown in the Utah analyses may reflect contrasting influences on the part of Utah grandmothers: during the early stages of the demographic transition, some Utah grandmothers may have continued to encourage or facilitate high fertility while the "early innovators" were already beginning to discourage rapid childbearing (see Jennings, Sullivan & Hacker 2012). While the Scanian population also included mothers experiencing the early stages of the transition, the more important role of the paternal grandmother in Scania may

signal continued discrepancies across women in terms of the familial resources the paternal grandmother represented. The Quebec St. Lawrence Valley population, on the other hand, embodied a natural fertility regime while most of the families included in the Quebec Saguenay population would also have practiced natural fertility. As a result, Quebec women bore children across the breadth of their reproductive lifespan and shared their own mother, the maternal grandmother, with several sisters. Each woman's reproductive phase intersected with the vital status of the maternal grandmother to form a particular nexus; accordingly, sisters in Quebec were differently advantaged in terms of their reproductive outcomes. Inter-familial variations in the Quebec paternal grandmother analyses, as was the case in Scania, may be more clearly understood in terms of the differentiation of resources across families. Our findings, overall, highlight the importance of paternal and maternal grandmothers for reproductive behavior in quite diverse historic contexts. Grandmothers usually fostered high-fertility outcomes, but family configurations, the stages of the life course, resource allocation and the advent of fertility control mediated the relative importance of this effect.

Appendices A and B are available online at https://doi.org/10.11647/OBP.0251#resources

Appendix A: Women's risk of next birth, by select characteristics (maternal grandmother)

Appendix B: Women's risk of next birth, by select characteristics (paternal grandmother)

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21. The Challenges of Evolutionary Biodemography and the Example of Menopause

Shripad Tuljapurkar

Menopause in humans and post reproductive life in humans and other species challenge our understanding in demographic and evolutionary terms. This chapter outlines the questions that are key to an evolutionary understanding of menopause, and the failure of some well-known theories of aging to deal with these questions. The chapter then introduces and explains the concept of "borrowed fitness" in which post-reproductive ages can indirectly acquire fitness from reproductive ages. Several mechanisms for this kind of "borrowing" are then discussed, including the grandmother effect, the contributions of older males, and most generally, an approach based on the transfers from and to different ages, both reproductive and post-reproductive. We also discuss other theoretical advances in the understanding of the evolution of old age mortality. We suggest that further development of the transfer approach is the most likely to lead to advances in our understanding of the evolution of menopause.

Keywords: Menopause, Post-reproductive life, evolution, fitness, borrowed fitness, male success, grandmother hypothesis, transfers

Introduction

Biologists have been interested in life histories since, of course, Darwin (1859) and the biological analysis of longevity became important after the work of Medawar (1952). Fisher (1930), Lewontin (1965) and Williams (1966) showed that fitness was the result of an interplay between fertility and mortality, and Hamilton (1966) explained how changes in the age pattern of survival or reproduction could determine the strength of selection on a life history. Biodemography (Wachter and Finch 1997) originated from this base, and aims to integrate demography, anthropology, molecular and cellular biology, experimental and evolutionary ecology, and biomedicine, spurred on by funding aimed at improving human health and mortality.

Here I focus on evolutionary arguments about biodemography and in particular the example of extended lives and menopause. I first discuss the general questions that evolutionary arguments face in biodemography, especially the questions of what we seek to explain, and the time scales that we need to consider. Then I use the case of menopause to show how past work has produced a clearer understanding and a sharper framing of the questions and why much remains to be done. My focus is deliberately narrow and I do not mention or discuss here many valuable contributions that have informed and enlightened me.

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What do we seek to explain?

Evolution certainly did shape present-day human mortality. But how far back in history should we go to find a starting point: since the emergence of prokaryotes, or mammals, or primates, or hominids, or our own species, or modern humans? Using a really long-term view, the primordial life history (i.e. the initial condition) was clearly very simple, think a bacterium. The history of evolution has often (though not always) led to an increase in the genetic and phenotypic complexity of life histories (Bonner 1998), and so to the complex life cycles observed in the relatively recent past (as with history or museum collections) or in the present time. A constructive theory of life history evolution would, in my view, start with a primordial life history and establish conditions under which complex life cycles would evolve. Given a set of constraints, such a theory would predict something like the modern primate or human life cycle as a locally (or globally) stable equilibrium (or metastable equilibrium). In fact, the work I know does not attempt such a constructive theory of human life histories (or indeed of any other species). One possible approach to such a theory is to examine the sequence of positive mutations that lead to complex phenotypes and life cycles: while this has been done for some phenotypes (see, e.g., the discussion of the eye in Rogers 2011), there has been little similar work on life cycles. Another possible approach is to reverse engineer the results in Wachter et al. (2013) who show that under some conditions (e.g., recurrent deleterious mutation plus antagonistic pleiotropy) evolution can cause a complex life cycle to collapse into a simple one; while this may be going the wrong way, their results may provide clues to the reverse process. But in this chapter, in keeping with most other work, I assume that the starting point of evolutionary theory is a complex life cycle that is in some sense a precursor of a modern human life cycle. What historical time scale should we consider, what initial life cycle(s) should be used, and what should we seek to explain?

To see why time scale matters, note that modern humans have a generation time about 25 years, long compared with even most primates, so that only about 80 generations have gone by since the Roman Empire. Estimates of the strength of long-term natural selection are so small (Lewontin 1974) that significant genetic change is expected to take several hundred to several thousand generations. With Lewontin, I conclude that it is unlikely that genetic evolution shaped the last 2000 years of change in human mortality — in genetic terms we are surely not very different from the people of early Rome. Nonetheless, human longevity has certainly changed over that time, with a dramatic rise in the most recent 150 years or so. For example, the Human Mortality Database (http:/mortality.org) reports that Swedish life expectancy at birth rose from ~45 years (47 for females, 42 for males) in 1850 to ~82 years (84 for females, 80 for males) in 2015. This increase of over 82% in under 7 generations implies that mortality is very responsive to environmental change, or perhaps that selection has been extremely rapid, or both. We know that environmental factors such as better living standards and especially public health practices have led to a large decline in human mortality (starting in the 1850s, Szreter 2002, 2004). And it is hard to see why selection would be strong over this period: strong selection occurs, e.g., when disease or famine results in large and selective mortality, an episodic pattern that is not consistent with the recent decline in human mortality. I conclude that environmental change must have been the main driver in recent human mortality decline.

Following this reasoning, evolutionary arguments about human life history must take a longer-term view if they are to be useful. The goal of such arguments, then, is to start with a primate-like life cycle for humans before the dawn of agriculture, so over the past 100–1000 centuries, and explain why long-term evolution leads to the mortality and fertility patterns we observe over the most recent 5–10 centuries. We should also explain why mortality and fertility are so responsive to environmental factors. Of course, this approach still leaves us with the question, "what is the starting life cycle?" Not surprisingly, theories tend to be contingent — they focus on a particular feature (or features) of modern human life history, e.g., that females undergo menopause, or that female/male reproduction occupies only a limited age range, or that adult mortality increases exponentially with age in Gompertzian fashion, and seek explanations only of that (or those) feature(s). The starting life cycle is taken to be similar in most respects to that for modern humans, but different in the chosen feature(s). Then all theories face similar questions: how is fitness defined; in early/modern humans does the chosen feature "solve" some optimization "problem"; do mutation, evolution and/or constraints (trade-offs) lead to an "optimal" life history; if optimization is not used, does the direction of evolutionary dynamics lead from the chosen initial life history towards a recent human life history; is there empirical support for the theory?

Fortunately, we have theoretical tools (many developed by Charlesworth and reviewed in his 1994 book) that can be used to analyse some of the relevant evolutionary questions. Recent advances in these tools are described by Evans, Steinsaltz and Wachter (2013). But there are many challenges in applying these general tools to particular questions about aging. I focus here on a knotty evolutionary question — menopause.

Menopause and Post-reproductive Life

Evolutionary Puzzles About Menopause

An obvious but important fact is that biological selection acts only on phenotypes that affect biological fitness. Here, fitness is taken to be long-run growth rate; similar arguments can be made for a density-dependent situation. In humans, female fertility declines with increasing age and ends with menopause at age about 50 years, with modest variation in the latter age. If biological fitness depends only on female mortality and fertility, biological selection will be blind to phenotypic (and underlying genetic) traits at ages past menopause (Hamilton 1966). Because all individuals are continually subject to mutations of which most are deleterious (i.e. almost all mutations cause phenotypic change that increases mortality), a permanent loss of female reproduction after menopause should imply that humans have no fitness if they live past menopause. The resulting accumulation of deleterious mutations that act after menopause must therefore lead to high mortality and death at or shortly after the age at menopause (what Wachter et al. 2013 call "a wall of death"). In many non-human species, it is common to see such a sharply defined age at death when reproduction ends: witness examples such as the Pacific salmon or annual plants. However, this is certainly not true for humans. Gurven and Kaplan (2007) present data suggesting that early humans lived well past the age of menopause, and modern humans certainly do. Even a short but healthy human life after menopause needs evolutionary explanation. An adequate evolutionary explanation should likely also apply to some of the other social species that do have a post-reproductive life, such

as other terrestrial mammals and some marine mammals (Cohen 2004). In addition, human post-reproductive life has lengthened dramatically over the past two centuries. This change is a clear and remarkable case of evolutionary plasticity and also needs explanation, but that's a question that has not yet been addressed by evolutionary theory; later I do suggest a possible approach.

A related puzzle is that human mortality does not rise especially rapidly near menopause. Even in species without a post-reproductive life, late-age mortality does not always rise rapidly near the end of reproduction, as shown by Carey's (1992) finding of a late-age mortality plateau in medflies, an observation that has since been repeated for some other species of fruit flies (see papers in Carey and Tuljapurkar 2003), and in other species including nematodes and possibly humans (Vaupel et al. 1998). Empirical work in many other mammals (Gaillard et al. 1994) shows that there is a definite but gradual decline with age in mortality and fertility. Evolutionary arguments for these observations are also clearly needed but none have been made.

I note in passing that I restrict this discussion to evolutionary explanations. There are valuable papers (see e.g., the review in Wood 1994) that examine the mechanics of menopause in relation to the age-dependent decline in female fecundity. Taken as broader explanation, these theories lead to the view that menopause is merely an epiphenomenon of such a decline (Peccei 2001). While it is certainly the case that an analysis of rates of fecundity decline and their proximate causes (e.g., the rate of loss of viable follicles) is important, such analyses do not address the reasons why such rates or causes are evolutionarily stable for humans. Similar issues arise with optimality arguments about the rate of metabolic decline in *Drosophila* (Novoseltsev et al. 2001).

Post-reproductive Life Without Selection

I begin by briefly describing two approaches to menopause that are not (in my view) evolutionarily plausible arguments for long post-reproductive life. The first is the antagonistic pleiotropy argument (Williams 1957): this states that some alleles (of one gene, or perhaps of several genes) drive a trade-off in fitness components (survival, reproduction) in which late-age components decrease so that early age components can increase. Given that deleterious mutations are just as likely to affect old ages as young ones, and that antagonistic pleiotropy must weaken selection against mutations acting at old ages, it is hard to see why a life cycle built upon antagonistic pleiotropy would resist collapse to a simple limit. As noted in the discussion above of Wachter et al. (2013), the answer may lie in positive mutations, or perhaps in a kind of positive pleiotropy. But antagonistic pleiotropy is clearly not a useful evolutionary argument for post-reproductive life (for a very different perspective, see Olshansky and Carnes 2009).

The second argument has been championed by Olshansky and Carnes (1997, and many subsequent works). They start with the contingent view that for humans there is a tightly specified age range when menopause occurs, that fertility is zero after menopause and neither sex makes any contribution to anyone's reproductive success after that age. Hence in humans there would be no biological selection past menopause, but these authors argue that it is nonetheless possible to have a post-reproductive life (see e.g., Olshansky et al. 1998). The logic is that post-reproductive life best resembles the coasting of a post-mission spaceship.

If this argument is correct, we need only focus on two questions: what determines the age at menopause, and the post-reproductive period: how long, how healthy, and so on. These arguments say nothing about the timing of menopause but only speak to some aspects of the post-reproductive period.

Olshansky et al. (1997) argue for a "wearing-out" process in which the mechanical components of a human body, such as joints, simply wear out through repeated use and cannot be internally repaired. But it is not clear what determines the dynamics of the "wearing-out". We do know of cases, such as long-lived low-turnover proteins like crystallin in the human eye, in which relevant rates of decay are known (Toyama and Hetzer 2013), but we do not have such estimates for most components (however defined) of the human body, nor of variability in these rates. Decay rates for physiological systems are not well understood, though they are undoubtedly important. Since in this view there is no post-menopausal selection, we have no other biological principles or knowledge to predict mortality at post-menopausal ages, or indeed the length of life. It is tempting to appeal to a non-biological principle, e.g., reliability theory. But mortality predictions based on reliability (Gavrilov and Gavrilova 2001) can lead to quite arbitrary post-reproductive mortality patterns (Steinsaltz and Evans 2004). To sum up, as far as I know there has been no successful evolutionary argument along these lines.

Borrowed Fitness

A different evolutionary argument for human menopause is based on the claim that there is indeed biological selection at post-reproductive ages. Such an argument, at its core, uses the fact that biological fitness depends on context: when generations overlap the context includes, e.g., life cycle relationships between ages, mating pattern, or group influences on individuals. But how does this happen, given that there is no post-menopausal female reproduction? To make the argument work, all we need is for post-reproductive individuals to "borrow fitness" from younger reproductive-age individuals. How does this work?

The key idea is simply explained. Take any age (call it x) before menopause with female reproduction (fertility) $m(x)$, one-period survival $p(x)$, and cumulative survival to that age of $l(x)$. Then consider a post-reproductive age (call this y): there is no fertility at that age, just a probability $h(y)$ that a female individual lives from menopause to some post-reproductive age y. I say that the post-reproductive age y "borrows fitness" from the reproductive age x whenever $m(x) = m(x, h(y))$ and/or $p(x) = p(x, h(y))$ with m and/or p increasing as h increases.

In words, these assumptions mean that the pre-menopausal age x benefits from the presence of post-reproductives who survive to age y (there are several ways this can happen, see below). Say that we fix ages x and y, and consider the effect of changes in the post-menopausal survival h (defined above). Taking the fitness here to be the long-run growth rate r we have

$$(\partial r/\partial h) = (\partial r/\partial m)(\partial m/\partial h) + (\partial r/\partial p)(\partial p/\partial h) > 0$$

On the right above, the first factor in each term is positive (because selection at any reproductive age x always acts to maintain m and p), while the second factor in each term is positive by the "borrowing" assumption. (One of these second factors may be zero, but at least one is

positive). So the immediate consequence of "borrowing" is selection against reduction in the post-menopausal survival, h.

Note that "borrowing" can be viewed as an example of "positive pleiotropy", in that increasing old age survival acts to increase reproduction and/or survival at younger ages. This is a sharp contrast to the "negative pleiotropy" proposed by Williams (1957).

Mechanisms for Borrowing Fitness

There are at least four hypothesized mechanisms for "borrowing" fitness; all use long-run population growth rate as a fitness measure (for this and other relevant measures of fitness see Charlesworth 1994).

One is the "grandmother" hypothesis (Hawkes et al. 1998) that posits a positive effect of grandmothers (who are around only because of post-menopausal female survival) on the fertility of young females and/or the survival of infants. This hypothesis is supported by many field observations: for example, in many primates, and other social animals (many whales, elephants) it is well documented that older females help younger females with their offspring. While this is an interesting idea and evidence, how do we turn "grandmothering" into an evolutionary argument: specifically, what do we take as an initial life cycle, are the evolutionary dynamics driven by some trade-off, is menopause an endpoint, what is the effect size? Rogers (1993) made progress on the evolutionary questions using population genetic theory and a trade-off to examine one form of the "grandmother" hypothesis. He analysed a situation in which older individuals "give up" some of their fertility in order to increase survival and/or fertility at younger ages. He used growth rate as a fitness measure, and did a "local" analysis that identifies conditions under which evolutionary change would favour reproductive decline with age but not at the cost of survival. Rogers also attempted a test using comparative data on modern humans but the results were not conclusive; however his analysis was an important step. Considerable progress on the analysis of this kind of "borrowing" has also recently been made by Pavard and Branger (2012).

A second and more comprehensive mechanism is "transfers" as described by Lee (2003). Transfers occur from post-reproductive individuals to individuals at reproductive ages or younger. These transfers can be quite general: of resources such as property or money, knowledge, environments such as dwelling sites or hunting/foraging areas, and so on. Transfers increase the survival and/or fertility of the recipients and thus favour post-reproductive survival. With transfers and parental investment in offspring after birth, Lee shows that the force of selection against a mutation that raises mortality at any age is a weighted average of the Hamilton effect and a new intergenerational transfer effect: this argument explains both post-reproductive survival and declining juvenile mortality. Lee (2008) made real progress towards a dynamic evolutionary theory using simulations of single-sex populations, with recurring deleterious mutations, and incorporates sharing with kin in agricultural and hunter-gatherer societies (e.g., the data and reviews in Gurven 2004, Kaplan and Gurven 2005). His initial state is a life history with a wall of death at 80, fertility similar to that of known hunter-gatherers, and a time interval of 75,000 years. As predicted by his theory, the simulations lead to equilibrium life cycles with high infant mortality and increased post-reproductive survival. Lee's approach is the most general that uses one sex, and can be formulated to include the "grandmother" hypothesis.

A third mechanism for borrowing fitness is the "old fathers" hypothesis (Tuljapurkar et al. 2007) that posits a nonzero fitness for old males because some of them mate with pre-menopausal females, which means that there should be selective value in post-menopausal survival (directly on males and indirectly on females who share autosomal genes with males). Tuljapurkar et al. conduct a "local" analysis similar to Hamilton's, using a two-sex analysis to show that there is positive selection for post-menopausal survival, and also present data that show their hypothesis is supported by anthropological data on the fertility patterns and mating behaviour of humans in the past and even in contemporary society. If Lee's (2003) one-sex analysis is extended to allow for 2 sexes with diverse mating patterns, the "old fathers" hypothesis would fit right in.

A fourth mechanism, also conceptually related to Lee's (2003) analysis, is the work by Robson and Kaplan (2003) that posits an extended life span as a mechanism for developing and transferring information across generations. The latter theory is explicitly an optimality argument, and so does not answer the evolutionary questions I raise above. But the ideas are interesting, and supported by data on hunting ability in some societies (Gurven et al. 2006). This work is perhaps best seen as fitting into Lee's (2003, 2008) framework of transfers.

Things to Be Done

The discussion here lays out general requirements for evolutionary theories of the human lifecycle. I have discussed the kinds of questions that such theories can answer, the nature of the time scales for the action of evolution, and the importance of initial states. Even with these criteria, evolutionary theories are contingent, assuming initial states that lack only one or two salient characteristics of modern human life cycles.

I discussed human female menopause as an example, and argue that significant progress has been made in the synthetic work by Lee (2003, 2008). But the discussion should also make clear that much remains to be done. A two-sex extension is important and should be useful in analysing documented human marriage patterns. Using such an extension, several questions need study: (a) does the evolutionary equilibrium found by Lee (2008) persist; (b) how does the equilibrium depend on the initial state, and on the strength of selection; (c) what happens if we consider separately density-independent and density-dependent dynamics; (d) how can we apply a suitable version of Wachter et al.'s (2013) methods to study how the equilibrium states depend on mutational pattern?

None of these advances or theories explain why the human life history has been so environmentally plastic, or whether environmental response depends on age or developmental trajectory. There may be lessons to be found in adapting the stage-dependent approaches that work so well in plants (Horvitz and Tuljapurkar 2008), and progress in that direction may show whether evolutionary arguments are useful in understanding the past and future of another stage, that of disability (Fried et al. 2004).

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SECTION 7: EVOLUTIONARY DEMOGRAPHY OF FAMILY STRUCTURES, HOUSEHOLDS AND CULTURAL TRANSMISSION

Values and beliefs passed socially among people, within and across generations, greatly influence demographic patterns and the evolutionary processes associated with them. In this section, we combine four chapters on the structure and stability of the family unit with a paper that outlines a theory of culture for evolutionary demography. We have joined family and culture together here because households and family structures are vehicles for the transmission and replication of cultural norms and values (among many other things, of course). The family is where much of the learning of social rules and norms occurs. Families and culture also fit together in light of recent evolutionary studies of human behaviour that illuminates ways in which non-genetic but nonetheless heritable processes affect behaviours and demographic patterns. Demographers are very familiar with topics on this theme, from studies on family and household effects, or on how fertility decisions are affected by social norms. Such processes are especially important today because of increased awareness of the fact that the genetic system is not the only relevant system of inheritance that one must understand if one aims to achieve a "full picture" of the hows and whys of demographic patterns.

In the first chapter of this section, Borgerhoff Mulder provides us with theoretical insight into the formation and stability of pair bonds, or the social unions among couples that are often a key component of households and family structures. She does this via an overview and critical examination of Bateman's Principle, a concept with deep roots in evolutionary biology, but that is not widely known in demography. Bateman's Principle is based on an observation that there is often more variation among males than females for fertility. Patrick Bateman, the biologist after whom the principle is named, reasoned that these differences in variation were due to underlying differences in the costs of reproduction, and that these in turn would have other consequences for sexual behaviour. For instance, males would be less discerning and more eager in the pursuit of sexual partners and females would be more crucial for offspring survival. Bateman's Principle has been explored and debated; a debate reflected here in this book. Borgerhoff Mulder, also a founder of human behavioural ecology and well known for decades of influential fieldwork, updates the theoretical understanding and associated methodological approach to improve the usefulness of Bateman's principle for human demography. This effort yields insight into factors affecting the stability and maintenance of family units in general. Borgerhoff Mulder uses her critique of Bateman's idea to demonstrate what an unusual species humans are, in showing such great variability in customs for mating, marriage and sex roles. This variability is undoubtedly driven by the wide ecological conditions to which our species

is adapted and by our capacity to transmit traits culturally. By digging into Bateman's principle more thoroughly, Borgerhoff Mulder leaves us with a more agile tool (grounded in modern sexual selection literature) for analysing the role that sexual selection might play in structuring the variability in human mating systems.

In the second chapter, Rotkirch, a sociologist who has enthusiastically adopted an evolutionary perspective in her work, gives us an excellent overview of the evolutionary demography of marriage, beginning with the rather pointed truism (at least in the evolutionary human sciences): "We know quite a bit about why and how people have sex, much less about why and how they have spouses", which sets the stage for an informed and fascinating review that brings readers new to the topic up to speed as well as providing fresh ideas for expert researchers in human marriage. Rotkirch analyses data to highlight issues affecting long-term unions, empirically focusing on high-income societies. This chapter is a nice example of how interdisciplinary work can focus attention on new questions; a shift in focus away from who young people find attractive to how and why long-term relationships are maintained is long overdue in the evolutionary literature, especially given the amount of the life-course devoted to retaining, as opposed to finding, a partner.

In the next chapter, Jennings, an anthropologist who works on topics overlapping several fields, gives a helpful overview of research on households, drawing particularly on historical demography, and including issues of definition and measurement. From this, we learn how families and households have changed over time and how their structures affect evolutionary patterns. Importantly, Jennings shows that many of the life history decisions we conceive of as being individual-level decisions are in fact mediated and negotiated at the family level. The argument that larger or nested structures are critical for understanding human demographic patterns is rapidly growing in human evolutionary demography, which is another area of common interest with classical demographic approaches. This chapter is another that highlights areas where evolutionists could learn a lot from more traditional demographic approaches, such as how families and households evolved and how their structures affect evolutionary patterns. She raises, for example, the question of power structures within the household, which has been a significant focus in historical demography, but not so much in evolutionary approaches. Here she notes the contradiction between historical demographic approaches, which find evidence that young women are the least powerful household members, and evolutionary approaches, at least some of which effectively assume young women have considerable power, because of their high reproductive value. Work combining different perspectives that conflict with one another may be particularly fruitful in helping us make progress in understanding human demographic behaviour.

Combining empirical tools while extending the theoretical motivation to understand the formation and stability of families and households, the next chapter by Willführ et al. tackles, in expert fashion, challenging questions of quantification and the statistical modelling of households and family units. The answer is, in short, to use well-constructed fixed-effects models, for which the authors provide great guidance and a thorough overview focused on family reconstitution databases (note: this chapter also complements that by Dillon et al.). The recommendations offered by Willführ et al. will be of great interest to historical demographers, but also to anyone interested in mixed-effects models, which is just about any researcher

studying nested data (extremely common across social sciences, and in both anthropology and demography in particular).

In the last chapter in this section, Colleran gives a thorough treatment of what a theory of cultural evolution for evolutionary demography should look like. Colleran presents a novel and complete theoretical rationale that builds on inspiration from a classic demographic paper by Hammel and is augmented with a combination of human behavioural ecology and cultural evolution. Colleran offers expert guidance on how to transform our thinking about the impact of culture in ways that have theoretical and methodological implications. A subtlety of the chapter is the advice provided on how to operationalize and study the very concept of "culture", which is too often reduced to a categorical variable in a regression in many areas of social science; effectively, this is a call for both demographers and evolutionary researchers to include more anthropology in their work and to realize how integral cultural processes are to all human behaviors. Any social scientist interested in the measurement, definition or causal relevance of culture in demographic behaviour will find this interesting.

22. A Theory of Culture for Evolutionary Demography

Heidi Colleran

Evolutionary demography is a community of researchers in a range of different disciplines who agree that "nothing in evolution makes sense except in the light of demography" (Carey and Vaupel 2005). My focus here is a subset of this research (henceforth "evolutionary demography" or "evolutionary anthropology") that originated in anthropology in the late 1970s and which typically examines micro-level phenomena concerning reproductive decision-making and the evolutionary processes generating observed patterns in reproductive variation. Scholars in this area tend to be more involved in long-term anthropological fieldwork than any other area of the evolutionary sciences. But card-carrying anthropologists are declining among their number as researchers increasingly come from other backgrounds in the biological and social sciences, with an associated decline in the contribution of ethnographic work. Most practitioners identify with the sub-field of human behavioural ecology — the application of sociobiological principles to human behaviour — and distinguish themselves from the sister fields of evolutionary psychology and cultural evolution. Human behavioural ecology has been criticized for abstracting away the details of both culture and psychology in its focus on adaptive explanations of reproductive behaviour, and for its commitment to ultimate over proximate causation. This chapter explores these critiques. Inspired by E. A. Hammel's seminal paper "A theory of culture for demography" (Hammel 1990), I examine how the culture concept is used in evolutionary research. Like Hammel, I argue that a theory of culture for evolutionary demography requires engaging more seriously with (and in) ethnographic work. I highlight some challenging examples to motivate discussion about adaptive reproduction and natural fertility. Going further, I advocate for cultural evolution as an integrative framework for bringing both culture and psychology into the core of evolutionary demography research. This will involve expanding our theoretical and conceptual toolkits: (1) building and testing proximate mechanistic models, (2) delineating and evaluating causal claims at multiple levels of analysis and time scales, and (3) exploring co-evolution or feedback between demography and culture.

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Why Has Culture Not Been a Central Concern in Evolutionary Anthropology?

Following the publication of E. O. Wilson's *Sociobiology* (Wilson 1975), and the "wars" that ensued (Segerstråle 2000), three streams of research on the evolution of human behaviour emerged: evolutionary psychology, human behavioural ecology and cultural evolution (Laland and Brown 2002). Of these, human behavioural ecology emerged directly out of socio-cultural anthropology (Borgerhoff Mulder and Schacht 2012). Working mainly at the micro-level these researchers were interested in the demography of underrepresented small-scale populations living in marginal environments (Howell 2000; Blurton Jones 1986; Borgerhoff Mulder 2000; Mace 1996, 1993; Chagnon 1979; Kaplan 1996; Hurtado and Hill 1996; Marlowe 2010; see Sear and others 2016b; Kennett and Winterhalder 2006; Kramer and Boone 2002). A principal concern was how the social and physical aspects of life in different ecologies affect the ability of individuals and their genetic lineages to maximize reproductive success (i.e. contribute genetic material to future generations). Key to the approach was the idea that we could learn about the evolutionary history of our species by studying populations living in conditions and pursuing life-ways that may closely approximate those of our ancestors.

Today, many evolutionary anthropologists work more often with comparative and large-scale databases than with primary anthropological data, and many identify as evolutionary demographers, but their concerns about adaptive reproductive behaviour are largely the same (Nettle and others 2013; Sear and others 2016a; Mattison and Sear 2016). In a globalizing world, practitioners now routinely explore reproductive behaviour beyond small-scale societies, and questions and methods are shifting. Statistical and formal models are becoming more sophisticated. Cross-cultural and macro-level studies are becoming relatively common (see for example Borgerhoff Mulder and others 2009, 2019), detailed case studies more rare. Applied and policy-oriented research is gathering steam (Gibson and Lawson 2014; Tucker 2007). Much work has crystallized around explaining global fertility decline, in response to Vining's clarion call that it is the "*central theoretical problem of human sociobiology*" (Vining 1986). Increasingly there are calls for more emphasis on "modernizing" or "modern" populations, meaning societies that have experienced the demographic and epidemiological transitions and which now exhibit low fertility and mortality rates (Mattison and Sear 2016 though see; Borgerhoff Mulder 2013 for an alternative view).

Many of our socio-cultural anthropology colleagues would deny that evolutionary demography so practiced even resembles anthropology (Ingold 2007). First, the field originated out of and remains based on applications of animal and economic models to reproductive behaviour (Cronk 1991) and has little time for non-evolutionary approaches within anthropology. Second, it involves a strong individualism, both methodological, in the sense that explanatory models take individuals as the unit of analysis, and ontological, in the sense that higher-level social phenomena are taken to be aggregations of individual level properties (they are typically not viewed as "social facts" in and of themselves). Third, the field maintains a fairly sharp distinction between proximate and ultimate explanations (Nettle and others 2013; though see Borgerhoff Mulder 2013) that foregrounds the fitness benefits of behaviour and brackets out (suspends judgment about) cultural processes. Many see this as downgrading cultural life — the

very object of much anthropological enquiry — to a secondary or less causally interesting role, and it has made for an uneasy relationship with socio-cultural anthropologists, who do not recognize themselves in these commitments (Sahlins 1976; Ingold 2007; reviewed in Colleran and Mace 2011).

The reasons for the lack of culture, so to speak, are partly sociological: establishing human behavioural ecology as a respectable quantitative field of enquiry meant side-lining or subsuming culture for much of its early development (see Laland and Brown 2002 for review). Face-offs with evolutionary psychologists — who argued that contemporary human behaviour is adapted to Pleistocene conditions, and therefore cannot be considered adaptive today — meant that practitioners were initially concerned with defending the study of adaptive behaviour at all (Smith and others 2001; for recent review, see Stulp and others 2016). In contrast to evolutionary psychology's typical focus on universal characteristics of human psychology, behavioural ecologists showed that demographically relevant behaviour varies: between individuals and populations, across subsistence economies and over time, in adaptive ways related to fundamental energetic and other trade-offs (Kaplan 1994; Turke 1989; Hurtado and Hill 1996; Lawson and Borgerhoff Mulder 2016; Mace 2000; Cronk and others 2000).

But there are also profound theoretical reasons. The field draws on and develops life-history theory (Stearns 1989; Kaplan 1994; Charnov 1993), evolutionary game theory, and other theoretical approaches developed beyond anthropology, and a rational-actor approach to human decision-making — similar but not equivalent to that in economics and demography (Robinson 1997; Becker and Lewis 1973) — has become a central explanatory device. As in other fields, rational choice does not necessarily imply conscious reproductive strategizing, only that the pursuit of *proximate* aims such as status striving, wealth accumulation, social desirability, or any number of other cultural features, correlates with reproductive success, which is assumed to have been true for most of human history. A second claim in this field is that individuals come pre-loaded with *reaction norms* that evolved over long evolutionary time-scales: these refer to the genetically encoded range of responses an individual can express in a set of environmental conditions. This range, in theory, enables behaviour to remain consistent with fitness maximization (though see Baldini 2015).

The combination of a black-boxing of reproductive decision-making (henceforth the "rational-reproducer" model) and a kind of ecological relativism (meaning that you cannot fully understand particular reproductive outcomes outside of the particular ecological conditions they occur in) has been extremely successful (Nettle and others 2013; Sear 2015b). But this orientation leaves two deep questions about human reproduction unexplored. How does culture actually contribute to demographic outcomes? And what does the psychology of reproduction look like? Evolutionary anthropologists have been asking themselves these questions for a while (Borgerhoff Mulder 2013; Mace 2014; Roth 2004), but a clear way forward has not been articulated (see Colleran 2016 for a recent attempt).

Culture in Demography: The Emergence of Anthropological Demography

Demography and anthropology go back a long way and many foundational anthropologists were acute observers of demographic patterns. But demographers and socio-cultural

anthropologists have come to distrust each other's methods and insights over time (Scheper-Hughes 1997; Randall and Koppenhaver 2004; see Roth 2004; Colleran and Mace 2011 for comparison with evolutionary anthropology). Socio-cultural anthropologists have long critiqued the limited role given to culture in demographic research, and the lack of qualitative and interpretive analysis to draw out the dimensions of social life that are not measurable using quantitative survey instruments and population level analysis (Price and Hawkins 2007; Greenhalgh 1990; Hammel 1990; Hammel and Howell 1987; Cleland and Wilson 1987; Pollak and Watkins 1993; Behrman and others 2002; Hirschman 1994; Fricke 1990; Randall and Koppenhaver 2004; Scheper-Hughes 1997). A seminal paper in this literature is Hammel's "A theory of culture for demography" (Hammel 1990), the title of which I adapt only slightly for the current chapter. Hammel (1990) described the use of "culture" as "mired in structural-functional concepts that are about 40 years old, hardening rapidly, and showing every sign of fossilization" (p. 456). He argued that the study of demographic behaviour has actually been hampered by the widespread use of "culture" in different inappropriate guises and advocated a much greater use of fine-grained studies and ethnography. He also emphasized the importance of feedback over both the short- and long-term: culture shapes behaviour, actors redefine culture, behaviour shapes cultural change.

These concerns led to the development of the sub-field of anthropological demography (Kertzer 2005; Bernardi and Hutter 2007; Basu and others 1998; Fricke 1997), which examines the complexities of demographically relevant behaviour and the attitudes, perceptions, concerns and anxieties associated with it. Anthropological demographers call into question the methods and classifications used to define culture and other foundational analytical constructs in demographic data collection; they reject the decontextualized rational-actor model inherent in most demographic research, and they critique the causal assumptions (and lack of feedback) in demographic models. The field now draws widely on research that is often only tangentially connected to demography: anthropological studies of menstruation, pregnancy and childbirth, contraceptive choice, access and use, new reproductive technologies, infertility, HIV and the spread of STIs, migration, mortality, development, and many other topics besides. Their focus is on how society, politics and culture shape the biological experiences of birth, death and migration (e.g. Kreager 2017; Pooley and Qureshi 2016; Kanaaneh 2002; Johnson-Hanks 2007; Bharadwaj 2016; Caldwell and Caldwell 1987; Scheper-Hughes 1993).

Anthropological demographers have directly engaged in critiques of demographic practice, and their methodological influence has been substantial. Anthropological methods such as focus groups or open-ended interviews are now regularly used in "mixed-methods" studies and to design better quantitative data collection instruments (Randall and Koppenhaver 2004; Basu and others 1998). Basic analytical categories such as "household", "traditional versus modern", "reproductive decisions" and concepts such as "ideal family size", "natural fertility" and "insurance effects", all central to large-scale demographic data collection and analysis, have been given more nuanced treatment, even revised, following the critical interventions of anthropological demographers (Randall and others 2011; LeGrand and others 2003; Bledsoe and others 1994; Randall and LeGrand 2003; Johnson-Hanks 2002; Johnson-Hanks 2005; Olaleye 1993; Randall and Coast 2015). Others have shown that the

way demographic data is collected affects the data that gets produced in many ways, from underrepresenting vulnerable populations through survey definitions (Randall and Coast 2015) to misinterpreting what cannot be talked about for lack of knowledge or interest (Randall and Koppenhaver 2004). Despite this impressive impact for such a small field of enquiry, the theoretical contributions of anthropological demographers still remain on the fringes of mainstream demography (Bernardi and Hutter 2007; Johnson-Hanks 2007). Many socio-cultural anthropologists have given up on collaboration with quantitative researchers at all (Scheper-Hughes 1997). While evolutionary anthropologists regularly cite the work of anthropological demographers, sadly, the reverse is not true: they have long viewed the evolutionary work as "thoroughly teleological" (Hammel and Friou 1997 cited in; Roth 2004).

Cultural Evolution as a Unifying Framework

The prospects for integrating both culture and psychology into evolutionary demography are more promising now than they ever were. The main reason is that the field of cultural evolution (or dual inheritance theory) — the third of the research streams that emerged after the sociobiology wars — has developed into a multidisciplinary field that quantitatively studies both the transmission of culture and the population level dynamics of norm psychology (Cavalli-Sforza and Feldman 1981; Boyd and Richerson 1985). Cultural evolution has ushered in a major change in our orientation to human evolution: our evolved psychological capacities themselves arose out of a long history of learning from and living with others and these social learning skills — in addition to the cost-benefit calculations of human behavioural ecology — have allowed interacting groups of people to produce cultural innovations that may have radically altered aspects of our physiology, anatomy, and psychology in crucial ways (Henrich 2016). While these cultural transmission mechanisms (or learning biases) evolved to help individuals acquire adaptive behaviour, allowing rapid calibration to the environment, the two inheritance channels (culture and genetics) can become decoupled, or even generate conflicting pressures (Boyd and Richerson 1985; Cavalli-Sforza and Feldman 1981).

The crucial innovation by these researchers was to formalize a set of quantitative tools for analysing and modelling the dynamics of culture. Contemporary cultural evolutionary research is expansive, incorporating processes of cultural selection, mutation/innovation, drift and migration (Mesoudi 2011), niche construction (Odling-Smee and others 2003) — whereby individuals modify the environments they live in, affecting the selection pressures they are subject to, and thereby creating feedback in the evolutionary process — and other non-genetic inheritance channels (Jablonka and Lamb 2005), cognitive and symbolic evolution (Sperber 1996) and cyclical processes of change (Turchin and Nefedov 2009). The field draws on population-genetic and epidemiological diffusion models (Cavalli-Sforza and Feldman 1981; Boyd and Richerson 1985) to examine change in the frequencies of cultural traits over time; models and experiments of social learning and cognition to understand how individual characteristics give rise to population level distributions of cultural traits (Henrich and Boyd 1998; Henrich and others 2005); and macro-evolutionary and phylogenetic studies of societies and languages (Mace and Holden 2005; Jordan and others 2009; Mace and Pagel 1994) to make explicit the path-dependent histories of culture

as well as identifying sources of shared ancestry. Cultural evolution is now a thriving multidisciplinary arena for experimental, observational and quantitative work at multiple levels of analysis.

Cultural evolutionary theory and human behavioural ecology have led a parallel existence for most of their development because of a range of different starting assumptions and overlapping conceptual categories (Borgerhoff Mulder 1998; Colleran 2016). Cultural evolutionists have also had their disagreements with evolutionary psychologists, who have tended to consider that culture is "evoked" by fitness-relevant environmental experiences (Barkow and others 1992), and not "transmitted" through learning and interaction and therefore separable from fitness constraints. Increasingly though, ideas from cultural evolution are percolating into evolutionary demography (for review see Colleran 2016). The combination of genetics-style "population thinking" with social psychology-inspired behavioural models in cultural evolutionary theory broadens the methodological and theoretical landscape for evolutionary demography beyond optimality and rational-reproducer models. As an overarching framework, cultural evolution holds great promise for bridging some of the gaps between what socio-cultural anthropologists and evolutionists care about, namely, a focus on the socially constructed nature of human cultural systems on the one hand, and a commitment to using model-based, quantitative methods to develop evolutionary theory on the other. It does this through providing quantifiable connections between individual decision-making, observation and learning, information flow in (structured) populations and group level cooperation and competition.

But this expanded set of theoretical tools comes with a need to relax the often sharp distinction made by behavioural ecologists between proximate and ultimate (i.e. functional) explanations (Nettle and others 2013; see, for example Borgerhoff Mulder 2013; Laland and others 2011), as well as a much more thorough incorporation of individual differences in cultural evolutionary theory. It also comes with a need for much more ethnography, and greater rapprochement with socio-cultural approaches to demography. The fields of evolutionary demography, cultural evolution and anthropological demography have largely ignored each other over the years, but have much to gain from greater communication. In the rest of this chapter, I discuss the gaps and overlaps between them. This involves discussion of assumptions related to the culture concept, reproductive decision-making, natural fertility, maladaptive reproductive behaviour, and proximate/ultimate causality.

The Culture Concept in Evolutionary Demography and Cultural Evolution

Hammel (Hammel 1990) described two major tendencies in demographic theorizing about reproduction: "sociological" approaches that tend to underplay individual agency through homogenizing culture-concepts, and "economistic" approaches which tend to universalize individual rationality, giving complete agency to individuals. This distinction captures well the stereotypical critiques made against cultural evolution and human behavioural ecology, respectively, but in reality both fields make use of these tendencies in different ways and so cannot be summarized under this simple typology.

An example of a sociological use of culture is the *culture-as-identifier* approach to describing social phenomena (e.g. the French fertility decline, high-fertility among Irish

Catholics). Cross-cultural evolutionary research makes frequent use of such high level population descriptors such as subsistence type, religious or ethno-linguistic groupings (Sellen and Mace 1997; Bentley and others 1993; Mace and others 2005; Henrich and others 2006) which can act as a stand-in for "culture". In this formulation, all that culture does is effectively label a particular demographic pattern in time and space. While it does often identify something about a particular collectivity that we seem to readily imagine as "cultural", the designation is merely descriptive and does not advance any theoretical claim about why that cultural group behaves in the way it does. Culture here is doing descriptive or classificatory work, not explanatory work. Some human behavioural ecologists have directly criticized this approach in cultural evolution by highlighting that behaviour can be just as varied within a particular collectivity as it is between them (Lamba and Mace 2011). However, much the same critique could be levelled at other common descriptors used frequently in demographic research, such as "educated", which indicates, through membership of a particular category, a person's likely reproductive behaviour (i.e. that they may have lower fertility than their less-educated peers), but says nothing substantive about why their behaviour is different. This formulation does not involve a theory of social action, or any theoretical claims about the transmission, acquisition or negotiation of either culture or demographic behaviour.

Comparisons can of course be finer grained, such as those in multi-community settings within the same ethno-linguistic groups (Colleran and others 2015, 2014; Alvergne and Lummaa 2014). This approach appeals to *culture-as-context*, a form of natural experiment where the effects of some cultural features can be to some extent isolated from the broader cultural milieu. Contextual approaches are becoming widespread in demography, and are increasingly used to disentangle the levels of aggregation at which a purported variable has causal power (Kravdal 2002, 2012; Stephenson and others 2008). This represents a more nuanced treatment of the culture-as-identifier approach, especially where there is explicit measurement of proxy variables at different levels of social organization. However, when used without any mechanistic links between different levels of analysis, such as social network connections, this approach suffers from similar limitations to the approach above.

Other lines of research compare reproductive outcomes among communities with similar economic, cultural or ecological backgrounds but which nonetheless have distinctive institutional or socio-cultural features such as inheritance or marriage systems (Leonetti and others 2007; Gibson and Sear 2010; Gibson and Gurmu 2011; Holden and Mace 2003; Mace and others 2003). This characterizes culture as the playing out of a set of largely autonomous rules or institutions, such as kinship or social stratification. This is *culture-as-structure*, and implies that social action responds to pre-set and exogenous structures of organization. Under this model demographic change is simply the outcome of individual responses to infrastructural or institutional change, but we do not learn why or how those structures themselves evolve. Instead, structures, rules and institutions are being continuously rebelled against, reinterpreted, reformed and updated through individual and collective actions. In the jargon of evolutionary theory, institutions and social structures coevolve with changes in population, ecology and economics.

A fourth approach is *culture-as-ideology*. Take, for example, the ways that reproduction is moralized in every population. People are often aware of the "right" levels and rates of

reproduction in their particular environment, the right parenting strategies. Individuals can easily point to others who have had too many or too few children according to their social or cultural context. We see these unspoken rules everywhere. One way to think about this is to consider reproduction as a form of collective good (Kohler 2000), since the resources of the local environment are needed to jointly produce the children of multiple co-resident families, lineages and others. Indeed the "cooperative breeding" model of human evolution does precisely this (Hrdy 2007), and a relatively large literature now shows that conflict and cooperation with kin is an important factor in women's reproductive outcomes (Sear and Mace 2008). As part of this, an evolved ability to internalize the social norms of a particular group is likely to facilitate this kind of cooperation (Gavrilets and Richerson 2017). Coordination on locally adaptive social norms that regulate appropriate reproduction can be achieved via the sanctioning of norm violators (Fehr and Gächter 2002). Ideologies of reproduction could be shaped by kin interactions and interests that themselves structure the costs and benefits of particular reproductive actions — what Leonetti has called "kinship ecologies" (Leonetti 2008). These can themselves vary according to structural-cultural features — lineality (Pollet and others 2009) or inheritance system (Gibson and Sear 2010) — but they are also affected by macro-level cultural and economic changes that alter social network structures, which can determine how prominent or influential kin are in the reproductive lives of women (Newson 2009; Newson and Postmes 2005; Colleran 2020).

The strong emphasis on individual costs and benefits to reproduction in human behavioural ecology means that the culture concept is not well-developed beyond the idea that it forms part of the "socio-ecology" (Cronk 1995). This phrasing is a nod to the fact that culture is important in the determination of reproductive outcomes, but it is thought to be just one element among the set of "proximate" determinants of demographically relevant behaviour that are often, though not always, a secondary concern (Nettle and others 2013). This is a form of economistic approach that gives a large amount of agency to individuals to figure out the best reproductive strategy under a given set of circumstances (the rational-reproducer). In fact, for many evolutionary demographers, the success of a cultural trait itself will often be associated with its ability to confer fitness benefits on individuals, for example a marriage rule that delays marriage for men, which, by separating reproductive generations of women, adaptively reduces reproductive competition between them (Alvergne and Mace 2012). This is *culture-as-adaptation*: cultural traits are themselves adaptations that help populations optimize their reproductive success. Both human behavioural ecology and cultural evolution make use of this kind of conceptualization.

Adaptive Culture?

However, the causal claim here is a strong one: it says that because culture itself evolved to help us acquire adaptive behaviour, the genetic program is ultimately in charge. If a cultural mechanism is maladaptive (reducing fitness over time), then natural selection should logically weed it out of the broader population, since those individuals and groups that practice it will eventually be out-reproduced by those that do not. This idea that cultural adaptations have primarily functional benefits remains closer to classic ecological

and materialist anthropology and archaeology (Harris 2001) than to contemporary socio-cultural streams in anthropology, and is exemplified in its extreme form by E.O. Wilsons claim that:

The genes hold *culture* on a *leash*. The *leash* is very long, but inevitably values will be constrained in accordance with their effects in the human gene pool (Wilson 1975).

Human behavioural ecologists have traditionally subscribed to this interpretation, often implicitly if not always explicitly. Indeed the concept of "adaptive lag" — the idea that when humans adapt slowly to changing environments there will be a period of suboptimal behaviour — and which is often appealed to regarding fertility decline, is a logical conclusion of this view of culture (Laland and Brown 2006). Cultural evolutionary research differs on this point, in three ways.

First, for cultural evolutionists the success or fitness of a particular cultural trait is not as strongly tied to assumptions about fitness maximization, and is instead inferred from frequency changes of a trait in the population over time, assuming certain learning-rules or structures. Much cultural evolutionary theory is not strongly committed to strictly Darwinian or selectionist approaches (Lewens 2015). This means that other non-adaptive processes can drive the spread of a particular cultural trait in a population. Crucially, it allows for the spread of explicitly genetically maladaptive traits. This logic is the basis for most cultural evolutionary work on fertility decline (Colleran 2016). This more permissive version of cultural evolution is one that Lewens (Lewens 2015) has described as "kinetic" (broadly, non-selectionist) and which is often broad enough to encompass many different kinds of change over time. An advantage of this is that it can potentially connect with more socio-cultural approaches to demographic change that do not focus on adaptive functions of behaviour.

Second, the fact that humans are continually interacting with, modifying and sometimes constructing their socio-ecological environments means that evolutionary pressures themselves are also constantly evolving (Laland and Brown 2006; Odling-Smee and others 2003). This appreciation of the centrality of feedback in the evolutionary process is a hallmark of the "niche construction" perspective. A niche construction approach, within the broader framework of cultural evolution, has implications for how we expect individuals and communities to adapt to and change their socio-ecologies on short to medium time scales, thus removing the need to appeal to adaptive lags and temporarily suboptimal behaviour (Laland and Brown 2006). Again, this orientation to the evolutionary process makes connections with socio-cultural anthropology, by allowing the participatory character of cultural life to be more explicitly framed in evolutionary terms.

Third, some cultural evolutionary models assess the fitness of a cultural trait in terms of its function at the group (or institutional) level rather than at the individual level. Because culture comes in packages of institutions, norms, beliefs and practices, some elements of which can be adaptive, they can have many different effects: reducing interaction costs in social networks or brokering cooperation, generating regularity in behaviour though institutions, norms and sanctioning, or entrenching power-relations and divisions of labour. A cultural trait that causes some groups or institutions to spread at the expense of others, via population growth, expansion, migration or other means of cultural prestige or soft power can in principle spread by between-group cultural selection (Richerson and others 2014).

In theory individually costly behaviours can spread in a meta-population if the aggregate outcome is beneficial to the group (Boyd and Richerson 2002). Again, fertility decline is a good example where this logic could be applied. Historical fertility declines during the Industrial Revolution are thought to have been generated through feedback between population density and technological innovation, which spurred economic growth (Galor 2011). The interconnectedness of contemporary nation states through labour and migration transfers, innovation and capital, has increased the levels of interdependence between groups in international trade and supply networks to an unprecedented level. Technologically advanced countries appear to be able to effectively down-regulate each other's fertility rates through competition and cooperation for increased economic productivity (Dang and Bauch 2010). Fertility reductions can drive temporary rises in the rates of economic growth by altering age structures and the amount of people available to take part in wage labour, a phenomenon known as a "demographic dividend" (Bloom and others 2003). When considered in a multilevel framework, demographic benefits at one level of aggregation can trade off against costs at another.

Individual Differences

If evolutionary demography tends to focus overly on individual differences, then cultural evolutionary theorizing tends to undervalue them, by rarely including individual resource constraints on reproductive options. Instead, cultural evolutionary models relevant to demographic behaviour have tended to assume that individuals are undifferentiated with respect to their opportunities to access information, can perfectly sample from cultural learning models and are free to enact their reproductive preferences (reviewed in Colleran 2016). Having said that, empirical experimental studies in cultural evolution are much more focused on individual variation and on the selective use of social learning strategies dependent on a wide range of constraints and incentives (Mesoudi and others 2016; Kendal and others 2018), so this difference between theory and empirical strategy is unlikely to last for long.

While cultural evolution is now a broad school of thought incorporating the evolution of socially transmitted information, technologies, norms and institutions, the culture concept most widely used is broadly "ideational" or "informational": *culture-as-information*. This definition conceptually fuses information transmission with behaviour (culture is information capable of affecting behaviour that is transmitted socially (Boyd and Richerson 1985)), and the innovation-diffusion models typical of cultural evolutionary research usually assume a tight relationship between information flow and behavioural expression (Henrich 2001). This formulation is very close to the ideational models prevalent in demography (Cleland and Wilson 1987) which are also often modelled using diffusion dynamics (Casterline 2001; Rogers 2010). Is this tight link between information diffusion and behaviour justified? In theory, the frequency of a cultural trait (say, a belief about the value of having fewer children) within a specific group should not only influence the chances that an individual adopts the trait, but also the chances that it is translated into behaviour (actions consistent with having fewer children), and there is some social psychology evidence suggesting that feedback between individual and group "norms" may have this effect (Smith and Louis 2008). But the process by which transmission of social information is translated

into actual behaviour has not been a focus for cultural evolutionary theory. Indeed there is plenty of evidence that people say one thing and do another, that subjective intentions do not predict behaviour (Armitage and Conner 2001; Ní Bhrolcháin and Beaujouan 2019) and this is an anthropological truism: the distinction between ideal and real culture. Some models in cultural evolution take this partly into account by allowing individuals to vary in their propensities to adopt particular behaviours and/or by allowing behaviours to be probabilistically adopted (Kandler and Steele 2009; Kendal and others 2005). Nonetheless, diffusion dynamics of the type typically examined in cultural evolution are known to be sensitive to individual variation, for example in wealth and income heterogeneity (Kandler and Steele 2009) and population sub-structure (Laland and Kendal 2003). A greater focus on how these effects may influence cultural evolutionary dynamics, as well as empirical tests of these hypotheses are needed.

Cultural evolutionary theory has been at the forefront of modelling how demographic properties such as population size or connectivity crucially affect the accumulation and loss of culture over time (Henrich 2004; Powell and others 2009). There has been much less focus on how culture might affect demography. Many early ecological anthropologists (the researchers most similar to much contemporary evolutionary anthropology), aimed to show that the demographics of small-scale populations were culturally regulated, through ritualized warfare (Rappaport 1984), culturally determined age-structures (Roth 2004), or other forms of cultural equilibrium that maintained a balance between population growth and carrying capacity (Harris 2001). The question of cultural population regulation is an old one in anthropology and there are countless examples in the ethnographic literature of cultural institutions, rules, taboos, rituals and practices affecting reproductive opportunities (Hammel and Friou 1997). This angle has been neglected by both cultural evolution and evolutionary anthropology, but is one which anthropological demographers would have much to say about.

Proximate and Ultimate Causality: A Distinction that Hampers More than it Helps?

Following Tinbergen's delineation of four different "why" questions in evolutionary analysis (proximate, developmental, ontogenetic and ultimate) and Mayr's distinction between proximate and ultimate explanations (Tinbergen 2010; Mayr 1961; though see Laland and others 2011), evolutionary demographers often expect different kinds of explanation to be mutually consistent and enriching (see Colleran and Mace 2011 for an overview). Nonetheless, they do assign distinct causal powers to different kinds of explanation. Proximate mechanisms, for example, do not have the causal power to fundamentally de-rail the ultimate motivations that humans were endowed with over millennia of evolution. Where they do exhibit mismatch, this will be a temporary state of affairs (adaptive lag), and will most likely be corrected over the long term. Evolutionary demographers agree that zooming in on the cultural nuances of a particular behaviour in a particular context will undoubtedly reveal interesting details about the local perception of costs and benefits, as well as the various meanings associated with reproductive activities. Some, but by no means the majority, give ethnographic details in their publications. Still, the majority of practitioners defend the benefit of abstracting away from these details to get at the underlying long-run

evolutionary logic. When faced with the criticism that culture seems undervalued in their research, evolutionary anthropologists often point out that culture is conceptually already in the models: culture is part of the socio-ecology. This conceptual move, to incorporate proximate cultural mechanisms into the very definition of adaptive behaviour, allows the practitioner to avoid having to define culture at all. This makes the socio-ecology a slippery concept to work with; because it is unclear which parts of culture contribute to adaptive reproduction, and which ones do not.

Much the same can be said about the concept of natural fertility. Originating in the 1950s work of the demographer Louis Henry (Henry 1961; later Coale 1971), natural fertility refers to the age-specific pattern of fertility that is assumed to emerge in the absence of deliberate control of the number of children being born. If there is no *parity-specific stopping* in a population (indicating that people stopped having children after a certain desired family size was reached), natural fertility should result in a pattern of regular birth intervals. Both physiological and cultural constraints can generate this baseline pattern: fertility can be naturally limited by anything from nutritional status to breast-feeding practices, from marriage-rules to post-partum sexual taboos. This means that while the level of natural fertility (the number of children born/surviving) can vary dramatically across cultures (Bentley and others 1994), we should still be able to judge a natural fertility population from the age-specific pattern of reproduction. In practice, many researchers do not use age-specific fertility profiles to determine if their study populations are experiencing natural fertility: more often, the absence of significant modern contraceptive use is the proxy.

The distinction between natural and controlled fertility turns less on the difference between physiological versus cultural determinants of reproduction, and more on the idea of conscious or planned behaviour versus unconscious or unplanned behaviour. In the famous words of the demographer Ansley Coale, reproductive decision-making can be more or less part of a "calculus of conscious choice"(Coale 1973). Viewed in this light, a lack of parity-specific stopping coupled with compliance with strong reproductive norms can be taken as evidence of unconscious (and therefore natural) fertility, even where cultural norms end up lowering overall fertility rates. Parity-specific stopping, on the contrary, is almost always thought to be conscious and, implicitly (though this is unclear), to a large extent outside the realm of cultural norms. It is important to note that no human population exhibits maximal biological reproductive output: cultural and other constrains are everywhere in operation (Lawson and Borgerhoff Mulder 2016).

Even with the conceptual de-emphasis on cultural determinants in favour of a form of deliberative decision-making, the natural/controlled distinction is hard to justify in real-world populations (Bledsoe and others 1994; Johnson-Hanks 2002; Caldwell and Caldwell 2003; Bledsoe 1996). There is clear evidence that: (1) regular patterns of birth spacing typical of natural fertility profiles can be generated as much by the deliberate use of modern contraceptives as by a lack of them, in line with locally appropriate spacing norms (Bledsoe and others 1994); (2) women's perceptions of what counts as "modern" contraception are culturally inflected, often leading them to use methods of fertility control that are not typically counted in large-scale surveys (Johnson-Hanks 2002; Colleran and Mace 2015), and (3) the majority of twentieth-century fertility declines are more likely to have been driven by

reproductive strategies that are not dependent on the number of children already born (Daniel J. Hruschka and others 2018).

Natural fertility ultimately raises more questions than it answers, because casting such a wide net over the causal structure of reproductive behaviour does not help to delineate causal theories about that behaviour. To a large extent, the same is true for the socio-ecology. Evolutionary demographers have repeatedly staked out research territory through their focus on ultimate explanation and there can be no doubt that this has been fertile ground (Stulp and others 2016; Nettle and others 2013; Sear 2015a; Colleran and Mace 2011). Nonetheless, it is the motivations and perceptions that tell us not only what is locally interesting about reproductive behaviour, but in many cases, what is important for a causal understanding of it. Simplified models are necessary for an evolutionary understanding of behaviour in the broadest sense, but they are not sufficient to explain why reproduction varies the way it does empirically. Proximate explanations are not just "how" explanations, they are also often "why" explanations (Borgerhoff Mulder 2013).

Do We Have a Comprehensive Theory of Reproductive Decision-making?

There is much talk of "reproductive decision-making" in evolutionary demography. Mostly this amounts to a reductive but extremely widely applicable rational-reproducer model focused on how people integrate over the various costs and benefits of particular reproductive activities to optimize reproductive success. It is not a requirement that such "decisions" are conscious, nor even that they are psychological — they can be "taken by a woman's physiology" (Sear and others 2016a) where, for example, energetic conditions preclude conception. While intended to be integrative in much the same way that socio-ecologies are integrative of biological and cultural mechanisms, it is doubtful whether any practicing evolutionary anthropologist thinks this is a comprehensive theory of reproductive decision-making. Anthropological demographers have strongly criticized this decontextualized approach in demography for not taking into account how culture structures the opportunities for reproduction, since both the extent to which "costs" or "benefits" are interpreted as such, and the actual values they refer to, vary substantially across contexts (Cleland and Wilson 1987; Lesthaeghe 1980; Pollak and Watkins 1993; Sahlins 1976). Socio-cultural anthropology has largely abandoned any attempt to make pan-human psychological claims in favour of understanding culture-specific rationalities. These are the cultural lenses through which all behaviour is interpreted, reflected upon, incentivized and challenged.

Very little evolutionary work has focused on the actual psychology of reproduction, let alone how culture subtly or overtly influences the perception and selection of reproductive choices. To the extent that there is research on the psychological mechanisms underlying fertility decision-making, it is fragmented and based mainly on research with WEIRD populations: Western, educated, industrialised, rich and demographic (McAllister and others 2016; Henrich and others 2010).

In demography, the now-large literature on "ideal fertility" has tried to address some aspects of the gap between planned and unplanned fertility, by focusing on the "unmet need" of women for modern contraception (Casterline and Sinding 2000). This refers to the difference between the number of children women say they want and the number of children

they actually give birth to. Other frameworks such as the theory of planned behaviour have been incorporated into demography as a way to capture the connections between intentions and behaviour, though not without question (Bachrach and Morgan 2011). Again, there is substantial evidence that these formulations may be insufficient: people often do not have clear reproductive goals, are inconsistent or ambivalent when it comes to enacting the preferences they report to researchers, and indeed often construct their ideal family size as they go through their reproductive lives (reviewed in Ní Bhrolcháin and Beaujouan 2019; see also Mason 1992). Opportunistic rather than deliberative reproductive decisions seem common (Randall and LeGrand 2003; Johnson-Hanks 2005). Much more basic research in this area is needed. Cultural evolution can be useful, through its focus on the evolution and dynamics of norm psychology, as can more comprehensive theories of social action, such as the theory of conjunctural action emerging from anthropological demography (Johnson-Hanks and others 2011).

"Maladaptive" Cultures of Reproduction

The explanatory strategy of subsuming proximate mechanisms in order to focus on ultimate outcomes is easy to defend when proximate and ultimate explanations are consistent. It is much harder when they conflict. The most obvious example is the global transition to low fertility, which does not appear to optimize reproductive success (Colleran 2016; Borgerhoff Mulder 1998). Because of its global reach and seemingly law-like patterning, fertility decline is finally drawing the focus of evolutionary demographers to proximate mechanisms (Sear and others 2016b; Colleran 2016). But the ethnographic record offers a cornucopia of examples where the reproductive behaviour of "traditional populations" either does not align with a *prima facie* genetic program of fitness-maximization, or where practices explicitly designed to increase fertility have actually had the opposite effect (for review see Paul 2015).

A striking example involves the ritual practices of *otiv bombari* among the Marind-Anim of western Papua, Indonesia (Irian Jaya), which mandated that upon a woman's marriage or resumption of active village life after childbirth, she participate in sexual intercourse with all the men of her husband's sub-clan (often up to ten or more men) over the course of a single night. These practices were intended to promote fertility among other things, in line with a rich cosmology that required the collection of growth-promoting semen mixed with the vaginal fluid that follows ritualized sexual intercourse (Baal 1966; reviewed in Paul 2015; Knauft 1993).

In fact, the practice was implicated in abnormally high rates of chronic cervical inflammation among women, leading to widespread sterility and substantial depopulation as a result. The logic of fitness-maximization teaches us to expect this practice, or even this population, to eventually be lost through natural selection. But the Marind did not die out due to this "maladaptive" cultural mechanism, and until after the colonial encounter in the 1950s, neither did the practice. Depopulation was largely compensated for by the kidnapping of women and young children during expansionary raiding expeditions associated with head-hunting, and otherwise through adoption. It is estimated that up to 20% of the population was supplied in this way before pacification: once money came into the picture, available data show that up to 25% of children in some communities were purchased in the

post-pacification period (Knauft 1993). These children were raised as full members of the Marind, often without the knowledge that they were from another ethno-linguistic group (Baal 1966).

The practice of collecting life-giving sexual fluid, through ritualized or serial sexual intercourse and/or wife sharing, was widespread across south coast New Guinea, parts of the highlands, and some other areas of Melanesia in the twentieth century (Knauft 1993). These were neither peripheral nor transient cultural practices and are an important theme linking widely differing linguistic and cultural groups in Melanesia. The Marind-Anim in particular were a highly demographically successful ethno-linguistic group, with an extended alliance system incorporating many neighbouring groups (and different language families). Internal relations were peaceful among some 16,000 persons and without any discernible hierarchical political structure, despite the fact that their cultural practices substantially influenced their demography and within-group genetic relatedness. The strategic use of adoption to bolster population declines is not unique to this group — in fact it is a feature of many other cultural groups around the world, too (Paul 2015).

It should be clear that paying attention to these cultural mechanisms reveals more than just some interesting details about how individuals might be interpreting their reproductive choices. The entire causal structure driving the maintenance of both a cultural practice (e.g. *otiv bombari*) and the ethno-cultural population that espouses it (the Marind) is brought to light by a deeper understanding of its internal cultural logic. Whether the individuals involved in this case were maximizing genetic output seems, if not irrelevant, then a problematic focus at best: lineages and groups were certainly being reproduced over time, but not necessarily via the production of genetic kin. As Paul (2015), in his treatment of the tensions between cultural and genetic inheritance, asks: "*by what right do we give the genetic [channel] preferential treatment in judging the whole system just described as 'maladaptive'?*" I would go further: if cultural practices like *otiv bombari* and ethno-demographic expansion are mutually reinforcing, why call any part of it maladaptive? And at what level (individual, group, institution) is it maladaptive? This example provokes us to think harder about the foundations of an evolutionary approach that claims that culture, broadly construed, is for calibrating individual behaviour to local ecologies in the service of reproductive success. The opposite scenario, where demographic activity (expansion, adoption, kidnapping) serves to ensure cultural continuity, is also clearly possible (Paul 2015). A co-evolutionary approach to demographic and cultural evolution that does not privilege one factor over another as being more causally forceful is needed.

All Cultures Are Cultures of Reproduction

We do not need to rely on examples in "natural fertility" contexts to see how culture influences reproduction in ways that alter demography at higher levels of aggregation. We can apply the same kind of thinking in a large-scale context. Consider contemporary western Germany, which has one of the lowest fertility rates in the world and some of the best maternity conditions, a public healthcare system and a high quality of life (among other things, a recent poll showed that Germans get more sleep than any other nation in Europe). And yet in western Germany a widespread stigma surrounds the return of women to the workplace after they become mothers, contributing to higher part-time and unemployment

rates among childbearing women (especially highly educated ones) and to persistent gender pay-gaps and inequality in the workplace (Grönlund and Magnusson 2016). Women who do return to work may be pejoratively referred to as *Rabenmutter* ("raven mother"): a derogatory term has been used in Germany for centuries to describe women who abandon their children and are thus considered bad parents. This phenomenon is pervasive in western Germany, where a traditional male-breadwinner model of the family dominates, but not in eastern Germany which was part of the socialist DDR (German Democratic Republic). In the west, highly educated women are more likely to remain childless and less likely to have children outside of marriage than in the former East Germany, where women tend to start reproduction at an earlier age and more readily envisage being working mothers (Bernardi and Keim 2017). As recently as 2012, only 27% of the highest-educated western German women used day-care facilities, compared to over 70% in the former East Germany. Opinions differed dramatically, too: 32% of all western German women agreed that pre-school children suffer when their mothers return to work during this period and 42% thought that family members should do the childcare. In the former East only 13% agreed that children suffer in this way and only 17% agreed that family should take up the childcare (Schober and Stahl 2014).

These differences show how the experience of different political regimes and historical events can persist in the reproductive decision-making of women today. These underlying values, which developed over historical and not evolutionary time periods and which have a range of demographic effects, only make sense within a particular cultural context. This kind of path dependency often gets overlooked in evolutionary demography. While this example is obviously not about a socially mandated practice like *otiv bombari* among the Marind, and while the institutional and economic context is arguably more complex, the social stigma of being a *Rabenmutter* in (western) Germany is nonetheless great enough that it is keeping many qualified women out of the workforce. This taboo is also at work among career-minded women avoiding childbearing (Bernardi and Keim 2017), and is therefore at least partly implicated in the continuing shrinking of the population, the persistent gender inequality in the workplace and other economic and social impacts. And to stretch the analogy with the Marind, the downstream effects are also comparable. As Germany's "indigenous" population declines and its age structure becomes unbalanced, leaving fewer young people and women to work and raise taxes, the state has resorted to "importing" its workforce through unprecedented levels of mass migration. This situation is currently the topic of heated debate as Germans revisit questions about their cultural values and identity in a contemporary multicultural context.

Distinguishing Causality in Cultures of Reproduction

These examples are not meant to claim that culture alone determines reproduction. Rather they serve to complicate our picture of the relationship between culture and demographic outcomes, and highlight the fuzzy distinctions between natural and controlled fertility. Should the Marind-Anim be described as a natural fertility population, when their reproductive decisions so obviously involve highly planned kidnapping and adoption of persons from unrelated groups? Should evidence that highly autonomous Western German women are culturally incentivized — probably largely unconsciously — into "stopping" behaviour be considered

part of the spectrum of natural fertility? Neither seems an appropriate description. Instead, the point I want to emphasize is that culture and demography co-evolve, sometimes to the benefit of a cultural entity (e.g. an ethno-linguistic group, institution or trait) and sometimes to the benefit of a demographic entity (e.g. an age-cohort or family lineage). Of course, such neat distinctions between cultural and demographic entities are not always going to be clear-cut, but this only further highlights the need for a co-evolutionary approach to addressing these questions.

But identifying causal mechanisms turns out to be harder in high fertility populations where cultural and genetic motivations may seem more in sync than in low fertility ones where the mismatch may be more obvious. This is a problem for our field because theories that are functionally equivalent (i.e. lead to equi-final outcomes) are not necessarily causally equivalent (Okasha 2006). Compare the following causal claims about the same hypothetical population:

> (1) Fertility is high because a history of political oppression and warfare in this region means that there are strong family ties and a high premium on demographic expansion at the expense of neighbouring ethnic groups. Group members collectively monitor these high-fertility norms and violations are sanctioned with ostracism. Contraceptive behaviour is forbidden and punishable by temporary exclusion from food-sharing networks.

> (2) Fertility is high in this non-industrialized "natural fertility" context (there is little evidence of contraceptive use or parity-specific stopping behaviour). People rely on traditional life-ways and a dense kin-network to support cooperative breeding. Women that use contraception have fewer resources and occupy marginal social network positions, which may indicate lower phenotypic quality or strategic birth spacing to avoid maternal depletion, to optimize reproductive output.

These statements could both be true. But they offer very different insights into the causal structure of fertility behaviour. It is important to qualify the causal claims of the second vignette with those of the first and vice versa. Doing so reveals that what looks like natural fertility may in fact be a highly deliberate and strategic use of reproduction for socio-political aims (see also Kanaaneh 2002). Take, for example, a point of apparent convergence between evolutionary and anthropological demographers: the — at first glance counter-intuitive — use of "modern" contraceptives to increase rather than decrease fertility in parts of sub-Saharan Africa (Bledsoe and others 1998, 1994; Johnson-Hanks 2002; Mace and Colleran 2009; Borgerhoff Mulder 2009; Alvergne and others 2013). The two sub-fields interpret the same behaviour similarly, but under different theoretical assumptions. Anthropological demographers have interpreted this behaviour as consciously strategic: women use modern contraceptives to optimally space births *in order* to achieve high fertility within a particular cultural context (Bledsoe and others 1994; Caldwell and Caldwell 1987). Evolutionary demographers, in contrast, typically focus on the inferences they can make about underlying trade-offs, for example, how improving mortality rates lead to increased competition between children, generating incentives to space births or slow down reproduction (Alvergne and others 2013).

High Fertility Is Neither a State of Nature, Nor Culturally Determined

The foregoing discussion raises broader issues related to how high fertility levels are characterized beyond anthropology and demography. First, contemporary high fertility is often thought to be culturally determined, exemplified in much public and even academic discourse about reproduction in the international development literature. In contrast, the low fertility of women in WEIRD (Henrich and others 2010) societies is often characterized as highly autonomous and somehow outside the realm of cultural norms. The example in Germany shows that this is not the case. Why should it be the case anywhere? We should instead assume that in all populations, reproduction is both negotiated by individuals and enculturated in them by the context they live in, subject to conscious and unconscious biases, and part of the feedback cycle between demographic, ecological and cultural conditions. This will help us avoid problematic distinctions between culturally constrained versus autonomous decision-making, as well as the problematic classification of some groups as in a state of natural fertility and therefore "traditional", frequently on the basis that they have many children, and others as "controlled" or "modern" because they have very few.

A second issue relates to how ancestral high fertility in human societies is often conceptualized. Here culture is rarely invoked, and fertility rates are seen as largely ecologically determined. Influential models related to the Neolithic transition, some of which take human behavioural ecology as their explanatory framework (Kennett and Winterhalder 2006), have regularly characterized reproduction in largely energetic terms. This same tendency is also reflected in more recent modelling on the ecological sustainability of the human population (e.g. Weinberger and others 2017). In general, there is a revealing disjunct between how transitions to high fertility are theorized compared to transitions to low fertility. In the words of the palaeo-demographer Bocquet-Appel:

The major difference between the two demographic transitions is that the cause of the NDT [*Neolithic Demographic Transition*] was unconscious, determined by the mechanical effect on maternal energetics of the invention of the agricultural economy, while the essential cause of the CDT [*Contemporary Demographic Transition*] was conscious, the will to control mortality and reproduction. (Bocquet-Appel 2009).

Naturalizing high fertility as the logical physiological outcome of resource availability is problematic for many reasons, chief among which is that it downgrades the causal power of culture in creating high fertility contexts. If we agree that both low and high fertility in contemporary contexts are plausibly driven by cultural evolutionary dynamics, shouldn't we apply the same principles to ancestral fertility? While we know that fertility rates among extant, and presumably, ancestral hunter-gatherers are low compared to those of farmers (Bentley and others 1994, 1993), this difference is most often interpreted in terms of resource constraints on reproduction, much less in terms of cultural ones. Recent cultural evolution work has begun highlighting how the cultural features of "small-scale", egalitarian socio-political systems can influence demographic patterns. For example, social norms that level the reproductive playing field in a population via suppression of the reproduction of high status individuals, may be an important strategy for maximizing both within and between-group cooperation (Gavrilets and Fortunato 2014; Bowles 2006). This is a kind of *reverse-dominance*, where the weak can combine forces to dominate the strong,

and such mechanisms are thought to be a fundamental feature of hunter gatherer social organization (Boehm 2001), which has important implications for our understanding of their demography.

The transition to farming was as much about changing cultural processes, inter-group dynamics and the rise of new kinds of inequality as it was about the nutritional or ecological benefits of changing resource-availability. And we know that in Europe at least, the process of population growth during the Neolithic was not smooth. Boom-and-bust population dynamics (Shennan and others 2013) strongly indicate endogenous causes of population fluctuations, rather than climatic events. The first farmers were not as successful in their cultivation of crops as were hunter gatherers in their own subsistence activities (Bowles 2011) and the transition came with steep increases in a range of diseases and pathologies, increasing mortality rates (Bocquet-Appel 2011). The general picture is of higher fertility as an adaptive response to higher morbidity and mortality (Page and others 2016) in which cultural processes tend to play, if anything, a minor role. But simulation-work has shown that it would have taken a joint emergence of cultural institutions and technological innovations to explain why new, initially less profitable and higher-risk subsistence practices would have been consistently adopted and maintained by ancestral hunter gatherers (Bowles and Choi 2013). Cultural mechanisms including rules regulating appropriate behaviour, property rights, marriage and dispersal would all have contributed to reproductive ideologies. Complex exchange and trade networks were also a key feature of the Neolithic transition (Ibáñez and others 2015) and these networks would have contributed significantly to the diffusion of new cultural information and technologies, as well as buffering the risk of losing this accumulated culture (Derex and Boyd 2016; Powell and others 2009). How culturally mediated reproductive decisions contributed to these dynamics is as yet unknown. A better understanding of the mechanistic basis of reproduction and how it scales up to generate population-level patterns has enormous contributions to make to our interpretation of these changes in our evolutionary history.

Expanding the Toolkit for Studying Reproduction

Hammel (1990) outlined three components of an anthropologically respectable theory of culture for demography: (1) micro-level explanatory mechanisms grounded in demographically-relevant social networks; (2) appreciation that social information is continuously updated, modified and anticipated by interlocutors, and (3) a much stronger reliance on indigenous or emic categories and interpretations, via detailed ethnography.

The first of these is already well established in evolutionary anthropology, with social networks becoming a major focus in recent years. Although relatively little of this work has focused on demographic questions, social networks have been embraced as a means to capture the dyadic and interconnected influences on reproductive and contraceptive decision-making (Mace and Colleran 2009; Alvergne and others 2011; Colleran and Mace 2015; Borgerhoff Mulder 2009). This work builds on seminal work by demographers in the 1990s and 2000s (Kohler and others 2001; Rutenberg and Watkins 1997; Bongaarts and Watkins 1996; Montgomery and others 1998; Behrman and others 2002) and is an important route to integrating cultural evolutionary theory into evolutionary demography. For example, demographic work has shown that opinion leaders and people with central social network

positions have a disproportionate influence on women's contraceptive use and ideation (Valente and Pumpuang 2007; Gayen and Raeside 2010; Kincaid 2000; Gayen and Raeside 2007), consistent with prestige-bias models of cultural evolution (Richerson and Boyd 2005). The specific contraceptive methods that a community ends up endorsing can be highly path-dependent when women rely on their social networks for contraceptive information (Entwisle and others 1996; Kohler 1997). Threshold effects have been shown to be important, as has the size, composition and density of ego-networks, all of which can enable rapid dissemination of new information that facilitates behavioural and cultural change or by strongly reinforcing anti-contraceptive norms (Montgomery and Casterline 1996; Kohler and others 2001; Colleran and Mace 2015; Colleran 2020). The network structure of larger meta-populations can also change the rate at which cultural change proceeds both within and between communities (Borenstein and others 2006; Derex and Boyd 2016; Powell and others 2009).

Second, the continuous nature of social information updating is less well formalized in evolutionary demography, although the state and context-dependent nature of theorizing in human behavioural ecology can handle stochastic or frequency-dependent change in ecological or cultural circumstances. Work focusing on how exogenous institutional or economic changes, for example changing land inheritance practices, have affected reproductive outcomes allows us to make inferences about changing parental investment decisions (Gibson and Gurmu 2011; Colleran 2014). A greater focus both on this kind of research and on even finer-grained perceptions of and attitudes to social change (Schaffnit and others 2019) will help to understand how the costs and benefits of reproduction are interpreted.

Third, the incorporation of "insider" viewpoints and detailed ethnography is much needed. Some evolutionary researchers engaged in long-term field research include discussion of proximate mechanisms to contextualize the results of their work, but this remains a minority. There is much to be gained through a deeper engagement with the insights as well as the publishing models from anthropological demography. Demographic categories and research protocols that we as WEIRD researchers take for granted are often inaccurate (Hruschka and others 2018). Survey instruments are in some cases overly blunt. We must not give up the idea that participant observation and a deep engagement with ethnographic literature can yield transformative insights for our field, leading to new hypotheses, models or approaches — this is as true for human behavioural ecology as it is for cultural evolution. Evolutionary researchers have largely embraced the challenge of explaining culture using quantitative models, but we should not assume that the theoretical landscape has been fully explored. Theory emerges as much from the iterative feedback that comes from regular close attention to the lived experience of our interlocutors as it does from the theorizing we engage in from a distance.

But evolutionary anthropologists differ significantly from other anthropologists in their publication practices, focusing more on short-form scientific publications, with almost no extended ethnographic work or monographs. Anthropological demographers are notable for their contributions to both genres. While some disciplinary journals such as *Human Nature* welcome descriptive or ethnographic articles, many evolutionary anthropologists struggle to have their work accepted in flagship anthropology journals, in part because of an anti-evolution

bias, further discouraging them from investing in ethnographic writing. One way forward may be to develop new venues and approaches for the publication of ethnography with numbers.

Closer attention to ethnography brings many benefits: it may help avoid the polarization between cultural and economic determinants of reproduction (Pollak and Watkins 1993) and de-emphasize the distinction between proximate and ultimate causation that is sometimes inappropriate when talking about contemporary reproductive contexts. Instead, a focus on the multilevel and co-evolutionary nature of human demographic behaviour is needed. This highlights that different parts of the system of demography are driven by different evolutionary processes. Perhaps we can partition the understanding and analysis of fertility behaviour into different conceptual components: for example, the origins, spread and maintenance of low fertility behaviour (Colleran 2016). Each of these can be tackled with different methods and data, but under a common framework that provides tools to connect the different parts.

It is in providing these connections that a broader cultural evolutionary framework is useful. This also makes room for reciprocal causation (Laland and others 2011), emergent properties (Smaldino 2014) and group level causation (Richerson and others 2014), all of which necessitate that macro- and micro-level phenomena are not merely reduced to one another. It should lead us to take the spatial and social structures of human populations more seriously, as well as the interactions between different parts of a population, because they generate both boundaries and conduits to cultural diffusion, changing patterns of (cultural) evolution. The identification and measurement of these patterns will help us to better understand the patterning of reproductive outcomes and to highlight the power-dynamic, structural and institutional contexts of reproduction. Evolutionary demographers have examined socioeconomic patterns in reproductive trade-offs, especially in highly developed economies (Fieder and others 2005; Nettle and Pollet 2008; Barthold and others 2012). However much less research has focused on interactions between different social strata (Colleran and others 2014).

Finally, it is important to recognize that the time frames over which we as empirical researchers work (a few generations at most) may not generalize well to evolutionary time scales. We know, for example, that in the mid to long-term, low fertility does not seem to provide a clear fitness benefit for individuals, though it certainly seems to pay off socioeconomically (Goodman and others 2012). There are trade-offs between the rather "myopic" short-term scales of behavioural adaptation and the longer-term adaptations of groups and lineages. This does not mean we cannot study these dynamics in an evolutionary context, but it does mean that we probably can't easily generalize from the logic of one generation's trade-offs in a particular population to the reaction norms of the evolutionary past (e.g. Baldini 2015). We should not abstract away the historical events that generate meaningful interventions in the cultural life of populations, and which alter the conditions for reproductive decision-making in ways that may have nothing to do with fitness maximization.

Concluding Remarks

This chapter is a first attempt to sketch out what a broader cultural evolutionary approach to evolutionary demography might look like. Demographic behaviour is part of the "complex whole" of behaviour, the webs of significance as Geertz put it, that we have spun and in which we are suspended (Geertz 1973). Reproductively relevant behaviours are learned and acquired as part of the cultural repertoires of social groups that have particular histories and value systems. Of course, these are coevolving with the fundamental ecological and energetic constraints of a place and time. But the implications and benefits of bringing "culture" closer to the core of evolutionary demographic thinking, are enormous. Doing so requires addressing the interrelations between culture and demography through different disciplinary traditions, at different levels of analysis, from different causal directions and through mechanistic thinking that engages with ethnography. Rethinking some of our basic assumptions about human reproduction is part of this endeavour.

No one should pretend that this will be an easy undertaking, or that we yet have all the tools we need. One obvious place to start is by incorporating cultural evolutionary theory, which currently combines population-genetic thinking (Cavalli-Sforza and Feldman 1981; Boyd and Richerson 1985) with psychological models from social psychology and behavioural economics to model the transmission of culture, as well as macro level analyses of path-dependent cultural change (Mesoudi 2011). Even here we do not yet have a ready-made quantitative landscape laid out before us, waiting to be applied judiciously to demographic behaviour. If the measurement of culture and its impact on population dynamics were straightforward, we would not have the tortuous history of anthropology that we do.

But while theoretical developments continue in modelling and experimental approaches to cultural evolution, we need ethnographic work to develop it further. More than any other evolutionary research field, evolutionary demographers are engaged in the complexities of fieldwork and the very real challenges of trying to quantify human social life. They already grapple with the tensions between qualitative and quantitative research and the difficulties of combining "insider" (emic) and "outsider" (etic) perspectives, either explicitly in their writings or pragmatically in their fieldwork (Mulder and others 1985; Wiessner 2016; Colleran and Mace 2011; Roth 2004). Nonetheless, they can often find themselves in what feels like an epistemological no-man's land, being neither completely committed to abstract models nor completely engaged in the kind of thick description typical of socio-cultural anthropology. This position should be seen as a strength and not a weakness, since these researchers are well placed to bridge the quantitative/interpretive gap as a result. Evolutionary anthropologists should feel free to pursue exploratory research that may not be immediately quantifiable. Some of course do this already, but ethnography has been greatly undervalued and underserviced in comparison to quantitative analysis.

By incorporating culture more fully into evolutionary demography, both in terms familiar from cultural evolutionary research and in terms familiar from socio-cultural anthropology, perhaps the different paradigms can become less mutually invisible. In doing so we would do well to avoid rehashing debates that have already occurred in demography, which are highly relevant to the (cultural) evolutionary analysis of reproductive behaviour. Whether a more interdisciplinary evolutionary demography requires that individual researchers employ

varied research methods, or that different disciplinary specialists come together, is difficult to gauge (Bernardi and Hutter 2007). Whatever combination of the terms "evolutionary", "cultural", "anthropology" and "demography" we decide to use, here I join other evolutionary anthropologists committed to making an integrated anthropology a basic component of evolutionary research (Fuentes 2016; Hewlett 2016; Wiessner 2016). A renewed enthusiasm for the insights of our colleagues in socio-cultural anthropology and greater engagement with proximate mechanisms, rather than avoiding them in the service of ultimate arguments, will undoubtedly expand and enrich the theoretical and empirical foundations of evolutionary demography.

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1 Note this chapter has been posted on the Open Science Framework website since 15/10/2019, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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23. Bateman's Principles and the Study of Evolutionary Demography

Monique Borgerhoff Mulder

Over the last 40 years, investigators have been applying ideas from the body of theory known as sexual selection to the behaviour of humans, breaking exciting new interdisciplinary ground. The Darwin-Bateman Paradigm has been central to this endeavour, essentially the idea that males are more competitive over mates than are females, and that mating success affects reproductive success more strongly in males than in females. Less known among social scientists is the fact that biologists continue a vigorous debate over the validity of this paradigm. In this chapter I take social scientists into some of these issues, to see how and why the study of the operation of sexual selection on males and females has changed as a result of clearer theory and better methods. The simple takeaway message is that in many species gender roles are much less distinct than original investigations of the Darwin-Bateman paradigm might suggest. Applying some of the emerging insights to humans, we find a surprisingly limited general understanding about the extent and patterning of variability in reproductive success in either sex. Furthermore, success in the marriage or mating pool is associated with a range of reproductive outcomes, both positive and negative, for men and women. A new methodological approach is proposed for studying the effects of mating success on reproductive success which may help to sort through some of the extensive variation in our species. More generally, the chapter argues that an updated understanding of Bateman's work might serve to guide evolutionary demographers today, just as Bateman's original work steered research in the early days of sociobiology.

Introduction

In 1948 Angus Bateman published a paper of enduring influence on evolutionary biology in which he tested Charles Darwin's ideas on sexual selection (Bateman 1948). From his experiments on fruit flies, he observed that the variance in the number of offspring left by males was greater than that left by females, and that this was largely due to variance in the number of females with whom males sired offspring. From this he inferred differential eagerness and discrimination over mating among males and females, and that this difference results from the fact that males can produce millions of small relatively cheap sperm whereas females produce fewer, larger, and relatively more expensive eggs. These sex–specific behavioural patterns were referred to as "coy" and "promiscuous", for females and males respectively. Arnold (1994) has clarified that Bateman actually derived three principles from his experiments: males have greater variance in reproductive success than females (Principle 1), males have greater

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variance in mating success than females (Principle 2), and reproductive success will increase with number of mates for males but not for females (Principle 3). The corollary of a stronger "Bateman gradient" (the regression of reproductive success on mating success) for males than females was that sexual selection will typically act more strongly on males than on females.

After several decades of relative obscurity these ideas were picked up by and Robert Trivers (1972) and Michael Wade and Stevan Arnold (1980), albeit focusing on rather different driving forces of sexual selection — Wade and Arnold on anisogamy (differences in the size and cost of sperm and egg, i.e. prezygotic investment) and Trivers more generally on sex differences in parental investment (pre and post zygotic). Bateman's three principles emerged as cornerstones to the study of sexual selection, structuring analyses of sex differences, the evolution of mating systems and the patterning of parental care. Indeed, as of October 2017 2018 Bateman's paper has been cited 3508 times, and enshrined as the Darwin-Bateman Paradigm (Dewsbury 2005; Parker & Birkhead 2013).

Over the years Bateman's ideas have undergone considerable challenge and re-examination (e.g., Hrdy 1986; Gowaty 1997; Klug et al. 2010), with critiques cantering on empirical, experimental, and theoretical considerations. Yet the basic intuitive logic of the paradigm survives this critique (Jones 2009; Krakauer et al. 2011; Parker & Birkhead 2013; Anthes et al. 2017; Henshaw et al. 2018). Furthermore much comparative evidence from the animal kingdom, including humans, supports the three principles (e.g., Janicke et al. 2016). That said, these critiques have significantly amplified and refined our understanding of the sex roles and mating strategies, and the conflicts between males and females more generally (Jones 2009; Anthes et al. 2017; Henshaw et al. 2018).

The objective of this chapter is to provide an update on the contemporary significance of Bateman's principles for human demography. Social scientists typically view biological approaches to gender and reproduction as deterministic, and with good reason (Wood & Eagly 2012). This is because evolutionary social scientists' expectations regarding sexual selection (recently reviewed in Puts 2016), and their heavy reliance on (presumed inherent) differences in parental investment between the sexes, often evoke stereotypic fixed gender roles (Borgerhoff Mulder 2010). This seriously mischaracterizes the diversity and patterning of gender differences in the ethnographic record (Eagly & Wood 1999). There is plenty of evidence that, for example, the division of labour is highly variable between societies (Bird 1999), that (like our non-human ancestors, Hrdy 1986; Hrdy 1997) women exhibit highly variable roles with respect to mate choice (Scelza 2013), and that this variability can be explained in part by socioecological factors as predicted by evolutionary models. Adult sex ratio, for example, influences attitudes towards promiscuity (Schacht & Borgerhoff Mulder 2015) and the patterning of violence (Schacht et al. 2014). Indeed we may not be quite the sexually-selected "peacock" some studies have suggested (as argued by Stewart-Williams & Thomas 2013).

Here I review the central role that Bateman's principles played in launching human sociobiology and evolutionary psychology as empirical fields of investigation [2]. I then examine the critiques of the paradigm, highlighting those of most relevance for human studies [3], before returning to current understandings of Bateman's principles in human demography, bringing attention to the new challenges that emerge and some possible ways forward [4]. I end with remarks on future horizons and intersections with societal values [5]. The second section is primarily of historical interest, and serves largely as an introduction to early human sociobiology

and evolutionary psychology for those unfamiliar with these fields. The third section is more technical, providing an update on how debates over Bateman's work, in both experimental and theoretical literature within evolutionary biology, are opening up new questions with regard to the study of multiple mating, sexual selection, the measures thereof, and the inferences that can be drawn. This will be of most interest to human evolutionary demographers who want to follow developments within the nonhuman literature, whereas Section 4 explores the implications therein for our empirical work as human evolutionary demographers, and draws further links to the standard demographic literature, identifying future directions. The final section examines, briefly, how changes in the study of sexual selection reflect changing social mores.

Bateman, Sociobiology and Evolutionary Psychology

Bateman's three principles, particularly through their influence on Trivers' (1972) characterization of the relationship between parental investment and sexual selection, were central to the founding of human sociobiology (Alexander 1974; Chagnon & Irons 1979) and evolutionary psychology (Symons 1979).

In the early days of applying evolutionary theory to human social behaviour the focus appears, at least in retrospect, to have been on demonstrating continuities between humans and other mammals, and indeed other animals more generally. Such continuities were justified on the basis of the shared evolutionary history of humans and nonhuman primates (e.g., Lovejoy 1981). However, the fields of human sociobiology and evolutionary psychology really took off with demonstrations that theory developed to explain variability in behaviour among birds, mammals, fish and insects could shed light on human patterns of sexual dimorphism (Alexander et al. 1979), mating systems (Dickemann 1979), and sex-biased parental investment (Hartung 1982). In other words, evolutionary scientists began to employ arguments for analogy (that behavioural similarities might arise from convergent evolution in the face of similar social or ecological challenges), as well as arguments for homology (similarities arise from common ancestry).

Initial interest focused on a low-hanging fruit — the greater variation in male than female reproductive success. In many small scale societies, including those with prescriptively monogamous marriage like the Pitcairn Islanders (Brown & Hotra 1988) and those living in complex states (Betzig 1986; Betzig 2012), men showed greater reproductive variability than women. Furthermore polygyny was not only widespread (Flinn & Low 1986), but patterned within populations according to the "polygyny threshold model" (Orians 1969); effectively following the prediction that polygyny will be more pronounced where men vary greatly in the resources they hold and women (or their families) select men according to their resources (Borgerhoff Mulder 1990). Such data were interpreted as strong evidence that human behaviour was a product of natural selection insofar as its variability within and between societies could be explained by theory developed for non-humans.

Researchers were also motivated to investigate the causes of differential reproductive success among men, noting that success in the reproductive sphere often correlates with success in the cultural, social or economic sphere (Irons 1979). For example, men with exceptional hunting skills (Kaplan & Hill 1985), or the ability to make efficient (or adaptive) marital decisions under specific ecological conditions (e.g., brothers sharing a wife in environments with limited

arable land, Crook & Crook 1988) show higher reproductive success than men without these traits. Even traits like the propensity to murder (Chagnon 1988) or rape (Thornhill & Thornhill 1983) might, under certain circumstances, be seen as adaptive strategy (insofar as the trait is associated with enhanced male fitness), although many such claims were controversial (Smith et al. 2001). Ambitiously, Irons (1979) suggested that success in the reproductive sphere might not only map onto, but also shape, emic definitions of success across different cultures.

Because of their interest in evolutionary processes investigators focused on variability in reproductive success (or fitness) and its determinants, often relying (explicitly or not) on Bongaarts' (1976) intermediate determinants of fertility — such as child survival (Sear et al. 2002), birth intervals (Blurton Jones 1986) or length of the lifespan (Perls et al. 1998; Penn & Smith 2007). This effective rapprochement to the discipline of demography (*sensu strictu*) was exemplified in papers identifying the principles of ecological (Low et al. 1992) or evolutionary (Low et al. 2002) demography that increasingly drew the interest (and collaboration) of more conventional demographers.

In retrospect, while foundational to the fields of sociobiology and evolutionary psychology, much of this work now appears quite coarse. Work was primarily correlational, with little attention to the development or transmission of traits. More specific critiques emerged: for example, Hrdy (1986; 1997) pointed repeatedly to the absence of attention to female strategies and counterstrategies against male control (see Borgerhoff Mulder & Rauch 2009; Scelza 2013), and Smith et al. (2001) drew attention to the tendency to deploy overly simplistic adaptationist logic. Interestingly, the view that women had little autonomy in pre-demographic populations has some cogency for demographers (Folbre 1983; Campbell et al. 2013). Furthermore the assumption that sex roles are universally structured by Bateman's principles has led to greatly exaggerated inferences regarding sex differences within the field of evolutionary psychology (as explored by Stewart-Williams & Thomas 2013). Yet, despite these problems, a body of theoretically-motivated empirical and interdisciplinary analyses was emerging, prompted by the hypothesis that the lower investing sex (men) follows very different reproductive strategies than the heavier investing sex (women).

Challenges to, and the Current Status of, Bateman's Principles

Within the sexual selection literature critiques have crystalized as a result of experimental, technical and theoretical advances. Problems have become apparent in the design of (and hence inferences from) Bateman's original experiments (most recently reviewed in Tang-Martínez 2016). At the same time new techniques have allowed for accurate determination of paternity (Birkhead 2000), and theoreticians have expressed concerns with the assumption of a deterministic relationship between anisogamy and post-zygotic parental investment (and sex roles) (Kokko & Jennions 2012). Each of these developments has challenged the idea that females necessarily benefit less than do males from multiple mating, and have prompted a much broader exploration of the theoretical significance of Bateman's gradients for the operation of sexual selection (Sutherland 1985; Klug et al. 2012; Parker & Birkhead 2013). The following section draws on the literature within evolutionary biology, and provides a more technical update on how these debates over Bateman's work are opening up new questions with regard to the study of multiple mating, the measures thereof, and the inferences to be drawn.

The procedural and statistical errors in Bateman's work have been much reviewed (e.g., Tang-Martínez 2012). Flaws have been identified in assessing paternity (Gowaty et al. 2012). Statistically, an overestimate of subjects with zero mates and an underestimate of subjects with more than one mate results in systematically-biased estimates of offspring number for males and females (Snyder & Gowaty 2007; Gowaty et al. 2012; see also Collet et al. 2014). Furthermore, not only Bateman, but those who cited the paper, chose to overlook the results of experiments labelled as "Series 1 to 4", emphasizing only the later "Series 5 and 6". In the earlier series females not only mate multiply but appear to benefit in terms of fitness therefrom. Indeed, if all the data are combined Bateman's third principle does not hold (Snyder & Gowaty 2007). Such successive simplifications of complex data sets can lead to the emergence of paradigms, which in themselves can act as further blinders to perceiving alternative patterning in the data (Tang-Martínez 2016). In this case, the Darwin-Bateman Paradigm has been formalized in textbooks, such as the early and highly influential text for human sociobiologists and evolutionary psychologists (Daly & Wilson 1978) and later texts (Buss 1999; Barrett et al. 2002). As a result, misrepresentations of male and female behaviour appeared in the literature that went far beyond what Bateman actually saw; in fact, Bateman's work was entirely non-behavioural.

Empirical findings that females can also benefit from multiple matings are now commonplace (Hauber & Lacey 2005; Simmons 2005; Clutton-Brock 2009; Gerlach et al. 2012). These findings do not of course mean that Bateman was wrong (Wade & Shuster 2005; Krakauer et al. 2011), nor that anisogamy (the initial sex differences in investment in reproduction) is irrelevant. Rather the debate that has arisen from trying to make sense of these "exceptions" has led to improvements in the modelling of sexual selection, specifically with respect to identifying causal priority in processes that are inherently complex and co-evolutionary (e.g., Jennions & Fromhage 2017). While the specifics of model dynamics need not concern us here, the pursuit of coherent and consistent models (McNamara et al. 2000; Kokko & Jennions 2008) and unbiased estimates of variance in reproductive success, variance in mating success, and the Bateman gradients (see Anthes et al. 2017 for a recent overview) are raising issues of direct relevance to the practice of evolutionary demography.

First, consider sex differences in the cost of reproduction. Sperm are of course cheaper to produce than eggs — this difference lies at the root of who is identified as male or female (anisogamy) (Kokko & Jennions 2012). While the literature is far too broad to cover here (for a good early review, see Wedell et al. 2002), the assumption that insemination is cheap is challenged in many systems, particularly insects, where seminal fluid, nuptial gifts, even body parts are contributed to females as part of male mating effort. For some species the critical sex differences in costs of reproduction that underlie Bateman's principles may have been overemphasized; and in some cases, they are reversed. Accordingly, males may not always be selected to mate indiscriminately, and male mate choice can be adaptive (Gowaty et al. 2002), and males have to face trade-offs between the number of females they inseminate and the quality of those females (e.g., Pélissié et al. 2014). As we discuss in the next section, this raises questions about how much reproduction can successful men get away with.

A second issue to consider is the accumulating evidence that females are neither necessarily coy nor discriminating over mating multiple times, and that this can contribute to considerable reproductive inequality among females. This is particularly in evidence in cooperatively breeding animals (Hauber & Lacey 2005), and again the literature is enormous

(Clutton-Brock & Huchard 2013). With the advent of molecular methods of determining paternity, fieldworkers found (first in many birds and now across taxa) that females engage in copulations with multiple mates regardless of the "social" mating system. Hypotheses for the adaptive value of this behaviour proliferated (Jennions & Petrie 2000), leading to active interest in polyandrous mating, dubbed (in the title of the opening chapter in a Themed Issue in Philosophical Transactions of the Royal Society) a "revolution" in our understanding of female reproductive strategies (Parker & Birkhead 2013). Accordingly, biologists' attention is now turning towards understanding both the causes of this variability among females, as well as the role of female competition (often overlooked in the literature, Stockley & Bro-Jørgensen 2011). Such competition has also been neglected within the human evolutionary demographic literature, as discussed in the next section.

A third development was the dedication of much empirical effort to both quantify Bateman gradients for each sex and, probably more importantly, scrutinize the legitimacy of inferences drawn regarding Bateman's third principle, the sex difference in slope of reproductive success on mating success. With respect to this latter goal there is now plenty of evidence that females benefit, and sometimes benefit proportionately more so than do males, from multiple mating (Hauber & Lacey 2005; Simmons 2005; Clutton-Brock 2009; Gerlach et al. 2012). This is the case even in the fruit fly family (Gowaty et al. 2002; Taylor et al. 2008) where Bateman did his work. Notably these "exceptions" occur not only in so-called sex-role reversed species (e.g., Jones et al. 2000) where (by definition) males provide more parental care than females and where such patterning might indeed be expected.

This initially unexpected patterning to Bateman gradients has led investigators to dig deeper into the mechanisms that mediate mating success and reproductive success (Tang-Martínez 2016; see also Henshaw et al. 2018). In some species the advantage to females from multiple mating appears to accrue through higher fecundity, in others improved offspring survival, and yet others longer lifespans (the role of genes and material benefits in contributing to these associations are as yet often unknown). Possible causes (or correlates) of these patterns are the costs of mating, the extent of paternal provisioning, and whether paternal provisioning exceeds maternal provisioning.

Species also differ in whether female reproductive success increases only when females mate with multiple males as opposed to mating repeatedly with one male. In observational studies we typically only know how often males and females mate, whereas in experimental studies (or studies where paternity is assigned through genetic markers) we may know only the number of sires of a female's offspring. With all of the evidence on how females can cryptically select (post copulation) which male fertilizes her ova (as in sperm competition, for example, Eberhard 1996; Birkhead 1998; Gasparini & Evans 2018), a male's observed mating success does not necessarily proxy for the numbers of offspring he sires. Conversely where sires are determined through genetic analysis, as in Bateman's experiments, investigators have no idea how many males a female mated with, only how many males have sired her offspring (Dewsbury 2005). In the nonhuman literature multiple mating by females can positively affect a female's reproductive success through various mechanisms — the nutrients in semen, the provision of nuptial gifts, additional care from extra pair mates, or backup partners if the current one dies. But it is also clear that multiple mating can have negative outcomes for health and lifespan, as reviewed both long ago (Snowdon 1997) and more recently (Tang-Martínez 2016). How these patterns

intersect with number of sires and/or number of matings is as yet unclear, a question that has potentially important implications for evolutionary human demography as discussed below.

The final points to emerge from this recent interest in Bateman's gradients are methodological, but important with respect to the drawing of accurate inferences from empirical studies of the relationship between mating success and reproductive success (Anthes et al. 2017). First, arithmetically it is necessarily easier to detect a larger number of sires when females produce a large number of eggs, so statistical associations are problematic and need to be corrected (Gagnon et al. 2012). Second, the causality may go the other way (Collet et al. 2014). If highly fecund females attract the attention of extra-pair males on account of their fecundity, the high fertility of multiple mating females will be a consequence of their reproductive performance rather than a cause (Ketterson et al. 1998; Gerlach et al. 2012). And third, a trait such as body size (associated with egg production in many species) could cause both greater mating success and greater fertility, such that correlations between mating and reproductive success may be partially spurious (Anthes et al. 2017).

Evolutionary biologists are increasingly recognizing these problems. Furthermore, in pursuit of the broader objective of differentiating the effects of sexual selection (big individuals get more mates) from those of natural selection (big individuals produce more, and larger, offspring), biologists are now starting to decompose the distinct components of mating success (Jones 2009; Pélissié et al. 2014; Janicke et al. 2015), and the paths whereby mating success affects reproductive success (e.g., Henshaw et al. 2018), issues picked up in the next section.

Bateman and Contemporary Evolutionary Demography

What does the current status of the Darwin-Bateman Paradigm mean for our understanding of human reproductive strategies, and evolutionary demography more generally? I address key points arising from the assessment above with some examples from studies in evolutionary demography and life history. This in no way substitutes for a full review of the relevant literatures, although I make references to standard demographic literature where relevant; further, I rely heavily on studies with which I have been associated. The goal is to highlight lacunae in our current understanding of human reproductive strategies, and to explore how a modern understanding of Bateman's work might serve to guide us again, as it did in the early days of sociobiology.

Variance in the Reproductive Success of Men and Women

Undoubtedly, many results from the earlier sociobiological studies (Section 2) hold. Men typically have greater variance in reproductive success than do women. In addition it is clear that there are strong associations between measures of culturally valued traits (*sensu* Irons 1979) and the reproductive success of men (and to some extent women), not only in traditional (pre-demographic) societies (Smith 2004; Nettle & Pollet 2008) but also in industrial populations (Stulp et al. 2016). These associations proxy as estimates for selection coefficients of single generation individual reproductive success on cultural success, and are of similar magnitude to selection coefficients estimated for nonhumans (as reviewed in Nettle & Pollet 2008). Further indications of significant variance in male reproductive success comes from genetic data from distinct patrilines; these studies reveal high rates of some Y-chromosomal lineages going extinct with others expanding markedly (Zerjal 2003; Balaresque et al. 2015). Recent studies improve

methodologically on earlier work on sex differences in variance in reproductive success and, where possible, use data less prone to bias (towards or against the culturally successfully), for example church records of births and deaths (Courtiol et al. 2012). With such materials for eighteenth-/nineteenth-century Finns, sex difference in variance in reproductive success can be apportioned quantitatively to men's higher variance in early survival, in ever-marrying, in their number of marriages and the fertility of their wives.

Before moving on to women, it bears noting that there is little precise understanding of what limits variability in male reproductive success. Consistent with the polygyny threshold model (Orians 1969), reproductive inequality among men should increase with increasing wealth inequality, but it doesn't (Ross et al. 2018). Physiologically there are few relevant constraints so the answer to this question turns more on social norms and trade-offs. While there are likely to be societal benefits from men coming to some amicable agreement over the partitioning of reproduction (e.g., Alexander 1979; Hawkes et al. 1995; Henrich et al. 2012), the precise means whereby this happens are unclear. Of course paternal provisioning and complementary biparental care play a big role in structuring the trade-offs that shape men's reproductive strategy (Kaplan 1996; Kaplan et al. 2009; Hooper et al. 2014), but how exactly these check acute competition among males is still unclear. What might account for the relatively (compared to non-humans, see below) muted male variance? There may be aspects of male provisioning that are not infinitely divisible among wives, mates and offspring that render the marrying of many wives fitness-depleting (Fortunato & Archetti 2009). Or perhaps the explanation lies in the particular structure of wealth inequality. Ross et al (2018) show how when wealth is not only very unequally distributed but skewed towards the very few men who own the lion's share of the wealth, there are simply too few men to pass the polygyny threshold holding too higher percentage of the wealth, serving to reduce both polygyny and male reproductive variance (see also Oh et al. Under review). Or maybe the effects of social censor (effectively punishment) of deviant males who break the norms of sexual behaviour are sufficient (as Smith et al. 2001 proposed in their critique of evolutionary arguments concerning rape); even then, however, the origin of these values regarding morality and justice need explanation, as they are clearly not universal.

Let us turn now to the general expectation from the Darwin-Bateman Paradigm that women will exhibit less variability in reproductive success than men. As reviewed above, there are both theoretical and empirical grounds for scrutinizing the underlying assumptions and evidence associated with this idea. Looking specifically at humans, Brown et al (2009) catalogue how variation in women's reproductive success may have been seriously underestimated. One problem is poor data on extramarital births, although this is a problem for birth histories of both males and females, (and more so for males if children typically reside with their biological mothers than their biological fathers). A second problem arises from the almost universalistic assumption regarding the critical importance to women of paternal care in their offspring, namely that their reproductive success is highly contingent on male care. While biparental care is undoubtedly a central adaptation within human evolution (Kaplan & Lancaster 2003), this does not mean that under specific circumstances women cannot show as great a variance in reproductive success than men, nor that they cannot find provisioning elsewhere (Hrdy 2005; Kramer & Ellison 2010). Indeed among the Pimbwe farmers, fishers and hunters of Tanzania there is no significant variance in mating or reproductive success between men and women

(Borgerhoff Mulder 2009; Borgerhoff Mulder & Ross 2019). Furthermore men must also be concerned with the survival of their children and the paternity of their next child, and this can lead to somewhat counter-intuitive predictions (*vis à vis* the Darwin–Bateman Paradigm) regarding what is, or is not, in both men's and women's fitness interests (Moya et al. 2016). However perhaps the most serious problem noted by Brown et al. (2009), arises from the labels that we typically use for marriage systems — monogamy, serial monogamy and polygyny. Notably serial monogamy proxies for serial monogyny but not serial monandry. As such, even though polyandrous marriage as an institution is rare, our reliance on formal labels makes women who mate and/or marry multiply appear to be much less numerous than they are (see also Starkweather & Hames 2012). In short, because humans are so variable in how they organize both their production and reproduction, and the labels for marriage systems are somewhat male-biased, we suspect that human populations are unlikely to conform uniformly to Bateman's first (and second) principles as initially expected.

To address this lack of systematic data with which to compare variance in reproductive success between men and women, and how such sex differences might differ from those of non-human social mammals, Ross et al. (In revision) analysed reproductive records from 97 small scale societies. We find that humans in these populations show significantly lower sex differences in reproductive inequality than non-human mammals. More intriguingly for the argument here, however, the difference between humans and non-human mammals (and particularly non-human primates), primarily reflects increased reproductive inequality among women rather than decreased reproductive variance among men.

This result suggest that much more attention should be directed in evolutionary demography to the factors responsible for variation among women, and the patterning of competition among women (for an overview see Fisher et al. 2013), possibly within the theoretical framework of cooperative breeding (Cant et al. 2009; Hill & Hurtado 2009; Lahdenpera et al. 2012; Mace & Alvergne 2012). Cooperative breeding promotes reproductive competition within families over the use of communally held resources (both material resources and access to helpers). Some have suggested that humans evolved as cooperative breeders, on the grounds that in many societies families/households consist of multiple adults, and reproductive-aged adults often help to provision or care for children at cost to their own reproduction. Intriguingly many issues here align with discussions among demographers over the definition of a household (Randall et al. 2011) and the cleavages therein with respect to intergenerational transmission (Quisumbing & Maluccio 2003) and illegitimacy (Koster 2018).

What We (Don't) Know about Bateman's Third Principle in Humans

Perhaps because of the lack of interest in variance in reproductive success among women, there are few published Bateman gradients for humans.[1] Moorad (2011) focuses on a colonizing population with polygyny and relatively high fertility (nineteenth-century Utahn), and Jokela et al. (2010), Käär et al. (1998) and Courtiol et al. (2012) on socially monogamous populations where multiple mating basically derives from divorce or widowing (twentieth-century US

1 This does not mean that such information would be unavailable after a systematic literature review, insofar as fertility can additionally be inferred from parity distributions. Notably, however, data on childbearing across multiple unions for males and females are rarely available in national censuses (Guzzo 2014).

citizens, eighteenth-/nineteenth-century Sami herders, and eighteenth-/nineteenth-century Finns respectively). In each case only men clearly benefitted from mating with different partners, whether through simultaneous (polygyny) or successive marriages (see, for more evidence on outcomes associated with second marriages, Forsberg & Tullberg 1995; Leonetti et al. 2007). Less conventional sex differences in the effects of mating success and reproductive success are found in the Pimbwe (Borgerhoff Mulder 2009) and the Hadza hunter-gatherers of Tanzania (Blurton Jones 2016), as discussed below.

Rather than simply counting partners to determine how mating strategies affect fitness, others have focused on the mechanisms whereby individuals can acquire multiple mates — through extra-marital affairs (including informal polyandry and partible paternity) as well as through multiple and/or serial marriages (or partnerships) with divorce. These issues (recently reviewed by Scelza 2013) are examined first, insofar as they can impact associations between partner number and reproductive success, before proposing a new way of unpacking the Bateman gradient.

Extrapair matings can potentially increase men's reproductive success (as long recognized by evolutionary social scientists, Perusse 1993; see also von Rueden et al. 2011, who show that high status men can simultaneously increase marital reproductive success as well as acheive more extramarital relationships). Women too can increase their reproductive success through extramarital affairs, as shown for the southern African agropastoral Himba (Scelza 2012), although in all these kinds of studies the bias of differential discovery risk, as noted above (Gerlach et al. 2012), remains to be addressed. Of relevance here are studies of the effects of informal polyandry on women's fitness. While formal polyandry is rare, systems in which women have sexual partnerships with multiple men who bear some economic responsibility for the children they have sired ("informal polyandry" Starkweather & Hames 2012) are much more common (and likely underreported). Furthermore, there are some societies (notably in Amazonia) with so-called "partible paternity" (a belief that a woman's multiple lovers contribute biologically to her pregnancy); here children "fathered" by more than one man show enhanced survival rates, perhaps because of additional provisioning or some other safety net afforded children with multiple fathers (Hill & Hurtado 1996; Beckerman et al. 1998). However, there is as yet little understanding of the full range of fitness costs and benefits of such informal polyandry systems (Walker et al. 2010; Scelza & Prall 2017), and more generally of whether or not the provisions of multiple fathers should be viewed as substitutes or complements.

Turning to more formally recognized unions, divorce and remarriage for men is generally associated with greater reproductive success; indeed successive marriage is the primary factor contributing to the persistent finding (reviewed above) that men have higher variance in reproductive success than do women (e.g., Jokela et al. 2010). In part this is because, after a divorce or widowing, men are more likely to remarry than women (as seen consistently, for example, across historical populations, Dupaquier et al. 1981), in part because of a tendency to marry second wives much younger than themselves (e.g., Starks & Blackie 2000), and in part because of longer reproductive lifespans. Nevertheless in many studies reverse causality, or confounding factors, may be at play — is there anything special about men who do go on to second marriages; for example, in the Bantu population with whom I work, men with three or more marriages tend to have fewer surviving children, perhaps because they are unable to keep partners for any length of time (Borgerhoff Mulder 2009). Furthermore, remarriage does not

necessarily bring a man fitness advantages — gains in fertility with new mates must be offset against any reduced survival of existing children, particularly if his divorced wife remarries (Daly & Wilson 1998) or if her resources become stretched. Using estimates of some of these parameters across a number of small scale societies (e.g., Sear & Mace 2008) Winking and Gurven (2011) nevertheless show that men's benefits from remarriage commonly exceed the costs. So why don't men divorce more often, or more generally why don't men's reproductive interests conflict more starkly with those of women? Clearly the trade-offs will depend on many ecologically- and socially-induced factors such as the certainty of paternity in first and second marriages, the availability of unattached women, and the substitutability of paternal care, including the response of women to the withdrawal of their partners' support. These are all classic issues studied by behavioural ecologists interested in mating systems (Clutton-Brock 1991; Borgerhoff Mulder 1992; Moya et al. 2016) that would benefit from more systematic demographic analysis.

As regards the effects of divorce and remarriage on women, the situation is less clear. Generally demographers, especially those who conduct studies in western industrial contexts, find that widowed or divorced women who remarry have lower overall fertility than those whose first marriages are still intact (Cohen & Sweet 1974), although some women do make up for first marriage fertility deficits with their second marriage (Thornton 1978). Lower overall fertility among women who have had multiple marriages may be quite general insofar as childless women (or those with low fertility) are more likely to seek divorce/be divorced than women in more reproductively productive marriage — indeed across many cultures childlessness promotes divorce (Betzig 1989). Reviews of the divorce literature typically focus on the factors precipitating divorce in western countries, and on the much contested outcomes for children of divorced parents, with little to no investigation of the impact on (or associations with) reproduction (Amato 2010).

Even with the emergence of studies focusing on multiple-partner fertility (MPF, the production of children with more than one partner, see Guzzo 2014) estimating the effects of multiple partners on fertility have proven to be a "surprisingly difficult task" (:72). This results not only from the high data requirements for identifying MPF, the widely variable estimates resulting from different sampling and definitional procedures, and the strong sample selective forces at play. For example, although MPF individuals tend to have half a child more than those who reproduce with only one partner (although the children generally have poorer wellbeing and mental health outcomes). This higher fertility is partially attributable to the low education levels, early age at first birth, and deprived socioeconomic circumstances of MPF parents, effects not yet clearly untangled; furthermore, new relationships can, under some circumstances, precipitate new pregnancies to cement the relationship. Alternatively, where MPF is associated with lower fertility, this might reflect substantive causative factors, such as reduced support from kin (Harknett & Knab 2007) who are less inclined to invest in households with unrelated children. Lowered fertility among MPF individuals could also result from selective bias, such as infertility and/or marital discord, as well as the time lost to reproduction between marriages or partnerships. The complexity of these relationships is revealed in Lappegård and Rønsen's (2013) analysis showing that MPF among Norwegian women is most common in both the lowest and the highest socioeconomic strata — the former on account of high marital dissolution, the latter perhaps because of greater attractiveness of MPF to women with economic autonomy.

Clearly such nuanced studies of MPF, and indeed of the quality of partnership relationship (as reviewed in Balbo et al. 2013) can shed much needed mechanistic light on Bateman gradients for men and women.

In the evolutionary demographic literature, there are some scattered reports of women benefitting reproductively from multiple marriages. In a study of rural Bolivian women, those with three sequential marital partners have more children than woman with only one spouse (an anecdotal observation in Snopkowski 2016), which could result from remarrying wealthier men (as with US unmarried mothers, Bzostek et al. 2012). Similarly Indian Khasi women in second marriages show shorter interbirth intervals than women in first marriages (Leonetti et al. 2007), and Pimbwe women who marry three or more times show higher fertility and reproductive success by the time they reach menopause (Borgerhoff Mulder 2009). Causality, as Gerlach et al. (2012) note in their analysis of Bateman gradients, is of course again a problem here, insofar as self-selection (or the non-random assortment of individuals into different (here) marital statuses) can bias analyses; for example highly fecund and/or hard working women may attract the attention of new potential spouses, such that their high fertility drives their mating success. Furthermore if is quite plausible that other phenotypic traits, such as health, might affect both a woman's ability to re-partner multiple times and her production of surviving offspring, thereby creating a spurious correlation between mating success and reproductive success, as reviewed in Anthes et al (2017).

In sum, it is plausible, but in no way demonstrated, that multiple marriage or mating could be an adaptive mating strategy for women if the future fitness gains with different partners are greater than the future fitness with a current partner. A key parameter to consider here is availability of preferable outside options to the divorcing woman, which must be weighed against a host of social and ecological parameters which might include: the costs of lost paternal (assuming the children follow the mother) investment to child survival and subsequent success, the extent to which stepfathers exert negative effects on child outcomes, and, more generally, the strength of the social support network to which a woman has access and with whom her children might reside. Indeed, the varying significance of paternal investment may account for the association between high reproductive rates of extramarital sex and limited heritable wealth transmitted through the male line (Gaulin & Schlegel 1980). Again, systematic analyses, as larger comparative data sets become available, would pay off, especially if they pay attention to differentiating the factors precipitating marital dissolution and successful remarriage, how these might differ between the sexes (e.g., Snopkowski 2016), and what the specific mechanisms for differential fertility contingent on partner number might be (e.g., Lappegård & Rønsen 2013).

Finally, it is worth pointing out that demographers have been dealing with these issues for many years. At a symposium on marriage and remarriage in 1979 they debated the extent to which the negative effects of divorce and widowing on overall fertility rates were compensated by remarriage (Dupaquier et al. 1981). This problem was deemed "insoluble" (:4) by Ashley Coale, and indeed ensuing chapters in the conference proceedings demonstrate the outcome is highly contingent on custom, religion, the division of labour, and property inheritance. Furthermore, it is eminently clear that the autonomy women enjoy with respect to their sexual behaviour is strongly influenced by laws that preferentially punish women's adultery over that of men, by genital mutilation, and by intimate partner violence, rendering the measure

of mating success a complex phenomenon. For all these reasons demographers would surely agree with evolutionary biologists that the Bateman's third principle not only needs careful measurement, but is unlikely to hold in all populations, and needs further unpacking with increasingly sophisticated statistical methods.

Unpacking Bateman

Why might Bateman's third principle hold in some populations but not others, and more interestingly why? To answer this question, we need to agree on what we mean by the term mating success. As reviewed above, evolutionary biologists studying sexual selection still disagree over how best to measure mating success. For those of us studying humans, this problem seems, *prima facie*, more straightforward. Demographers and ethnographers have for many years relied primarily on simply counting marriages, although increasingly investigators use more culturally appropriate arrangements, as Guzzo (2014) reviews historically for the USA and as anthropologists determine for their particular field contexts (e.g., Borgerhoff Mulder 2009; Scelza 2012). Leaving aside "known unknowns", such the issue of misreported or unknown paternities (which could presumably be integrated into a Bayesian uncertainty coefficient on the basis of population level estimates, Anderson 2006), should we simply be counting the number of partners/spouses? Or would we benefit from a decomposition of some of the elements of mating success?

Nicholas Blurton Jones (2016) decided counting spouses was not the best way to go forward. In his analysis of Bateman gradients in Hadza foragers of Tanzania, he chose the proportion of adult lifespan spent married as the best proxy of mating success, irrespective of the number of individuals partnered. He finds that Hadza men who spend much of their adult life married have the highest reproductive success whereas there is no systematic relationship for women between their success (in keeping children alive, the outcome measure is not directly comparable) and the proportion of their lifetime spent married.

In my review (Borgerhoff Mulder 2017) I queried why the amount of time spent married was the best measure of successful mating. It seemed to me there was more to mating success than keeping a spouse. I suggested that (from a female's point of view) "…if husbands are important and I am stuck with a bad one, best to ditch him and skip to another, even if it costs me a little time" (:126, and of course precisely the same argument can be made from a man's perspective). In other words, I questioned whether the percent of adult lifespan married is, on its own, a good proxy for mating success. There is undoubtedly some intuitive sense to Blurton Jones' decision — the ability to retain mates is important, especially if they are high quality and if constant biparental care from biological parents is important to child outcomes. Furthermore, he is correct to recognize that the effect of mate number on reproductive success is neither the only, nor indeed necessarily the most interesting, dimension on which the two sexes differ (a point now well recognized in sexual selection theory, as reviewed above). But is amount of time spent married really the best operationalization of mating success?

Motivated by this question, and the more general struggle demographers face in drawing inferences about the effects of divorce and remarriage/partnering on fertility (on account of causality and potential spurious correlations), a priority for evolutionary anthropologists now is to unpack the Bateman gradient.

Accordingly, we (Borgerhoff Mulder & Ross 2019) decided to reanalyse an updated and larger sample from the Pimbwe, a rural Bantu population of fishers, foragers and farmers living in western Tanzania. Rather than differentiating extrapair matings from divorce/remarriage (difficult insofar as the first so often leads to the second), and premarital sex from first marriages (again challenging without exact dates of relationship formation and/or pregnancies) we took a simpler (and more catholic) course, one also more suited to future comparative studies. We decided to develop models within which we could distinguished the effect of the number of different individuals with whom he/she had been married (or partnered) from number of years a focal individual has been married or partnered (as well as from the timing of the partnership, and partner quality, not discussed further here). To do this we use local concepts of "marriage" based on co-residence or shared parenthood. In many respects our "number of different individuals" parallels the new wave in conventional demography focusing on multiple-partner fertility (reviewed above), although we also include marriages/partnerships that are childless insofar as these entail effort towards mating success. In this way we can start unpacking the concept of mating success.

We show first that while men and women both benefit from the number of years they are married, men benefit more than do women. This is consistent with the Bateman's third principle, and likely reflects the existence of some polygyny in this population, as well as longer male reproductive lifespans. Second, and contrary to Bateman's third principle, women benefit whereas men suffer reproductively from increasing their number of mates, holding constant the effective time-frame over which they have been married. In this way we reveal distinct, sex-specific pathways (with respect to "mating success") through which reproductive success can be optimized. In short, we propose a model for analysing the effects of the number of years an individual is partnered, the number of distinct partners, as well as the timing and quality of these partnerships. This decomposition of mating success into its various components may prove useful in structuring future comparative analyses of Bateman's third principle in a more systematic way, as in the non-human studies reviewed above. Furthermore, it will be particularly valuable as more individual-level trait (such as economic status and education) are incorporated into the model as weights affecting fertility both directly and through marriage.

Future Horizons

In reviewing the status of Bateman's contributions to human demography I have identified where the amplification and refinement of his ideas reveal lacunae in our knowledge as evolutionary demographers. Particularly puzzling is why men don't have greater variability in reproductive success, especially in small-scale societies with little rival familial wealth to distribute among multiple offspring. Despite polygyny and serial monogamy, human males fall at the low end of variance in male fitness among mammals. I also noted the unusually high (again from a mammalian perspective) reproductive inequality among women. Finally, I pointed to the need for a better understanding of both the patterning of the Bateman gradients, and the social and ecological factors responsible for this variability.

With respect to the Bateman gradient, I proposed an unpacking of the concept of mating success, for two reasons. First, it is becoming increasingly clear from the debates over the Darwin-Bateman Paradigm that sexual selection is a hugely complex process, requiring highly dynamic modelling of a large number of traits (both fixed and context dependent) that are changing in

both evolutionary and ecological time. Even in nonhumans, where experimentation is possible, there is as yet very limited understanding of how genetic architecture, environmental dynamics and social interactions affect the continuous evolution of sexually selected traits (Kuijper et al. 2012). Second, there have been some puzzling discrepancies within human evolutionary demography with respect to how mating success is measured — number of spouses or number of years in marriage (Blurton Jones 2016; Borgerhoff Mulder 2017). Examining the distinct effects of each (spousal years and number of different spouses) may prove helpful in parsing out various dimensions of sex differences in reproductive strategy across different human populations. As an increasing number of studies with individual level data become available from multi-sited field research (Lawson et al. 2015), large national surveys (Snopkowski & Sear 2013) and compilations of individual studies (Borgerhoff Mulder et al. 2009) such simplifications may provide a useful first step for understanding sex differences in reproductive strategy as captured by the Bateman gradient.

Evolutionary social scientists' study of the Bateman gradient, perhaps unsurprisingly, focus primarily on marriage and its effects on men's fitness. Most researchers hail from western cultures where, until relatively recently, marriage was believed to play a large role in structuring reproduction, and where divorce typically promotes marriages of older men to younger women (Starks & Blackie 2000); as noted by Guzzo (2014), the conceptual tools of demographers reflect, presumably with some lag, actual behaviour on the ground. Indeed, as Nicholas Blurton Jones has pointed out (pers. comm.), it is largely researchers with longitudinal data from societies very different from our own (Borgerhoff Mulder 2009; Winking & Gurven 2011; Scelza 2012; Blurton Jones 2016) who recognize the stark economic and social trade-offs for men *and women* in sticking with one partner as opposed to engaging in the often dangerous pursuit of new partners, although again this is of course changing.

Darwin and Bateman's arguments reflected contemporary cultural values regarding women. Darwin was influenced by Victorian standards of his day (Hrdy 1997; Dewsbury 2005) and maybe Bateman was aware of the concerted campaign to get women out of the labour force and into the home following their active engagement in industrial production and other war-related activities (Tarrant 2006). Current discussions of the Darwin-Bateman paradigm are increasingly taking place in a very different world, one where not only sex roles but sexual identities are far less binary than ever imagined in the past; as such, posing sexual selection within a more gender-neutral framework is appealing (Gowaty & Hubbell 2005). We have clearly moved far from the Darwin-Bateman Paradigm view of sex roles as heavily bifurcated between choosy females versus indiscriminate and competitive males, with respect to theory development, empirical understanding of the animal kingdom, and our conceptualizations of gender in human society. We understand now that not only are sex roles highly flexible, but that they can be difficult to assign on the basis of single traits, as indicated in recent debates over the foundational role of anisogamy (Schärer et al. 2012; Ah-King & Ahnesjö 2013; Kokko et al. 2013); in fact there are some species without anisogamy, where only "mating types" can be identified. As such, there are plenty of new avenues for exchanging ideas between the natural and social sciences yet to explore. Although the purpose of this chapter has been to open up new questions for thinking about sexual selection within evolutionary demography, there are equally intriguing strands to follow with respect to how our current social concerns shape our science.

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24. What Are Couples Made of? Union Formation in High-income Societies

Anna Rotkirch

Compared to the evolutionary psychology of mating, the evolutionary demography of unions is little developed. We know quite a bit about why and how people have sex, much less about why and how they have spouses. Yet couples continue to be a central building block of families, the biosocial tie within which most adults live, most sex takes place, and through which most children are made and raised. Arguably, sexual selection in humans happens through long-term pair bonds rather than short-term relationships.

Evolutionary theory approaches unions as reproductive contracts: a precarious balance of conflict and compromise between individual reproductive and sexual strategies. Sexual strategies are predicted to vary especially with age, gender and resources, but also with ecological and social conditions such as increasing gender equality and lower fertility.

This chapter discusses the formation of unions in high-income, increasingly gender-equal societies from the intersection of family demography and evolutionary studies. How is selection of spouses affected by having more highly educated women in the population? Why does contemporary family formation often involve a stage of cohabitation before marriage? I argue that sexual strategies theory could move beyond the division into short-term versus long-term pair bonds, and suggest that cohabitation represents one mid-term form of temporal and psychological commitment to a romantic partner.

Key words: unions, union formation, sexual selection, parental investment, marriage, cohabitation, reproductive strategies, sexual strategies theory, sex ratios, homogamy, gender equality

Introduction

Humans form pair bonds, something which is rarely found among other animals. While all sexually reproducing species engage in intercourse, very few team up with long-term mates. Lasting bonds based on sexual attraction and attachment are found among the majority of birds but only in a few per cent of mammals (Clutton-Brock 1989; Lukas & Clutton-Brock 2013). Such relations often include living and sleeping together, acquiring and sharing resources, protecting and raising offspring, and also high if not absolute sexual fidelity from at least one partner (Alexander 1979; Chapais 2011). These alliances are called "pair bonds" by biologists and "unions" by demographers. While the terms are not fully overlapping — for instance, a union often implies living together and social recognition of the relationship, which all pair bonds do not

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have — I will here use the words unions, couples and long-term pair bonds as synonyms. Humans as well as many other pair bonded species have both opposite- and same-sex couples. The main evolved emotions and behaviours relating to pair bonds — e.g., wanting to be together all the time when you are in love — do not vary much by sexual orientation. The differences and similarities between same- and opposite-sex couples open an important window for understanding sex differences (see e.g. Kolk & Andersson 2020) but are not the focus of this chapter. In what follows, I have tried to point out when a specific claim does vary by sexual orientation.

Throughout the contemporary world, most adults live in socially monogamous long-term unions, and most adults have one or at most two such unions during their lives (Cherlin 2017; Mayyasi 2016). Intercourse typically takes place between long-term partners (Wellings et al. 2006) and most children are born and raised by a couple. Paradoxically, however, we currently have many studies about whom individuals would like to have sex with, much fewer about why and how they actually commit to living with a spouse.

This chapter discusses recent demographic research on union formation in high-income societies, or societies currently at the mature stages of the demographic transition with its shift to longer life spans and lower and later fertility. The twentieth century witnessed three major changes in family life related to childbearing, gender relations and unions. Humans globally started having later and fewer children, patriarchal power was weakened and the institution of marriage changed character (Therborn 2004). These three changes were spearheaded by developments in the Nordic countries and Western Europe.

Traditional patriarchal societies are characterized by early and universal marriage, early and high fertility, and deference of the younger generations to the older generations and of women to men. Social control of sexual behaviour, especially of women's sexuality, is at the core of patriarchy. More liberal and individualised societies have later and lower rates of marriage, later and lower fertility, more equality between both the generations and the two sexes, and much greater leeway for individual sexual behaviours and gender identities. These changes in power relations and ecological conditions shape the lives of many contemporary couples.

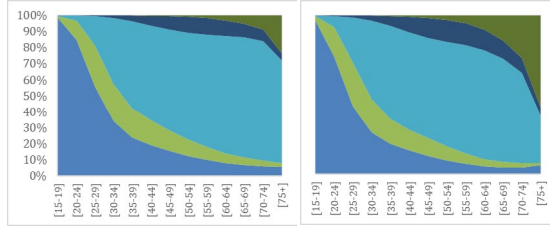
What does individualisation and liberalisation mean for how we fall in love and commit to a partner? How are evolved preferences enacted as women and men become more equal and have at most only a few children, relatively late in their lives? This chapter first provides an overview of long-term pair bonds as an evolved part of human sociality. I then outline their importance for the dynamics of sexual selection and the reproductive and sexual strategies of our species. The third part discusses changes and continuities in union formation in the light of recent demographic research from high-income societies.

A More or Less Durable Character

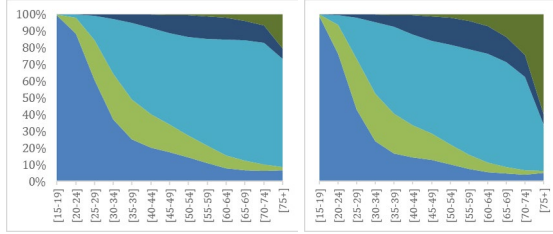
The ability to live in unions is a human universal, a permanent feature of our social repertoire. "In the human race the relations of the sexes are, as a rule, of a more or less durable character", observed Edward Westermarck in *History of Human Marriage* (1891), the founding classic of evolutionary family studies. Westermarck was the first to systematically document the huge variety of cultural norms and laws regulating human marriages. He was also the first to stress that marital institutions and norms would never have appeared in the first place without an evolved basis in the human psyche: the skill to create and sustain unions and the substantial emotional and cognitive efforts this entails. To Westermarck, effectively, "marriage" meant

what demographers now call "unions", and his history of human marriage built on the idea that marriages do not occur solely in our own species.

As noted by Westermarck, a first indication of the evolved nature of the pair bond is its universality: unions are prominent in all known historical societies and throughout the contemporary world (e.g. Low 2011; Eastwick 2013 and 2016). The prevalence of unions today can be observed from data on marital status compiled by the United Nations. Globally, almost 80% of both women and men have been married before they turn 50 (UNDP 20115 and 2018; Mayyasi 2016). Figure 1 illustrates marital status by age for men and women in the United Kingdom (1a-b) and Norway (1c-d), two of the countries for which detailed comparative data on both marriages and cohabitations is available. Norway and the United Kingdom are both wealthy countries, but have different welfare state systems and ranks in gender equality (in 2018, Norway was 5th and the United Kingdom 27th regarding gender equality, UNDP 2018). Yet the overall picture of partnership dynamics at different stages of the life course in the two countries is remarkably similar: marriage remains the modal type of living.



1a. United Kingdom, men, 1b. United Kingdom, women



1c. Norway, men; 1d. Norway, women

Fig. 1a–d Union status over the life time in the United Kingdom and in Norway in 2011 for men (left) and women (right) by age group.

Source: United Nations Marital database and UN data.

Despite the spread of divorce and alternative forms of living arrangements, most adults in the UK and in Norway marry, and marriage is typically followed by widowhood. As Figures 1a-d illustrate, living together without being married is not uncommon, especially among people in their 30s and 40s and in Norway. Cohabitation, spearheaded by Northern and Western Europe in the 1960s, is now spreading to most other parts of the world (e.g. Esteve et al. 2012). The question of what this entails for union dynamics and stability has stimulated much research in demography, less so in evolutionary studies.

The Figures also show that more men than women never live with a spouse. For instance, at age 45–49, right after their prime childbearing years, 16% of men in the United Kingdom and 18% of men in Norway are single (had never had a union), compared to 12% of women in the UK and 13% of women in Norway. The gender difference is due both to the fact that more boys than girls are born, so that males are overrepresented especially in younger age groups, and to greater variation in union formation among males. We also see that even if men are typically more often single in youth and middle age, women are more likely to be living without a spouse in old age. The gender gap in widowhood has narrowed in recent decades due to fewer wars and other gains in male life expectancy (Bildtgård & Öberg 2017).

Another indication of the evolved nature of pair bonds is how popular they remain. Even in wealthy societies with large individual choice, few wish to live without a long-term romantic partner. For instance, 90% of contemporary Finns state that they would ideally like to live in some kind of union. Around 75% say a monogamous union, without other romantic or sexual partners, is their own ideal. Within this group, two thirds would prefer to be married and one third prefers cohabitation without being officially married. (Kontula 2016, 40–41.)

Among Finnish women, a monogamous union is the most preferred type of living arrangement. Living-apart-together, or being a couple but not sharing the same household, is the second most preferred type, preferred by 13% of Finnish women. Among men, a monogamous union was also the most preferred type, while living in a union and also having many sexual partners was the second most preferred type, supported by 15%. (Kontula 2016, 40–41.) Interestingly, in addition to these slight gender differences, the wish for a long-term partner also appears to vary with biological fecundity. While most Finns in all age groups prefer to live in a union, this proportion is lowest among women who have passed their reproductive years. Hence the gender gap in union status among elderly people illustrated in the Figures above is not only due to differences in mortality, it also reflects personal preferences. Among Finns aged 55–74 in 2015, 26% of single women but only 7% of single men said staying single was their preferred way of life at the moment. Elderly women are also the demographic which is least likely to remarry (see e.g. Schweizer 2019 for data from the United States).

When comparing survey data over time, the proportion of single and post-reproductive women who do wish to have a partner has increased over time. This may reflect increases in living standards and more permissive cultural norms. And yet older women remain the demographic group to which unions are least attractive in Finland (Kontula 2016); similar results are found in Sweden (Bildtgård & Öberg 2017). Such gender and age differences in our attitudes to unions can be explained by sexual strategies theory, as discussed below.

The Evolution of Love

The ability to form and stay in couples is underpinned by a triad of love-related states: sexual desire, romantic love and companionate love (Aron et al. 2005; Feldman 2017; Acevedo et al. 2012). Combined, these characteristics define our common understanding of what a couple is. They also distinguish romantic partners from other close dyadic relations, such as those between parent and child, friends or siblings.

While lust and infatuation often characterise the first years of a relationship, companionate love is more common at later stages. These emotions feed into each other in intricate emotional loops (although desire and romantic love are, tellingly, not actually emotions, but rather states

of physiological and psychological arousal and addiction). Romantic love often includes a desire to have sex, but having sex can also make the partners fall in love — not uncommon, for instance, in arranged marriages, or in "friends-with-benefits" relationships. Companionate love, in turn, thrives on sexual and emotional intimacy. (Fisher et al. 2002; Bartels & Zeki 2004.)

This is not to say that all human unions include all forms of love; some have none. Unions are multi-layered and different states interweave differently during a long couple relation. As a dialogue between two married persons expressed it in a popular TV series:

> Marriage means different things to different people.
>
> – What does it mean to you?
>
> – Well, I used to think it meant there was this one person I would put above anyone else. Above myself.
>
> – And now?
>
> – Now I just hope I don't kill him.
>
> (*The Affair*, HBO Season 1 Episode 2, 2014)

Individuals who are not close genetical relatives lack a direct incentive to cooperate altruistically for prolonged periods of time (Hamilton 1964). This challenge can be overcome as lust and romance make two unrelated persons bond closely — to the extent of putting the other "above anyone else". Romantic infatuation can, in turn, foster a lasting attachment, which is more about respect and adjustment, or at least tolerating each other.

Distinct hormonal patterns and neurological correlates underlie the three main romantic and sexual feelings (e.g. Bartels & Zeki 2004). We also know that specific receptors, facilitating the expression of oxytocin and vasopressin in the brain, are associated with monogamous family bonds in both humans and prairie voles (Young & Wang 1998). That the same hormones have been linked to monogamy in both humans and a small rodent suggests that other species also fall in love.

During our evolutionary history, sex, love and attachment would have served to create a team of two, who, with the help of kin and in-laws, was strong enough to raise children and grandchildren. The expression "long-term" hence denotes at least some years of paired living, but can also expand into decades. This vague temporal definition (what is "long"?) may not be sufficient to understand what is going on in today's societies. Another interesting question is when "long-term" begins. Is it when the couple first meets, falls in love or moves in together? Is it when the bond is recognised by kin and friends?[1] Or maybe a union comes into being when both partners intend it to last?

One caveat is worth making at this point. In contemporary societies, mating success is no longer clearly linked to reproductive success, or the representation of an individual's genes in subsequent generations. Throughout human history and until very recently, striving for social status and accumulating resources were among the set of behaviours that translated into having both more children and more surviving children. This is not always the case any longer. During the demographic transition, increasing acceptance of sex outside marriage, use of birth control and social norms favouring low and late fertility have spread, and the link between heterosexual intercourse

1 "If I could choose a beginning / how about the first ending, / the one that made / everything possible", as Andrew Johnston (2008) suggests in his poem *Fireflies*.

and procreation has been severed (Mace 2008; Alvergne & Lummaa 2010; Goodman et al. 2012). This does not mean that contemporary union dynamics would no longer be shaped by evolved preferences, or that reproduction would happen without sensitivity to evolved environmental cues of the right time to have a child (Stulp, Sear & Barrett 2016; Stulp et al. 2016). Neither does it mean that humans are no longer subject to natural or sexual selection (cf. Courtiol et al. 2012). On the contrary, increasing prevalence of childlessness in high-income societies may intensify the strength of selection pressures today (see e.g. Fieder & Huber 2007; Barthold, Myrskylä & Jones 2012).

Sexual Selection in Unions

Long-term pair bonds have seldom evolved among animals. Interestingly, species who do have pair bonds also tend to have higher cognitive ability than other, closely related species, and to provide bi-parental care to their offspring (Shultz et al. 2014). Furthermore, once evolved, pair bonds rarely disappear from the psychological make-up of a species. This suggests that pair bonds are costly to develop and maintain, yet yield some important evolutionary benefits when they have been established on a species level. Crucially, this also suggests that opportunities for sexual selection in pair-bonded species continue long after the first mating.

Sexual selection, as originally defined by Charles Darwin in *Descent of Man and Selection in Relation to Sex* (1872), is driven by differences in mating success. Any trait that provides an advantage over a rival in securing a mate is subject to sexual selection. Darwin understood sexual selection to happen through two main processes: *intrasexual competition* among individuals of the same sex for access to individuals of the opposite sex, and selection of mates, or *mate choice*, between the sexes. He correctly predicted that in most species, females would be the choosing sex, while males would compete for access to females. In species with long-term pair bonds, however, sexual selection is often two-way and mutual, so that both males and females compete for mates.

Currently, sexual selection is defined, even more sparsely, as acquiring access to gametes of the opposite sex (Jennions & Kokko 2010). In addition to intrasexual competition and mate choice, access to gametes can also be achieved through violence, coercion and manipulation. *Sexual conflict* or conflict between men and women is therefore now also often included among the main processes of sexual selection (Borgerhoff Mulder & Rauch 2009).

In species with predominantly one-way sexual selection, so that males compete and females choose — the main template according to Darwin — access to gametes is largely dependent on the initial phases of courtship. By contrast, in a species such as ours, with mutual mate choice and long-term unions, access to gametes depends not only on acquiring a sexual partner, but also on keeping that partner and guarding him or her from potential rivals. Perhaps not coincidentally, one hypothesis to explain why monogamous pair bonds originally evolved in primates is male mate-guarding of solitary females (Lukas & Clutton-Brock 2013).

What goes on within pair bonds will hence be at least as important for sexual selection as getting a sex partner (cf. Strassmann 1981; Bird 1999). Most human offspring are born to a couple, who stay together for at least a few years. (Contemporary Europe has the highest prevalence of births outside marriage and they are a minority in all countries, see Coleman, 2013.) In contemporary high-income societies, never having a child is not only more common among people who never form any relationships, but also among those who have only brief spells of living together with a partner. For instance, among contemporary Finns who did not have children of their own, 45% had never lived together with a partner, 25% had had one brief cohabitation, and 19% had

lived in several short cohabitations (Jalovaara & Fasang 2017; see also Saarela & Skirbekk 2020). Moving in with a spouse is the end of fairy tales, but not of sexual selection.

Among couples who do manage to stay together, the quality of their relationship and how often they have sex will affect childbearing (see e.g. Witting et al. 2008; Schwartz, MacDonald & Heuhel 1980). When couples disagree, it is usually around communication, intimacy and sex, which in turn is related to marital adjustment and relationship satisfaction (e.g. Kurdek 1994; Byers 2005).

Male control over female fertility is culturally engrained in many societies (Strassmann et al. 2012). For a long time, marriage was also culturally understood to provide men with unlimited sexual access to their spouses. The concept of female choice and the feminist "right to say no", including refusing intercourse with her partner, spread more widely only in the 1970s; today marital rape is criminalised in most countries. (Hasday 2000.) This rapid cultural change is an instance where feminism has influenced one of the core dynamics of sexual selection and made it more costly for men to exercise sexual coercion.

Reproductive Strategies and Gender Equality

In *History of Human Marriage*, Westermarck criticised both religious conservatism and the social constructivists of his day, who theorised marriage as purely social institution. "I do believe that the mere instincts have played a very important part in the origin of social institutions and rules", Westermarck (1894, p. 5) noted, claiming that it is "impossible to believe that there ever was a time when conjugal affection was entirely wanting in the human race" (ibid., p. 360) comments that are still controversial or provocative in some academic quarters.[2] Westermarck also discussed sexual selection and conjugal affect in relation to childbearing. However, the link between parental care and sexual selection was formalised much later, when Robert L. Trivers formulated parental investment theory (Trivers 1972). *Parental investment* denotes all types of resources, including fertilisation, gestation, provision, protection, care and education, that a parent invests in an offspring, and which detracts from resources that could be spent on other offspring, existing or potential. It can also include mobilisation of other individuals to invest in the offspring, as is so often the case with human children who are reared cooperatively (Hrdy 2009; Sear & Coall 2011).

Parental investment theory predicts that the sex that invests more in offspring is also choosier with regards to sexual partners, while the other sex is predicted to experience more intense competition among its members for access to mates. This creates tensions or "trade-offs" in resource allocation. *Reproductive strategies* are a set of behaviours reflecting different solutions to such trade-offs, notably the trade-off between mating and reproduction. Another trade-off is that between reproduction and so-called somatic growth — investing in own growth, maintenance and development, compared to investing in others, e.g. a partner or a child (Bird 1999; see the introduction and other chapters for discussions of life history theory in this book).

Females of reproductive age are predicted to be the more limiting sex in mating behaviour, while males are predicted to gain more from mating with several partners. This general principle is valid, yet risks obscuring that "it takes two to tango" and the actual advantage will depend on actual access to new mates, or advantages of paternal investment (Kokko & Jennions 2003). In some situations, males will not benefit genetically from having many partners, while

2 For the debate about the nature of marriage between Westermarck and Émile Durkheim, the founder of sociology, see Roos (2008).

females can (Scelza 2013),[3] and in some societies both males and females can best increase their reproductive success through investing in one long-term partner (Brown, Laland & Borgerhoff Mulder 2009; Forsberg & Tullberg 1995). Among humans, the latter is predicted especially when families have relatively few offspring that are costly to raise (Strassmann 1981; Rueden & Jaeggi 2016) — as is the case in contemporary high-income societies.

Reproductive strategies will vary not only with gender, but also with health, age, social status and many other factors (Gangestad 2007, p. 322). They can exist on a species level and within different subgroups in a population. So-called *conditional reproductive strategies* are environmentally sensitive to ecological factors, such as the availability of material resources, kin support or partners (Marlowe 2010). For instance, abundant resources and population density can be expected to speed up union formation, making it easier to locate and settle with a partner, while resource scarcity and few eligible partners can be expected to delay it (Kokko & Jennmions 2012). Technological advances such as the bicycle, or currently social media, also serve to speed up processes of union formation by enlarging the potential mating market.

Sex Ratios and Intrasexual Competition

Studies of adult sex ratios and family formation have usefully illustrated ecological effects on reproductive strategies (South and Trent 1988; Low 1990; Durante et al. 2012). If the sex ratio is male-biased, i.e. there are more males than females in a given age group, male intrasexual competition is predicted to intensify. For instance, in the contemporary United States, higher male sex ratios are associated with increased male risk-taking in economic behaviour (Griskevicius et al. 2012).

Male-biased sex ratios are often a cue for sociosexual behaviours favouring monogamous unions and paternal investment, while a female-biased sex ratio is a cue for male promiscuity, and greater acceptance among heterosexual women of men having several partners or not committing to one partner only (Schmitt 2015). However, the results are not uniform, documented effects of sex ratios on sociosexual behaviour are usually small, and studies have often been correlational and lack individual longitudinal data (Schacht et al. 2017).

That sex ratios continue to trigger such stereotypical patterns of male and female romantic preferences even in more gender-equal societies can come as a surprise. There are, however, also other important dynamics at play, for instance related to intra-sexual competition. Social status and gendered norms channel how mate choice and intrasexual competition is acted out in different populations (Uggla & Mace 2017; Jonason & Antoon 2019). For instance, male–male competition need not manifest as physical aggression (Kokko, Klug & Jennions 2012). A recent study of counties in the United States found that, contrary to what could be expected, violence and crime, including rape and sexual assault, were lower when sex ratios had a surplus of men (Schacht, Tharp & Smith 2016). The authors stress that higher male sex ratios need not necessarily elevate male–male intrasexual competition through aggression.

High sex ratios can foster violence between men and intensify the oppression of women, limiting their freedom of movement and social contacts outside the household. Male competition can, however, also take the form of men "competing" in being good partners and investing fathers. In more gender-equal societies, having more men to choose from can also give heterosexual women

3 See Borgerhoff Mulder's chapter on the Bateman gradient in this volume.

greater bargaining power in romantic relationships, resulting in more marriages compared to cohabitation, earlier childbearing and more stable marriages (Pedersen 1991; Schmitt 2005). On the other hand, a surplus of women may instead make some men intensify mate-guarding efforts, including violent control of women (Schacht, Tharp & Smith 2016).

When Are Unions More Stable?

Unions can be seen as a compromise between male and female reproductive strategies, a *reproductive contract*. From this Darwinian perspective, a union is a transaction in which spouses exchange love, care, time and money, social status, etc. (Borgerhoff Mulder & Rauch 2009, p. 210). A central premise is that patterns of union formation and dissolution — of conflict, infidelity, divorce and remarriage — can be predicted on the basis of the reproductive interests of the spouses, which rarely fully align. The relative risks of entering and exiting a union for a specific individual are not stable over time, since individuals develop and age and their living conditions change. (Buckle et al 1996; Daly and Wilson 2000.)

Living as a couple, individuals have to tolerate close cooperation with another individual to whom they are usually not closely genetically related. Having a partner creates four main risks: the risk of desertion, the risk of the other free-riding in resource contribution to the joint household, the risk of abuse and violence, and the risk of the other taking up rival short- or long-term partners, who can detract household resources away from you and your children. This is true for all types of couples; male–female couples additionally also experience sexual conflict and antagonism due to differences in male and female reproductive strategies and interests.

Relatives pose an additional risk. Closely related individuals have aligned reproductive interests, and are often also emotionally very close to each other. Hence spouses face the "in-law challenge" of having to interact with unrelated relatives. In-laws can provide access to information, support and resources, but they can also undermine the solidarity between the spouses and cause strains. Couples often quarrel about how much they see which relatives, and these types of disputes can affect relationship satisfaction in a couple, often especially so among women (Kurdek 1994). A similar tension may arise with step-children. (Borgerhoff Mulder & Rauch 2009; Daly & Wilson 2000.)

The terms "reproductive *contract*" reflects the precarious balance of spousal harmony and division of work. As Bird (1999) puts it, conflicting reproductive strategies between males and females may result in "less than optimal compromises with regard to mating and parenting". In other words, one gender or one individual spouse can rarely "have it all" and both have to adjust. But when does cooperation prevail, and when are conflicts more likely?

If two individuals have a child together, their future reproductive success becomes linked. This will soften the in-law and step-child challenge; through the existence of a shared descendant, a parent becomes "inversely" related to the other parent of the child, as well as to his or her relatives (Hughes 1988; for the effect of having children on in-law relations in contemporary societies see Danielsbacka, Tanskanen & Rotkirch 2015). Having a shared descendant also alleviates all the other risks related to living with a spouse that were mentioned above, since both spouses are now invested in each other's reproductive interests.

This is why couple solidarity can be so very high, by far exceeding solidarity among other social dyads who are not related to each other, such as friends. Spousal solidarity and mutual commitment are likely to be especially strong in the following conditions: a monogamous union

with joint children, when there is low risk of desertion or other sexual partners, and when own reproduction is the main way to increase reproductive success, as opposed to investing in reproduction of own kin, e.g. in nephews and nieces (Alexander 1979; Daly & Wilson 2000; Moya, Snopkowski & Sear 2016).

Male and female reproductive strategies are more likely to conflict when there is a distinct trade-off between mating and parenting, so that one behaviour actually excludes the other. Whether or not that is the case will depend on the division of work in society, which types of work men and women engage in and with whom they do the work, and how children are raised (Bird 1991). By contrast, sexual conflict is predicted to be weaker, and sexual cooperation more prevailing, when both men and women gain greater benefits from investing in children, and when all adults can engage together in resource acquisition with high consumption benefits. (Bird 1999; 72–73; Wilson and Daly 2001.) Interestingly, it is this latter description that is typical for high-income societies, with dual breadwinner families, mixed labour markets and large freedom of movement, and very high investment in children by parents.

Time or Commitment?

An important addition to reproductive strategies came when sexual strategies theory was developed in the early 1990s (Buss & Schmitt 1993.) Sexual strategies theory predicts that, given mutual partner choice, men and women will value quite similar characteristics in their romantic partners: intellect, resources, social status, and so on. It also predicts gender differences in mating preferences. Women are expected to put relatively more emphasis on resources and commitment, and on investment in children by her partner, while men are expected to put relatively more value on youth, beauty, and other signs associated with female fecundity. Again, it is important to keep in mind that these sex differences are assumed to characterise both same- and opposite sex couples. (Buss & Schmitt 1993; Buss & Schmitt 2019.)

Sexual strategies theory thus added a temporal dimension to reproductive strategies, in separating between long-term pair bonds as opposed to short-term matings. Crucially, gender differences in sexual strategies are predicted to be more salient for short-term than for long-term partners. This is because the risks and possible benefits of a short-term heterosexual mating have always been drastically different for men and women: men usually risk at most acquiring a sexually transmitted disease, or a broken heart, while women additionally risk life (through pregnancy, childbirth, jealous partners, or social sanctions). Once aiming for a long-term relationship, however, the gender differences are attenuated and selection is more mutual. In a long-term relationship both men and women value trust, reliability, humour, and kindness in a partner. (Buss & Schmitt 1993; Gangestad & Simpson 2000; Schmitt et al. 2001; Eastwick & Hunt 2014; Eastwick et al. 2014).

Some gender differences appear to persist also in preferences for long-term partners. Contemporary populations still prefer the man to be older than the woman, and stress the earning capacity of men but the physical beauty of women. The evidence is there, although to what extent these differences generalize across societies (Sear & Marlowe 2009), or may disappear as society changes (Durante et al. 2016), remains debated. Often, the overall pattern of sexual and romantic preferences goes in the direction predicted by sexual strategies theory, although the magnitude of gender differences can vary (Zentner & Eagly 2015).

Sexual strategies theory has been debated for the separation between short-term mating and long-term unions. One pertinent critique is that commitment can be a more crucial factor than

duration, and Marzoli et al. (2017) suggest that a classification based on levels of commitment might be more suitable than the temporal distinction. This goes back to the question of when a long-term pair bond can be said to begin. Perhaps the turning point is found in psychological motivations, which will eventually translate into investments into the relationship? Romeo and Juliet had a relationship that was short in duration, but high in mutual commitment. (Not that it helped their reproductive success.)

Another open question is whether sexual strategies theory postulates a continuum or envisages only two categorical states. While the original articles did not exclude other time horizons, empirical research methodology has usually forced people to choose between short and long(er) relations. Do humans in reality choose between short-term versus long-term, committed or not committed, or are there mid-term relations?

Finally, preferences are of course not the same as behaviour. Actual mate choice in humans is much more complicated than stated general preferences, not least since two partners are choosing. Furthermore, both partners need to align not only their own preferences, but those of their social networks (David-Barrett 2019).

Partly in response to these critiques, an exciting new body of research has tested and extended sexual strategies theory into contemporary population-based studies of how unions are actually formed. Next, I will discuss first how union formation is altered with growing gender equality, and then the role of cohabitation in high-income societies.

Unions Without Children — An Evolutionary Novelty

In last decades union formation has become less tied to childbearing, and marriages are formed later in life and often preceded by cohabitation (Coleman 2013). Men and women in high-income countries become parents at an increasingly later stage, with women approaching a mean age of 30 for age of first birth in several countries (Balbo, Billari & Mills 2013).

The trend of having children later in life (if at all) while leading a sexually active life and often having a steady partner from early adulthood is unprecedented in human history. In preindustrial societies, postponement of age of marriage was used to regulate sexual behaviour and hence fertility and population growth. Today, while age at first birth has been steadily rising in developed countries since the 1970s, women and men reach physiological sexual maturity earlier than previously, and also have their first sexual intercourse earlier than previously. Young adults in industrial nations have on average five sexually active years before starting to live with a partner (Wellings et al. 2006, p. 1710), and then at least two to three more years living with a partner before having a child.

Consequently, young adults have almost a decade of sexually active years before parenthood, many of them with a steady partner or spouse. What this means for our evolved predispositions to form and live in unions has been but tentatively explored. Previously, union formation and childbearing were intimately linked, part of the same biological and cultural "package" and often of the same calendar year.

Are Women Marrying "Down" Now?

Who people actually choose as a spouse is influenced by homophily, or the attraction of similarity, although complementarity of the spouses is also important (Štěrbová & Valentová 2013). Spouses are often similar in many sociodemographic traits including ethnicity, religion, age, height and level of education (Buss 1985; McPherson, Smith- & Cook 2001); evidence for assortative mating

has also been found on a genetic level (Robinson et al. 2017). From the perspective of sexual strategies theory, spousal homophily can been explained with the importance people assign to compatibility, mutual choice, commitment, and smooth kin relations in a long-term romantic relationship. As with friendship, compatibility and similarity can be assumed to promote cooperation by increasing trust and synchrony in intentions and actions (Laakasuo et al. 2020). Homophily is found in both heterosexual and same-sex couples (e.g. Schwartz & Graf 2009) and is related to stability of the union and to having more children (e.g. van Bavel 2012).

Hypergamy or women "marrying up" socially used also used to be quite common (e.g. Mare 1991). Hypergamy in heterosexual unions has been explained with the relatively larger emphasis women put on their partner's resources and the relatively larger emphasis men put on their partner's fertility, as predicted by sexual strategies theory. The opposite of hypergamy, hypogamy, denotes women entering unions with male partners of lower social status than themselves. This can result from women of higher resources having the freedom to pick good genes.

In recent decades the traditional gender gap in education, wage working and income has diminished in many Western societies. The dual breadwinner model is again gaining strength also in countries dominated by the male breadwinner model in the twentieth century, and in many countries, women have outnumbered men in higher education, although men on average continue to earn more than women do (e.g. Grow & van Bavel 2015). This begs the question of whether actual partner choice also changes, as the composition of the population changes?

Apparently, union formation adopts itself quite flexibly to a changing marriage market. Contrary to widespread popular beliefs, women are not so "picky" that they cannot settle for a man less educated than themselves. Results from the United States and Europe show that, as women's educational levels increased, more unions were formed in which the wife was more educated than her husband (Qian 2016; de Hauw et al. 2017). The traditional approach, in which women marry men of at least a similar level of education, has given way to a new trend, with women marrying men with at most the same level of education. As Hauw et al. (2016) show using European Social Survey data from 1970 to 2010, proportions of hypergamous marriages (women marrying "up") have decreased as proportions of hypogamous marriages (women marrying "down") have increased in Europe. Especially women with high education appear to prefer hypogamy to remaining single.

However, increased similarity in the educational level of spouses does not necessarily mean that earnings are more evenly distributed. As women are generally more educated in a couple, the share of female earnings in households has increased (Klesment & Bavel 2017). Nevertheless, the tendency for women to marry men with higher incomes than themselves has continued both in the European Union (ibid.) and in the United States (Qian 2016). Of course, this partly reflects the fact that average male earnings remain higher than average female earnings.

Interestingly, the same research team found that European men have not become more likely to partner with women more educated than themselves. Instead, they now remain single more often (de Hauw et al. 2016). In fact, less educated men in Europe currently have *less* chance of partnering with a woman of a similar or higher level of education, compared to the situation a few decades ago. Adding to the confusion, highly educated men also appear to remain single more often, perhaps as a result of being so "sought-after" that they prefer to wait and see before committing (or prefer never to commit). The authors suggest that the paradoxical finding that women are more likely to marry down, while men are not more likely to marry up, could relate

to partner choice outside the studied population, or outside the conventional age ranges among spouses. We know too little to settle the question for now.

Neither does the current spread of female hypogamy mean that evolved preferences have changed. Preferences do predict with whom people partner up, but they can also be adjusted downwards if it is hard to find a matching partner (Gerlach et al., 2017). Comparing mathematical preference models with actual behaviour, Grow & van Bavel (2015) show that recent shifts in assortative mating in Europe may have taken place without changes in gendered age-related preferences. Based on predictions from sexual strategy theory, the authors assumed that women would prefer to marry somewhat older partners and men would prefer women in their mid-twenties, and that both men and women would prefer a spouse with similar education and a high income. Compared to the strength of preferences regarding income and education, effects of age-related preferences on partner choice were small, and may have remained unchanged despite a changed population structure.

Finally, as hypogamy becomes more common in Western societies, its effects on fertility may not have changed. Some studies find that spousal homogamy continues to be related to higher reproductive success. In the United States, spouses with similar levels of education more often become parents compared to spouses with different levels of education (Huber & Fieber 2011). Also, in the United Kingdom, educational homogamy is associated with higher numbers of children (Krzyżanowska & Mascie-Taylor 2014). The finding that fewer children are born in hypogamous couples may relate to lower relationship satisfaction in such unions, which in turn could reflect a worse "fit" between initial preferences and outcomes (Brines & Joyner1999; Gerlach et al. 2017). That hypogamy tends to be associated with lower fertility (van Bavel 2012) also suggests an intriguing explanation for the current baby bust in high-income societies.

The finding that educated women do actually "marry down" while less well-educated men do not "marry up" both supports and contradicts some popular discourses and social groups. For instance, currently a social movement of so-called "incels" (involuntarily celibate males) blame women and society for their difficulties in acquiring heterosexual partners. This movement is interpreted as primarily an antifeminist reaction (e.g. Tolentino 2018), but can also feed on the real change in relative disadvantage among low-income males outlined above. Incels, who of course, represent a tiny proportion of this demographic, often propagate some vulgarised evolutionary views about mate choice, but without noting the differences between ideal preferences and actual union formation — and namely, that most people do get a partner and mate choice is not the exclusive right of the most attractive "alpha" males and "alpha" women. Evolutionary demography, through its sensitivity to mate choice, mating markets and assortative mating, could provide a complementary perspective on the sources and manifestations of this movement.

Cohabitation as a Test Marriage

With the weakening of patriarchy, alternative forms of living as a couple have become more widespread, including cohabitation. However, although largely accepted, cohabitation has not become a full-scale alternative to marriage in Western countries. Couples who stay together but prefer never to marry remain a minority (Perelli-Harris & Adams 2015). Instead, cohabitation is typically the first phase of the contemporary union, a stepping-stone between being single and being married, incorporating ingredients of both lifestyles (Rindfuss & van den Heuvel 1990). Today, living together is usually followed by either separation or marriage, while marriage is

usually followed by widowhood (Jalovaara & Kulu 2018). This life course pattern of unions was apparent in the union data from Norway in Figure 1 above.

That cohabitation can be interpreted as a prequel to marriage was demonstrated in detail by a comparative study of changes in union status among women of reproductive age in Europe and the US during 1945–74, conducted by Perelli-Harris and Lyons-Adams (2015). The authors identified seven life-course patterns in contemporary unions, depending on whether they were first formed as marriages or as cohabitation, and on their timing. Among the older generations, born in 1945–54, as many of half of the women had married in their early 20s without first cohabiting, and had then remained stably married over their reproductive years. Over time, such direct and early entry into lifelong marriage has become increasingly rare: among the youngest generation, born in 1965–74, this type is common only in Eastern European countries. In its place, across the rest of Europe, the dominant union formation type is cohabitation followed by marriage.

From an evolutionary perspective, the most striking result of this study is the stability of unions. All respondents grew up after the liberalisation of extramarital sex, contraception, divorce and remarriage, yet most formed a union and had children with only one partner. The vast majority (50–70%) of marriages remained stable throughout the prime reproductive years of the studied women. This again underscores the cohesion and often well-functioning solidarity among contemporary couples in high-income societies.

The result also begs the question, what reproductive and sexual strategies are served by such a two-stage union formation pattern?

Similar factors contribute to relationship satisfaction or durability for cohabiting as for married spouses — we are clearly dealing with a similar phenomenon (a union), but in a different package. For instance, the "honeymoon effect" with one year of elevated happiness can be just as strong both for couples starting to live together as well as for those who directly enter into marriage (Baranowska 2010). True, the effects of cohabiting versus marrying also vary by country and socio-economic class, and can be hard to disentangle from other factors signalling spousal commitment.

A wealth of studies has compared the union dynamics of cohabiting versus married couples. They indicate that in general, cohabitations are characterised by lower relationship quality between the spouses (Brown 2004; Brown, Manning & Payne 2015). Cohabiting couples are more likely to experience conflicts, to be of lower socio-economic status, to separate, not to have children, and to have other extra-pair sexual relations (e.g. Kiernan 2004; Lyngstad & Jalovaara 2010; Fincham & May 2017).

Why would individuals then agree to cohabitate? Especially, why would women do so? Given a choice, for instance in a situation with a surplus of eligible men, as discussed above, most women do tend to favour marriage over cohabitation. On the other hand, women, especially in their prime reproductive years, are most likely to initiate a divorce, both in same sex and heterosexual unions (Daly & Wilson 2000; Kolk & Andersson 2020) — perhaps because it makes sense to switch to a better partner before it is reproductively too late.

Cohabitation appears to be favoured, by both women and men, precisely because investment and exit costs are known to be lower. Or as Wilson and Daly (2001, p. 9) put it, cohabitation is "less likely to endure than registered marriage, and the participants know it". Hence cohabitation can be interpreted as spousal testing, comparable to trial marriages known in other societies. Has cohabitation replaced the prolonged engagement period from previous centuries, and if so, to what end?

One interpretation is that the increased frailty of the institution of marriage has resulted in a prolonged testing period prior to getting married. Especially in societies where many young adults need years to complete their education and gain a foothold in the labour market, and where the decision on whether to have children or not is made at increasingly older ages, pairing up happens in a low-information environment. How can you know if the person you fall in love with at age 20 or 25 will be the best partner for you ten years later, when you are at last able to buy a house or consider having a child? Increasing living standards and cultural expectations combined with stalled economic mobility and lower levels of help from kin, as well as shared custody after divorce, have also rendered people more apprehensive about the consequences of a possible divorce for themselves and for their child(ren). Supporting this interpretation, across countries, cohabitation appears to have spread in tandem to, or slightly after, the spread of divorce (Sánchez Gassen & Perelli-Harris 2015).

Is Cohabitation a Mid-term Relationship?

There is another interpretation of why people cohabit, in addition to the risk of divorce: the enduring association between marriage and fertility, and the very high investments in both parenting required today (Mace 2008). Maybe the goalposts of marriage are higher due to the changes in ecological and social conditions.

If so, contemporary cohabitation may represent one example of a semi-long pair bond: a middle phase between short-term matings and highly committed, long-term relationships. Due to the postponement of children in marriage and high levels of parental investment in each child, cohabitation emerges as a mid-term, relationship, a "couple contract", preceding the reproductive contract, and preparing for the transition to parenthood.

At the end of the trial period, the cohabiting couple typically reproduces and marries, or marries and reproduces. Formal marriage and childbearing are now occurring within the same time frame, only a few years from each other, even if the sequence can vary. As a consequence, most European children grow up with married parents. Even in countries such as Norway or France, where more than two in five of first children are born to a cohabiting couple, most women have married within three years of becoming a mother (Perelli-Harris et al. 2013).

A telling example is the civil solidarity pacts (PACS) in France. Originally introduced for homosexual couples (instead of granting them full legal rights to marry), but open to all couples, registered partnerships quickly became popular also among heterosexual couples. PACS represents as a formalised mid-step between dating and marriage, providing some economic benefits related to, for instance, income taxation, but not the same inheritance and adoption rights as marriage (Cody 2009).

Europeans do no longer necessarily perceive marriage to be strongly culturally prescribed; as young Norwegians put it in one study, the decision to marry or not is "up to the couple itself" and not of huge significance. In the most liberal and gender-equal societies such as the Nordic countries, the decisive transition is not the wedding, but the decision to have a child together. "It is children that constitute a family, not the partnership as such, and parenthood, not entering a union, that symbolizes the transition to adulthood", the authors of this study conclude (Lappegård & Noak 2015).

This Norwegian qualitative study interestingly suggests that reproduction and parental investment remain at the core of contemporary marriages (Lappegård & Noak 2015). Where

marriage historically used to drive parenthood, the norms have now reversed: "when co-parenting works, marry".

The link between childbearing and marriage is also reflected in the desire to culturally manifest the family unit to kin and society at large. And vice versa: a perceived bonus of not being fully married is not to have to interact with in-laws to a great extent. For instance, compared to being married and to having a child, cohabiting in the Netherlands is associated with a larger distance from the couple's parents (Blaauboer, Mulder & Zorlu 2011).

Unions as Somatic Maintenance

A growing minority in developed countries have long unions but no children. Childlessness within marriages remains low in Europe, but is increasing (Kreyenfeld, M., & Konietzka 2017). The DINK or "double income no kids" phenomenon was popularised in the 1980s and has spread especially since the Great Recession that started in 2008 (Friedman 2013).

This hints at yet another explanation for the popularity of cohabitation: it is often economically and emotionally preferable to singlehood. In liberal, high-income societies, having a spouse is good for you: it leads to better health and happiness (Baranowska 2010) and most sex is had within unions (Wellings et al. 2006; Gangestad 2007). Especially for men, having a spouse has a strong and positive effect on individual happiness (Kohler et al. 2005).

When cohabitation is not testing for the best partner, a mid-term contract on the way to a longer-term reproductive unit, it can also represent somatic investment, a way of life more practical and optimal than being single.

In high-income societies, unions are the primary household unit for breadwinning, consumption, and intimacy. The alternative to living in a union is increasingly often, especially in Northern Europe, not to live with close kin, as would have been the case earlier, but to live alone, without any of the economic and psychological benefits from sharing a household (cf. Rindfuss & van den Heuvel 1990; Becker 1991).

It has been suggested that parental investment has risen to levels devoid of any relation to reproductive success (Mace 2008) — can the same be said for investments in unions without children? Are we witnessing a process of unions as runaway somatic maintenance? If so, unions as units of production, consumption and sexual intercourse will be thriving, while the link to reproduction may become ever more strained.

Summary: Evolutionary Demography and Pair Bonds

Too many evolutionary tales of sexual selection stop when spouses commit to live "happily ever after". While mate choice has been relatively well investigated in evolutionary family studies, more attention needs to be paid to what follows it. It is within long-term pair bonds that the life events most relevant to reproductive success in contemporary societies — the making and rearing of children — take place. If anything, the importance of unions to sexual selection in high-income, low fertility societies, with extremely high levels of parental investment by both mothers and fathers, is probably stronger than ever.

Our romantic behaviour is to a considerable extent based on evolved dispositions that are species-typical and need not be culturally transmitted, such as the ability to fall in love and to stay in love, or certain partner preferences. Evolutionary theory conceptualises unions as a reproductive contract, an outcome of different sexual and reproductive strategies that is

characterised by both cooperation and conflict. This entails developing mutual trust, altruistic sharing and team work, keeping offspring alive, mate guarding, maybe having to cope with co-spouses or lovers, and avoiding the risks of spousal neglect or aggression. How these aims work out determines how close each individual, and each sex, comes to its preferred control of resources, partner preferences, and family size (Borgerhoff Mulder & Rauch 2009).

Unions today fulfil several functions, including emotional support, sex, reproduction and provision of parental care. Low and late fertility means that more couples never have children, and if they do, they will spend many years living together both before and after raising children together. The prolonged periods of living with a spouse without children testifies to the importance of unions not only for reproduction, but also for wellbeing and resource accumulation.

The universality of unions in human sociality, as first described by Edvard Westermarck almost 140 years ago, is still occasionally challenged in the social sciences. As illustrated here, an evolutionary perspective on unions does not exclude or disregard the variety of marital systems and family arrangements we have. On the contrary, it is through a Darwinian perspective one may fully appreciate how rare it is to have long-term pair bonds embedded within other social groups — the family, a circle of friends, a neighbourhood — and how intricate and complex our routine activities appear from the perspective of other mammals. Few if any other species have similar arrangements; that spouses usually interact closely with their in-laws appears to be unique for humans. (Hughes 1988.)

Evolutionary demography assumes some gendered differences in the interests, costs and benefits of union formation, but acknowledges that there may also be considerable overlap between the sexes. Ecological and cultural variation is at the heart of evolutionary theorising about marriages (Low 2007). Union formation is shaped by both gendered and conditional reproductive strategies, the latter reflecting population characteristics such as ecological or educational sex ratios, as discussed in this chapter.

Sexual selection and parental investment theory remain absent from mainstream family demography, for instance from second demographic transition theory (Lesthaeghe & Surkyn 1988; Lesthaeghe 2014) or gender revolution theory (e.g. Goldscheider, Bernhard & Lappegård 2015), which at best are indifferent to evolutionary approaches. Some approaches do accept basic biological differences, for instance the physiological effects of pregnancy and lactation, the greater average physical strength of males, and gender differences in temperament (Zentner & Eagly 2015). This does not, however, an evolutionary analysis make.

At its best, evolutionary theory stimulates research and provides a theoretical basis for generalising the principles of human behaviour across populations, and across species. It adds predictive power to family demography, by highlighting how genetic relatedness affects family dynamics, and how gendered reproductive strategies interact with conditional reproductive strategies. On the downside, the Darwinian emphasis on mating and reproduction may overlook other important functions that romantic unions currently serve. Evolutionary studies have tended to focus on mate choice rather than mate keeping, on short-term sex rather than long-term shared lives.

Postponement of childbearing within unions is an evolutionary and societal novelty, with interesting and understudied consequences for how reproductive and sexual strategies are currently enacted. Recent research has emphasized how the same evolutionary process, such as male–male competition for females, can result in widely different social dynamics, and for instance serve to either decrease or increase women's social status and freedom.

Another example discussed here is cohabitation, which has established itself as a specific, semi-institutionalised form of a trial marriage. I have suggested that cohabitation represents a mid-term sexual strategy, with commitment levels much higher than in a short-term sexual relation, yet lower than in formal marriage. Future research could investigate how cohabitation at different stages of life reflects different solutions to sexual conflict and gendered reproductive strategies.

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25. Cooperation and Competition Begin at Home: Bridging Household Ecology and Human Evolutionary Demography

Julia A. Jennings

Households are the next social unit above the individual and are home to shared activities that can include resource production, distribution and transmission in addition to reproduction and co-residence. They overlap with biological and social kin groups but are defined by activities rather than by relationships alone. This chapter reviews literature from historical and anthropological studies of households with relevance for human evolutionary demography. Selected research on household effects on mortality, fertility, and intra-household conflict across different agricultural societies is presented to familiarize evolutionary demographers with concepts, issues, and findings in the interdisciplinary and comparative literature on the household. Household researchers have drawn upon evolutionary concepts as part of explanatory models but are less likely to test evolutionary hypotheses directly. Evolutionary researchers have focused on familial relationships, but seldom consider power structures within households and the effects of household composition and dynamics on behaviour. Areas with potential for mutually beneficial collaboration between evolutionary and household analysts are highlighted and advocated for. Such collaborations have the potential to advance our understanding of the determinants of demographic behaviour by joining rich data sources with theoretical frameworks drawn from evolutionary and household perspectives.

Introduction

The household is an important unit of social and behavioural analysis as it is the context in which essential decisions are made and activities are carried out including production, consumption, and social and biological reproduction. This chapter aims to draw the attention of researchers in evolutionary social science to concepts and issues in household research relevant to human evolutionary demography. Households overlap with, yet are distinct from, family and kin. While kin are studied in a diverse range of evolutionary analyses, especially those that draw upon Hamilton's inclusive fitness (1964a; 1964b), households are considered less systematically even while the datasets used by many evolutionary demographers comes from collection methods driven by household concepts including censuses, household registers, and household surveys.

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The literature on household analysis and theory has been informed by a broad collection of disciplines, including anthropology, sociology, economics, demography, and history, especially historical demography. These fields have contributed to household definitions, cross-cultural comparisons of household composition and structure, analyses of household change over time, and descriptions of the social, cultural, and economic forces that affect and are affected by household formation and dissolution, household activities, and the roster of household members. There is potential for productive dialogue and collaborative efforts among researchers interested in human evolutionary demography and households. The opportunities and constraints on individual actions that are shaped by households may be overlooked in individual- or family-level analyses, so it is important for evolutionary analysts to consider the effects of these domestic contexts. Household researchers bring rich social, cultural, and historical understandings of the household to bear in their research, and evolutionary perspectives can assist their efforts to make cross-cultural comparisons and understand household effects on outcomes and behaviours, such as those tied to survival and reproduction. It is time for concerted efforts to bridge across these scholarly traditions to integrate households carefully and explicitly in the analysis and interpretation of human evolutionary demography.

While some have speculated on the evolutionary origins of human households (Quiatt and others 1985; Quiatt and Kelso 1987) or whether certain household forms have some basis in biology or human evolutionary history (Smith 1993), these are not the aims of this chapter. Instead, I wish to argue for the importance of household settings and dynamics in shaping behaviours of interest to human evolutionary demographers. More specifically, this chapter illustrate cases where predictions drawn from evolutionary theory may or may not align with results from analyses of mortality and fertility that include household-level variables or studies of intra-household processes and dynamics. This discussion is limited to literature and data from agricultural communities, especially those that practice traditional or preindustrial agriculture. There is a wealth of research from historical demographic and anthropological studies of agricultural societies, much of which includes longitudinal data and detailed cultural, economic, and historical contextual information to allow for comparative analysis and the investigation of household dynamics and change over time. Further, many household analyses of historical and anthropological agricultural populations may not be familiar to evolutionary researchers as much of this research does not draw explicitly from evolutionary theory, even if there is overlapping interest in the underlying behaviours or outcomes. In addition, household activities may look rather different across different economic systems. For example, co-residence may operate differently in foraging groups than in farming groups. The focus on one economic system reduces some of this comparative complexity for the purpose of this chapter.

Background: Households as a Unit of Analysis

Households are a fundamental social unit in human societies and are considered the first level of aggregation above the individual by some analysts (Hammel 1984). In several disciplines, including anthropology, sociology, and economics, households are considered an essential element of social and economic organization. Households are the context in which many demographic activities and related decisions occur. The household concept is commonly distinguished from family or kin through a focus on patterns of activity rather than on biological and social relatedness, although household and family typically overlap to some degree. For

example, some kin may co-reside within the same household while other kin may reside in separate independent households. Household activities include some combination of resource production, distribution and transmission; biological and social reproduction; and co-residence. Despite the ubiquity of households, settling on a single, cross-culturally appropriate definition has proven difficult. Some prefer definitions that emphasize co-residence (Laslett and Wall 1974; Verdon 1998), while others stress genealogical relationships (Hammel and Laslett 1974), collaborative work and other tasks (Carter 1984; Laslett 1983), or control of property (Gray and Gulliver 1964). Households change over time as members enter, exit, and grow older, and this complicates attempts at definition even further (Keilman and others 1988; Carter 1984; van de Walle 2016; Murphy 1996). To address the fluid nature of households in statistical analyses, some analysts treat households as contexts through which focal individuals pass during the life course (Ruggles 2009; Ruggles and Brower 2003; Hareven 1974). In demographic literature, the household concept is often driven by the definitions developed for census enumerations and survey instruments, which may not accurately represent the complex social realities in which people live and carry out activities (Kriel and others 2014; Randall and others 2011). For instance, census enumerations are designed to reduce double counting and therefore must assign individuals to only one household even if they consider themselves to be part of more than one household.

For the purposes of this chapter, I adopt the household concept of overlapping spheres of densely shared activities put forth by Wilk and Netting (1984). Households are groups of individuals that share a combination of production, distribution, transmission, reproduction, and co-residence activities. It is essential to note that the specific details of a Venn diagram describing the set of overlapping activities characteristic of households varies within and between societies and is subject to change over time. This definition of the household focuses on what these social groups do, rather than what they look like (household composition or morphology) or their symbolic or cognitive meanings. These aspects of the household are important, but for this discussion, I choose a definition that is flexible, applicable across a broad range of societies, and encompasses matters of interest to evolutionary and demographic analysts such as resource acquisition, resource distribution and consumption, and social and biological reproduction.

Household characteristics, including size and composition, are affected by demographic processes. Individuals may enter a household through birth, marriage, or migration and exit a household by death, marriage, or migration. Demographic models of the household often use microsimulation to understand how different age-specific rates of fertility and mortality, the timing of events such as marriage or first reproduction, and practices associated with post-marital residence, affect households (Burch 1970; Wachter 1987; Dyke 1981). These computational models assume sets of fertility and mortality rates and rules about marriage and co-residence to explore the effects of different demographic regimes and varying types of norms and customs concerning household formation and dissolution on measures of household composition, such as the frequency of three-generation households. While demographic events shape household size and composition, there is evidence for feedback between demographic events and households, such that household characteristics also affect the risk of demographic events, some of which are described below. Households in this sense are more than a collection of individuals, and there exists a complex web of interactions among

individuals, their household contexts, and demographic events. Households can be considered a "knot of individual interests" (Laslett 1984), and decisions with consequences for fertility, mortality, migration, and marriage are made within the context of this sometimes collaborative and sometimes competitive group. Households, then, may have emergent properties, making them more than the sum of their parts (Netting and others 1984; Cobb 2017; Anderson and others 1994).

Household Contexts and Demographic Behaviour

The discussion that follows highlights examples of variation in associations among household characteristics and demographic outcomes that sometimes correspond and sometimes conflict with expectations drawn from evolutionary theory. The examples are taken primarily from historical demographic and anthropological studies of the household, which may occasionally reference evolutionary hypotheses but seldom perform systematic tests of evolutionary predictions. Collaboration between evolutionary and household researchers could contribute to the development of new systematic explanatory frameworks to aid in the analysis of the effects of household traits, such as the presence of certain coresident kin or the role of internal power dynamics and trade-offs, on outcomes of interest to both fields including mortality and fertility.

Mortality

Analysis of living standards in the past can provide insight into how household settings affect the wellbeing and decision-making of their members and the demographic consequences of inability to overcome economic stress. A comparative historical demographic study of populations in Europe and Asia conducted by the Eurasia Population and Family History Project (EAP) used time series of staple grain prices and the timing of demographic events to examine living standards in the eighteenth and nineteenth centuries (Bengtsson and others 2004; Tsuya and others 2010; Lundh and Kurosu 2014). In these innovative studies of micro-level demographic data, living standards were assessed using estimates of demographic responses to short-term fluctuations in food prices. This approach proposes that for households with low standard of living, even relatively minor increases in food prices could not be adequately managed with common household-level fall back mechanisms to smooth consumption, such as drawing down savings, selling assets, borrowing, poor relief, delaying purchases and sending out household members as migrants (Thomas and Leatherman 1990). During times of struggle, failure to smooth consumption affected demographic behaviour, as mortality could increase, fertility could be reduced, and marriages could be postponed. These responses may be seen as a way that household members adapted, or failed to adapt, to constraints both internal and external to the household.

In contrast, for households with relatively high standard of living, food price fluctuations did not affect household budgets to the same degree. Consumption could be smoothed such that the timing of births and deaths were unaffected. Thus, demographic responses to economic stress can provide insight into consumption decisions at the household level. This approach can uncover differential resource distribution within households as it identifies which household members were more likely to be allocated scarce resources in times of stress. For example, exposure to poor health is associated with socioeconomic standing and standard of living, but

it can also affect household economic production in its own right. Among poor households, the illness of a primary productive member can reduce production and drive the household into even more dire economic conditions (Leatherman 1996). When household allocation of increasingly limited resources favours the most productive members, usually adult men, others including children are at higher risk of under nutrition (Longhurst 1984). Findings from this line of research are of interest to evolutionary analysts, as decisions regarding household production, allocation, and consumption affect mortality risk in varying ways depending on historical, social, and household contexts.

In their comparative research on mortality patterns and responses to short-term economic stress, the Eurasia Project (EAP), found broad similarities in mortality levels across European and East Asian populations. However, responses to short-term food price variations were more diverse. Socioeconomic differences in mortality responses reflected the institutional and socio-political environments in which households were embedded. There was a stronger mortality response and socioeconomic gradient in mortality in Western communities than in Eastern communities that mirrored higher levels of inequality in landholding in the West and better organized state responses to shortages in the East (Lee and others 2004). In historical European communities, access to land protected children from variability in food prices (Bengtsson and others 2004). Even in relatively poor and remote regions, such as Southern Sweden, food-producing households were able to shelter their members from increases in food prices, as farming households could adapt their consumption and production to ensure that household members were fed before food was sold on the market. These findings have been replicated in other remote European communities, including Northern Scotland (Jennings and others 2017). Socioeconomic stratification in demographic responses to short-term stress accord with relatively straightforward predictions concerning access to resources and mortality risk.

However, the EAP project dug deeper into East-West differences by testing predictions about the role of household structure and composition in demographic responses to short-term stress. Their household models moved beyond concepts of unitary household decision making to consider agency and power structures within households in an approach consistent with bargaining models of the household developed in economics. While the balance of total production capacity and consumption needs of a household are important predictors of resource availability and the associated wellbeing of household members, models that view the household as a single, unified entity over-simplify the internal dynamics of household activities (Wong 1984). Instead of modelling households using a single production, consumption, or utility maximization function or framing household decisions in terms of an authoritarian head or group consensus, alternate approaches address the potentially competing goals of individual household members (Agarwal 1997). Thus, the household bargaining approach directly considers the competing interests of household members and the incentives and disincentives to cooperation (Mattila-Wiro 1999; Behrman 1990).

Households operate within a set of constraints and when essential resources, such as food or money, are insufficient to meet household needs, allocation within the household may have important implications for the survival and reproduction of household members. Even when food is sufficient, household allocation may contribute to elevated risk of malnutrition in some contexts (Messer 1983). Researchers have observed variation in resource allocation within households. Sometimes males are favoured, sometimes females are favoured, sometimes adults

are allocated more resources, and sometimes children are protected to the potential detriment of adults (Graham 1997). Resource allocation is associated with the health and wellbeing of individuals, especially potentially vulnerable household members such as children, elders, and in some societies, women (Das Gupta 1997). Allocation decisions are partly driven by cultural or economic preferences, which may or may not fit with evolutionary or behavioural ecological explanations. Such explanations may help address why households preferentially direct resources to certain members, and whether these differences can be understood in light of evolutionary theory.

In contrast to the socioeconomic gradient in mortality risk in Europe, in East Asian households, relationship to the head and other indicators of power and status within the household were related to mortality risk (Tsuya and Kurosu 2004; Campbell and Lee 2004). In Japanese villages, female infants were more vulnerable than male infants to short-term economic stress (Tsuya and Kurosu 2004). Among adults, women with less powerful positions in the household, such as daughters-in-law and non-stem kin members (those not the spouse, mother, or daughter of the head of household), faced higher mortality risks. Similarly, non-head adult men experienced higher mortality, an indication of the benefits of the authority conferred by household headship. Further, there were negative effects on the mortality of male children in households that co-resided with a grandfather, possibly indicating intra-household competition for resources.

In Chinese communities, the mortality of women and children was sensitive to the configuration of kin within the household (Campbell and Lee 2004). Intra-household allocation processes were associated with these differences in mortality risk (Campbell and Lee 1996). Widows, widowers, and orphans experienced higher chances of death. A woman's mortality was conditioned on whether she had produced an heir, as women with at least one son were at lower risk of mortality than those who had no children or only daughters. Elder males could make strong claims on household resources. In households with grandfathers, children had higher mortality, a finding that reflects how norms about allocation and power relationships may override a strategy drawn from evolutionary theory that would favour the survival of the youngest generation with greater reproductive potential over the eldest, post-reproductive generation.

Standing in the household hierarchy also affects mortality risks in community contexts outside of historical East Asia. For example, in contemporary rural Punjab, women's and children's mortality risk were associated with their relative standing in the household hierarchy (Das Gupta 1995). During times in the life course when women's power and autonomy were lowest, they experienced excess mortality relative to age-matched men. When their position in the household hierarchy improved, women's excess mortality risk was reduced. In this community, women's power and autonomy was lowest after marriage and during their early childbearing years, the period when reproductive potential is high. Indeed, even infant mortality rates reflected status within the household, as infants were more likely to survive if a mother gave birth in her natal household, where she enjoyed higher status, than in her husband's household, where her status was low. The intra-household allocation of authority and resources disadvantaged reproductive women, a group that expectations drawn from evolutionary theory would typically favour for resource allocation.

Fertility

Characteristics of households, such as household size, sibship composition, and the presence of certain types of coresident kin, can affect reproduction, one of the defining household activities. Evolutionary literature has influenced the analysis and interpretation of household data often drawn from historical demographic studies. Within this line of research, hypotheses concerning the relationships among household variables and fertility outcomes reference literature related to the grandmother hypothesis (Alvarez 2000; Hawkes and others 1998; Hawkes 2003) and models of human cooperative breeding (Mace and Sear 2005; Kramer 2010; Sear and Coall 2011) to support expectations about the potential fertility-enhancing role of post-reproductive household members or others who may help reproducing women, such as older children (Turke 1988). Indeed, comparisons can be made between life history theory and household ecology. For example, both approaches suppose a shared or pooled budget, although the units over which budgets are pooled differ, as life history theorists often focus on parent-offspring and alloparent-offspring pooling while household ecologists consider all household members as part of the shared consumption and production budget (Kramer 2018; Kramer and Boone 2002; Kramer and Ellison 2010; Reiches and others 2009; Laslett 1983; Hirth 2009; Hunt 1979; Reyna 1976; Schmink 1984).

While borrowing from evolutionary theories, historical studies have arrived at mixed results concerning household effects on fertility outcomes. Returning to the findings of the EAP group and related historical demographic research, household effects on fertility appear to depend on systems of household and family formation that differ both within and between the East and West (Dong 2016). Socioeconomic status was found to be an important resource for reproduction in historical Europe, while power within household hierarchies determined reproductive decisions in historical East Asia (Lee and others 2010). The kinds of individuals present in the household, especially certain types of coresident kin, were associated with fertility. In the East Asian populations, having a coresident elder female (mother or mother-in-law) in the household increased fertility of women, but only if the older woman did not have an aged husband to care for (Feng and others 2010). The presence of coresident married children lowered the chances of a birth, especially second order and higher male births, an indication that male births were restricted after a patrilineal heir was produced. In households with more than one married couple present, women's relationship to the household head signalled their position within in the household power hierarchy. Household heads and their spouses were more likely to reproduce and began their reproductive careers at younger ages. Women further removed from the household head were less likely to give birth.

Given the low frequency of extended coresident kin observed in historical Northwest European households relative to other regions of the world, such as East Asia, researchers have begun to expand beyond strict definitions of household co-residence to identify non-co-resident kin living in close geographic proximity and assess whether these relatives affected fertility outcomes. However, the effects of coresident kin in European and North American contexts may be mixed, especially in the case of post-reproductive adults. If small, nuclear family households were the norm, then the presence of the older generation would strain household budgets if ill, frail, or impoverished elders were taken into households unaccustomed to accommodating more complex kin arrangements (Hareven 1994; 1996; Kertzer 1995; Laslett 1988). However, a three-generation household would be an indicator of economic security, rather than strain, if it

formed when the elder generation retired and passed a farm or business to an heir or if three-generation co-residence was more commonly practiced among the wealthier classes (Ruggles 2009; Manfredini and Breschi 2013; Ruggles 2003; Alter 1996).

Given the opposing scenarios in which three-generation co-residence could occur historical European and North American communities, the effects of coresident kin may not easily align with simplistic predictions drawn from evolutionary theory, including cooperative breeding hypotheses. For example, in the historical United States, fertility was reduced by kin co-residence, especially in 3-generation households, possibly the result of overcrowding small dwelling spaces or poor health of the elder generation (Hacker and Roberts 2017), a contrast to the patterns described for East Asia. However, results were mixed concerning the fertility effects of spatially proximal kin, who would be nearby and could offer assistance yet were not subject to some of the unfavourable conditions associated with co-residence. Sometimes fertility was promoted, sometimes it was reduced, sometimes there was no clear effect, and differences were observed between different kinds of nearby kin, including paternal and maternal grandparents and siblings (Jennings and others 2012; Hacker and Roberts 2017; Willführ and others 2018). Some mixed results are found even in careful comparative analyses that construct similar measures and compare the effects of the same types of kin (for an example, see Dillon and others this volume). The integration of hypotheses drawn from evolutionary theory and social and historical analysis has the potential to contribute to the development of an analytical framework to address household effects on fertility that applies across a broader range of societies, enhances comparative research, and aids in the contextualization of these mixed findings. Bridging the household literature on competition and cooperation with the evolutionary literature on cooperative breeding and the smaller evolutionary literature on resource constraints and competition associated with fertility outcomes (Strassmann 2011; Mace and Alvergne 2012; Mace 2013; Schaffnit and Sear 2014) could be a fruitful avenue for collaborative research.

In addition to the fertility effects of cultural norms and expectations, such as differences in status afforded to age and sex groups within households described in East Asia, a household member's access to economic resources affects their bargaining power, which influences fertility behaviour. In Tanzania, birth intervals became shorter and fertility increased when women's power within the household changed (Lockwood 1998). With economic change, women's earnings, based primarily on rice production, were curtailed. When Islam began to displace traditional religious practices, gender norms shifted, and traditional postpartum sex taboos were not strictly enforced. Instead, women felt pressure to resume sexual relations sooner after a birth to shore up their relationship with their spouse, upon whom they were increasingly dependent. Thus, women's changing bargaining capacity within the household was associated with shorter birth intervals and higher fertility rates.

Fertility behaviour has been observed to respond differently to resource scarcity in varying household contexts. Fricke (1986) described household strategies among the Tamang of Himalayan Nepal. The Tamang practiced a diverse economy in an environment of resource scarcity, which included agriculture in marginal upland plots. Exchange and reciprocity among neighbours and kin groups were essential for the functioning of the household economy. Given diverse economic activities and the importance of extended kin networks, there was an incentive for high fertility. High fertility not only offset high infant mortality, but more children helped diversify the household economy and expand and reinforce local kinship networks

that households relied upon for exchange in times of need. In contrast, in historical Hungary, complex household formation and fertility control within households have been described as a strategy to address land scarcity by reducing the number of heirs and limiting the division of land (Andorka and Farago 1983). Similarly, in the land-limited and demographically saturated Krummhörn region of Germany, low fertility and high age at marriage limited family sizes within households (Willführ and Störmer 2015). Transmission practices, in this case impartible inheritance, also contributed to low fertility. Marriage and inheritance were essential decisions that balanced the demand for children and the needs of the youngest generation against the considerations of household alliances in Nepal or household landholding size in Germany and Hungary.

Intra-household Processes: Conflicts and Trade-offs Between Individual and Group Interests

Life history theory is concerned with how evolutionary forces shape responses to the trade-offs faced by organisms (Stearns 1992; Kuzawa and Bragg 2012; Hill 1993). Evolutionary researchers focus on trade-offs at the individual level, such as the allocation of resources to potentially competing processes, like growth, reproduction, and somatic maintenance. Households also face trade-offs, as their members must decide how to allocate limited resources within the household and must prioritize some household activities or members over others in times of scarcity. Indeed, reproductive ecologists conceptualize a shared energy budget at the level of the family or breeding community that is driven by cooperative breeding (Kramer and Ellison 2010; Reiches and others 2009), but a similar argument might be made for the shared budget at the level of the household. The decisions household members make when faced with trade-offs shape the characteristic household activities (production, distribution, transmission, reproduction, and co-residence) as discussed above with respect to household effects on fertility and mortality.

In addition to facing trade-offs, household members also negotiate potential conflict between individual and group interests. These conflicts can lead to household division and disagreements about the transmission of property and resources. Conflicts can also contribute to behaviours and decisions that may disadvantage some household members and privilege others. Given these fields' common interest in trade-offs, the development of joint household and evolutionary hypotheses about the internal dynamics of these important social units can advance our understanding of which household members are likely to benefit or suffer if risks and resources are distributed unevenly within the household and under what conditions unequal distribution occurs.

Household structure and composition affect household activities and vice versa (Netting and others 1984). For example, household economic productive capacity and consumption requirements are determined by the age and sex composition of household members. All other things being equal, a household with more net consumers relative to net producers should face greater intra-household economic pressure. The balance of workers to consumers changes as children are born into a household and grow up (Chayanov 1986; Hunt 1979). Thus, the internal "life cycle" of a household contributes to both consumption requirements and productive capacity. Household composition can be considered a determinant of intra-household competition for resources, especially in households with young children or elders.

In nineteenth century Orkney, Scotland, children were at higher risk of mortality in households with unfavourable ratios of consumers to producers and when twins were born (Sparks and others 2013). A similar pattern is found in contemporary Laos, where children in households with higher ratios of consumers relative to producers experienced higher odds of mortality, even after controlling for other household variables and unobserved inter-household heterogeneity (Tomita and others 2015).

Inheritance, retirement, and household division are times of potential conflict that highlight some of the tensions between individual and group interests. In societies where property is transferred between the generations, inheritance practices shape the transmission of resources. Inheritance is often a critical event in the household cycle and it is a time when the needs of the collective can conflict with the needs of the individual (Sieder and Mitterauer 1983). Household inheritance systems take two general forms, partible inheritance, in which multiple heirs have a claim, and impartible inheritance, in which only one person has a claim. Inheritance practices are a source of parent-offspring conflict in humans, especially when inheritance is impartible and one child is favoured over its siblings (Salmon 2008; Trivers 1974). Parents must balance offspring provisioning with maintaining a viable household economic enterprise, especially in contexts with limited resources, such as access to productive farmland. Inheritance practices can thus be viewed along a continuum of offspring provisioning, from equal provisioning of all offspring (some forms of partible inheritance) to directing all resources toward one offspring (impartible inheritance). Households do appear to use inheritance practices strategically and will, if possible, adjust household composition or manipulate social rules to adapt to either the underproduction or overproduction of heirs and ensure the continuation of the household (Goody 1976).

There are economic and ecological reasons to favour one heir over multiple heirs. If division of property is costly, impartible inheritance is often practiced. This is often the case for aristocratic families and farmers in contexts where smaller plots are ecologically or economically untenable or division is not allowed by the landowning class. Indeed, for farming households, it is possible to consider inheritance practices and access to marriage as part of a land-based breeding system where the combination of broader ecological conditions and the individual household's access to resources result in different strategies of inheritance (Hrdy and Judge 1993).

Inheritance, land, and household size and composition are closely related. If an economic niche is a prerequisite for marriage, then heirs may marry only after they inherit and non-heirs must remain unmarried or out-migrate (Engelen and Wolf 2005). If no such economic requirement for marriage exists, marriage will become more accessible. Population growth may be affected by these interrelated systems. In early-modern Saxony, population growth was higher and households were smaller in regions with partible inheritance, while larger three-generation households and slower population growth were associated with impartible inheritance (Berkner 1977). Impartible inheritance in this setting restricted population growth by stopping the fragmentation of land holdings, preventing the formation of new households, and encouraging the outmigration or celibacy of non-heirs.

The timing of inheritance often coincides with the retirement of the household head. In societies that lack access to financial institutions to save money for retirement or other insurance plans, the decision to step down from household leadership means an uncertain future for elders. In historical Europe, it was common for retiring household heads to insist

upon drawing up a contract with their heirs that specified precise levels of support as they aged (Gaunt 1983). Legal scholars at the time complained of the conflict between parents and their children. Heirs wished to inherit early, especially if marriage was conditioned on inheritance. However, parents were reluctant to give up their position and authority as household head and rely upon their children for their upkeep. Retirement contracts reflected the potential for, or at least fear of, the neglect of aging parents.

In complex household systems, such as those of joint-family households where multiple married couples and their families co-reside, household division was an additional source of intra-household conflict. Large households have certain economic advantages, as the presence of additional household members allows for the diversification of economic activities and hedges against risk (Lockwood 1998; Cohen 1976). Households that contain more than one married couple can reduce the magnitude of unfavourable fluctuations in the ratio of consumers to workers and benefit from economies of scale (Hammel 2005; 1972). Apparently aware of the economic benefits of large households, landlords often attempted to encourage large households and prevent their fission. In historical Russia, landlords were invested in the stability of farm production and prevented the division of households whenever possible (Czap 1983). In Poland, the interest of manorial estates in maintaining large households was often in conflict with peasant households that wished to divide (Kochanowicz 1983).

An analysis of the forces that unify and divide large complex households in Taiwan by Cohen (1976) provides a detailed account of the sources of tension within households and conflict between individual and group interests. Taiwanese households were complex, joint family households, where married adult sons remained in the household with their wives and children. The household head was responsible for the allocation of household resources and claimed the income or labour of any coresident children and redistributed it to household members. Children were entitled to the resources required for a proper and timely marriage and married sons could demand a portion of household income or a household division. Once children reached marriageable age, tensions arose between the generations in the household. The household head was primarily interested in the survival of the household estate, while married sons were motivated to claim as much of the household resources as possible to provision their spouses and offspring. For the older generation, a further disincentive to household fission was the position of the household head and spouse after division. The former head and his wife would become the dependents of one of the resulting smaller households, which would be a loss of status and power for the older couple.

The benefits that accrued to large households also led to the risk of household division. Cohen (1976) documented how brothers competed and argued over household partition. Their wives took sides in the arguments, especially if they felt that the intra-household allocation of resources was unequal or unfair. This was common when one married couple had more dependent children and therefore received a larger portion of household resources. The couple with fewer children, in effect subsidized the fertility of the couple with more children through the logic of income pooling and redistribution. Household fission became imminent when sharing and pooling of resources failed or if there was outright embezzlement of household funds. Cohen argued that household unity could only prevail if it was in the economic interest of all parties. However, not all sub-units of the household would benefit equally from household partition, as some received greater benefits from the efficiency of the larger

work group than others. The process of household division could disadvantage some former household members, especially if the benefits of large households were lost after partition. Interestingly, household economic diversification appeared to reduce the chances of household division, as the potential gains related to division of labour and task specialization were greater. It is possible that household and evolutionary approaches together can provide a framework to understand the dynamics of household conflict and fission.

Discussion

Households are the social unit home to intersecting activities that shape demographic behaviour, often some combination of production, distribution, transmission, reproduction, and co-residence. As such, they may overlap with family and kin groups, but they are not equivalent entities as households are distinct from families and feature different internal dynamics and pressures. Cultural practices and social institutions influence household effects on outcomes of interest to an interdisciplinary set of researchers, including survival and reproduction, as they are a factor in determining who is present in a household, the relative frequencies of types of household activities, and the structure of internal power dynamics and bargaining. Indeed, it has been argued that "households are the level at which social groups articulate directly with economic and ecological processes (Wilk and Rathje 1982: 618)." Given the essential functions and role of households in shaping individual behaviour and social contexts, it is time for closer collaboration among household and evolutionary analysts.

While there is a well-established body of work at the intersection of human evolution and kinship or family (Emlen 1995; Davis and Daly 1997; Salmon and Shackelford 2008) and evolutionary demographers frequently examine kin effects on behaviour, fewer evolutionary studies explicitly examine the effects of household composition, structure, or dynamics on behaviour. The challenges in household definition and measurement described above may contribute to this lack of integration, but there is potential for fruitful work at the intersection of human evolution and household analysis. I suggest that the rich understanding of household activities, morphology, and change that come from historical, anthropological, and demographic research can be usefully combined with theories and hypotheses from evolutionary analysis to understand behaviour. Together, these perspectives can enhance our knowledge of the ways that households affect life chances and complement evolutionary analyses of kin and family effects on demographic outcomes. Further, coordination among household and evolutionary researchers can advance the analysis and interpretation of rich historical and contemporary household data sources, development of theories and hypotheses that incorporate household variation and change within and between societies, and establishment of new collaborative research in the social and evolutionary determinants of demographic behaviour.

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26. Historical Family Reconstitution Databases in the Study of Kinship Influences on Demographic Outcomes

Kai P. Willführ, Jonathan F. Fox and Eckart Voland

Human life histories and demographic outcomes are impacted by kin behaviour in diverse ways, and human evolutionary theory is essential to understanding how environmental context and kin relationship moderate this behaviour in cooperative versus competitive directions. However, kin presence is simultaneously correlated with behavioural and non-behavioural factors such as risk of infection or familial wealth. As such, it can be hard to disentangle evolutionary effects from other factors correlated within a family. In this chapter we discuss how historical family reconstitution databases have assisted in the investigation of kin effects and their advantages in disentangling these behavioural kin effects from non-behavioural accompanying factors. A variety of family reconstitution studies exist across varying geographic and cultural contexts, and their application to kinship research has resulted in varying findings. This highlights how kin impacts differ depending on circumstance. We describe several family reconstitution studies and discuss the importance of evolutionary theory in understanding kin effects across different populations.

Introduction

Family matters for human life histories. Family is important in an active and operative sense, as social interactions between kin members can affect those members' outcomes. And family is important as a group identifier, marking groups where correlations in epidemiological conditions, socioeconomic status, genetic background, environmental and social conditions, and other factors may be common amongst their members. Kin, as the members of these groups, can affect outcomes both by their absence and their presence. For example, the absence of the biological father during early childhood may impact later reproductive behaviour and strategy (Shenk et al. 2013; Sheppard, Snopkowski, and Sear 2014), although this effect seems limited to Western, Educated, Industrialized, Rich, and Democratic (WEIRD) societies (Sear, Sheppard, and Coall, 2019). And the presence of additional siblings appears to have a direct effect on survival and fertility (e.g., Steelman et al. 2002; Fox et al. 2017). In addition, the presence of postmenopausal women tends to reduce the mortality of their reproductive daughters and those daughters' children (Sear and Mace 2008). For daughters-in-law, however, the impacts may rely on social strata differentials. Studies covering families at the lower end of the economic spectrum suggest

 https://doi.org/10.11647/OBP.0251.26

mothers-in-law increase stillbirth mortality (Voland and Beise 2005), whereas other studies indicate that in more affluent families they reduce the mortality of their sons' wife (Willführ, Johow, and Voland 2018). A study by Zang and Campbell (2018) suggests grandparental presence might also affect mortality later in life. They found that men in north-western China suffered from increased mortality if they resided as children with their father's parents. Depending on their relationship and other moderating factors, the impact of kin's presence or absence may constitute either a positive or a negative influence, and sometimes simultaneously both.

Darwinian or evolutionary theory helps to understand the variation in kin effects across kin types (e.g., paternal versus maternal grandmothers) and environments (e.g., resource constrained versus resource abundant). Evolutionary theory argues that evolved strategies and mechanisms interact with the contemporary environment and cause or determine the path of the individual phenotype over its life course. As we consider them, kinship effects constitute the impacts on individual outcomes arising from social interactions among kin. Social interactions are broadly defined to include any variety of active supportive or competitive behaviour.

Evolutionary theory provides a framework to understand when and why this supportive or competitive behaviour may arise. The supportive role of kin can be understood within the context of the cooperative-breeders model, where alloparental care is provided in support of the mother both to improve the outcomes of the offspring and allow for increased fertility from the mother. On the other hand, family resources per household member decline as the number of kin increase if these kin consume more family resources than they contribute. Resources are thus often scarcer in large families, with increased kin competition.

The role of grandmothers can also be understood using evolutionary theory. With supportive grandmothers, reproductive females exhibit increased fertility and lower offspring mortality, an effect attributable to grandmaternal support in childcare and knowledge transfer (Lahdenperä et al. 2004; Voland, Chasiotis, and Schiefenhövel 2005).

The kinship effects described above derive from social interaction and conscious or unconscious efforts to affect the outcomes of other family members. However, the presence of kin can also be associated with accompanying non-behavioural factors. This can create challenges in differentiating between the behavioural and non-behavioural impacts, and in identifying the role of evolutionary influences on kin interactions. The effects of sibling composition, for example, are often associated with both behavioural and non-behavioural influences. The impact of sibling composition on outcomes such as child mortality may result from behavioural factors such as sibling rivalry and competition, as well as from increased risk of cross infections associated with larger families or from improved family outcomes driven by the role of the familial unit in obtaining economic success (e.g., pioneer populations).

Separating behavioural from non-behavioural kinship influences on outcomes is possible if those non-behavioural accompanying factors are common to members within a family. This may not be true in all contexts but is a reasonable assumption for factors such as the risk of cross infections and socioeconomic status during childhood. When variation in unobserved factors occurs across but not within families, it is possible for researchers to exploit the role of family to identify kinship influences deriving from evolutionary factors. This requires information on

families observed over complete life courses, and, in some cases, across subsequent generations. Historical family reconstitution studies, with their provision of long-period information, are thus extremely valuable in the study of kinship from an evolutionary perspective. This chapter serves as both an introduction to historical family reconstitutions and their origins, as well as an argument of their importance in identifying the significance of evolutionarily driven behaviour in kinship interactions. We begin with an overview of the origins of historical family reconstitutions.

Origins of Family Reconstitution Studies

Although the pioneering work of family reconstitution studies is usually identified as that by Fleury and Henry in 1976, the history of family reconstitution studies starts with parochial studies of German population biologists from the pre-World-War-II period (e.g., Scheidt 1932). The context within which these scientists researched, and perceived connection to the racial ideas of the Nazis, has led to their works being ideologically tainted (Schlumbohm 2018). The procedure used by those early researchers, still used in contemporary studies, begins with the digitization of all marriages within one or more localities and recording the names of grooms and brides and their date of marriage in a database. Birth records that include the name of the parents along with the name and birthdate of the child are digitized in another database. The families are then reconstituted by linking the databases. The final step is to append parent and child dates of death from the available burial records. Fleury and Henry used these parish register data to reconstruct population levels and trends. These techniques were then further developed by E. A. Wrigley, R. S. Davies, Jim E. Oeppen, and Roger S. Schofield at the Cambridge Group for the History of Population and Social Structure (Wrigley and Schofield 1981; Wrigley et al. 1997).

From the beginning it was clear that using such family reconstitutions to estimate population levels and trends faced serious limitations. These included migration censoring (1), non-representativeness of the study parish/es (2), and record selection bias (3).

The problem of migration censoring is best illustrated with an example. Within Europe, many children left their parent's parish of residence as young adults, resulting in many unknown marriage and death dates. Since migrants tend to marry later, the observed mean age at marriage will be lower than the "true" mean age of marriage because later marriages among migrants are excluded (Kasakoff and Adams 1995). Although the impact of this may not be large, the problem persists in principle. Voland and Dunbar (Voland and Dunbar 1997) used a case study to show that the underestimation of age of marriage is small and is likely to be negligible for most research questions.

The non-representativeness of parishes or localities (2) and record bias (3) constitute source problems as demographic traits exhibit spatial dimensions. Some parishes are wealthier than others, some are under increased epidemiological stress due to high population density and/or trading traffic, and others suffer regularly from natural catastrophes like floods and droughts. The representativeness of the study parishes is therefore of enormous importance in establishing generality. Similar concerns exist regarding systematic bias in individual records within the parish registers. This bias is often present in historical parish registers, which prioritized socially important individuals and families. As such, historical family reconstitution studies must be carefully applied in estimating population

level statistics. However, they are of great value for studies focusing on the individual and family level, which can in turn help to understand trends at the population level (Dribe et al. 2016).

Like historical family reconstitutions, longitudinal studies of contemporary populations also provide long-period information. These include studies such as the 1958 National Child Development Study and British Cohort Study of 1970,[1] the Dutch Famine Birth Cohort Study,[2] The German SOEP dataset,[3] and the U.S. National Longitudinal Surveys.[4] These studies often combine demographic traits with anthropometric, medical, and socio-economic information. However, unless a subject expires between survey waves, they generally do not cover completed life histories. Completed life histories and those that link several subsequent generations are generally provided only by register-based family reconstitution studies. We survey five of these reconstitution studies below.

An Incomplete List of Family Reconstitution Studies in Use

In the following we briefly introduce five different datasets from Europe and North America. These datasets exhibit different advantages and are linkable to information from other sources. Our selection is to be understood as an introduction and makes no claims of completeness.

Many more datasets are carried together by the European Historical Population Sample Network (EHPS network) and are accessible online.[5]

Canada (Quebec, St Lawrence Valley)

The Programme de recherche en démographie historique (PRDH, Research Programme in Historical Demography) at the Université de Montréal reconstitutes the population of Quebec from the beginnings of French colonization in the seventeenth century. Data collection and processing is still underway; currently, data from the early beginning of the colony up to the year 1849 is available for statistical analysis. The dataset covers the complete population over the whole territory on which the colony was established at the time, mitigating selection bias and other problems of observation exits through emigration. The population was naturally fertile and experienced exponential population growth after the final migration wave in 1671 due to settler births (Charbonneau, Desjardins, and Légaré 2000). An updated report on the PRDH was published in 2018[6] by Dillon and colleagues (Dillon et al. 2018). The database was established to investigate the population history of New France including epidemiological, genetic, and many other research questions. Various studies focusing explicitly on kin effects include those studying parental loss (Pavard et al. 2005; Willführ and Gagnon 2013; Légaré and Naud 2001), sibling effects on marriage (Caron et al. 2017; Dillon 2010), and sibling effects on mortality (Fox et al. 2017).

1 http://www.cls.ioe.ac.uk/
2 http://www.dutchfamine.nl/index.htm
3 http://www.diw.de/de/soep
4 https://www.bls.gov/nls/
5 https://ehps-net.eu/databases
6 https://www.prdh-igd.com/en/updates

Germany (Krummhörn), 1720–1874

This family reconstitution project originated to evaluate hypotheses on human reproductive behaviour guided by sociobiology and behavioural ecology using the population of East Frisian Krummhörn, the marsh region northwest of the city of Emden, from eighteenth- and nineteenth-century parish records and tax lists. This database contains vital and social statistics for 118,778 persons (34,708 families) from thirty-three neighbouring parishes. Originally supported by the German Research Foundation (DFG), the German-language database is archived at the GESIS-Institute (Cologne) with the label ZA8630.[7] A comprehensive description of the database can be found at the study founder's website: http://eckart-voland.de/pdf/KH-LIT.pdf. Currently, there are sixty-seven scientific publications based on this database. A list of publications derived from this project can also be found at the study founder's website: https://eckart-voland.de/pdf/KH-LIT.pdf. Most of these publications are on kin effects, including studies on differential parental investment (Voland and Dunbar 1995), differentials in reproductive success (Voland 1990), the opposite effects of maternal and paternal grandmothers (Voland and Beise 2002), in-law kin effects (Voland and Beise 2005), the effects of having siblings (Fox et al. 2017; Beise and Voland 2008), the impact of paternal death on life strategies (Voland and Willführ 2017) and on the impacts of kin on the mortality of reproductive women (Willführ, Johow, and Voland 2018).

The Demographic Database (Sweden)

The Demographic Data Base (DDB) is a research unit at Umeå University, Sweden which provides comprehensive information about the Swedish population history for research, education, and archives.[8] The DDB consists of several datasets which provide different levels of information on the Swedish demographic history. Data for the POPUM dataset derived from catechetical examination registers (*husförhörslängder*) which cover vital events and marriages as well as detailed information about the individual residence since parishioners had to inform the minister about their in- and out-migration. In nineteenth- and twentieth-century Sweden there was substantial spatial variation in living conditions and socio-economic standards. Regions such as the Sundsvall region were industrialized, whereas other regions remained rural and agricultural. Studies on kinship using the DDB include those estimating the impact of consanguine marriages on reproductive behaviour and early mortality (Egerbladh and Bittles 2008), on family effects on social mobility (Miller 1995), and on the consequence of paternal loss (Wall 2002).

United States (Utah Population Database) from Early-Nineteenth to Mid-Twentieth Centuries

The Utah Population Database (UPDB) at Huntsman Cancer Institute at the University of Utah links historical family demographic records with the biomedical data of the descendants living today. The dataset includes more than 1.6 million individuals and has been an important resource for researchers in both the medical and social science fields. In addition, it has the

7 http://dx.doi.org/10.4232/1.12643

8 https://www.umu.se/en/centre-for-demographic-and-ageing-research/databases/

unique distinction of containing information on a Western polygamous society, as this was allowed within the Mormon religion for a short period in the nineteenth century. This social system allows researchers to disentangle paternal and maternal effects which are more difficult to separate in (serial) monogamous societies.

More generally, this dataset has proved a vast resource to research studying the genetic component of cancer risk. Studies using the genealogical data to investigate familial links in the incidence of breast and prostate cancer (Hunt et. al 1980 and Cannon et. al 1982, respectively) were among the first academic articles to apply the Utah Population Database towards research questions outside of its specific historical context. Articles related to cancer research continue to comprise most of the published research using the UPDB. Articles related to social science questions have a relatively younger history using the UPDB, beginning with studies of longevity and ageing. The first of these looked at the transmission of longevity through families, finding that excess longevity aggregates in families and arguing the role of genetics in driving these effects (Kerber et. al 2001). Since then, researchers have used the UPDB to look at questions related to partner loss (Mineau G, Smith K and Bean L, 2002), offspring impacts on paternal mortality (Harrell CJ, Smith KR, and Mineau GP, 2008), Grandmother effects (Hawkes and Smith 2009), and parental loss (Smith KR, Hanson HA, Norton MC, Hollingshaus, Mineau GP, 2014; Hollingshaus M, Smith KR. 2016; and Hollingshaus, M. S., Coon, H., Crowell, S. E., Gray, D. D., Hanson, H. A., Pimentel, R., & Smith, K. R. 2016).

Rural Finland, Eighteenth to Twentieth Centuries

Like the Krummhörn database, this dataset derived from protestant parish registers and was initially compiled to study human reproductive behaviour from a biological perspective. The projected is overseen by the Human Life-History Group, an academic research group based at the University of Turku, Finland.[9]

Currently, the data consists of eight separate Finnish populations which differ regarding co-residence and marriage systems. Demographic data are augmented by individual information on socio-economic status and occupation, as well as by structural information on climate, conflicts, epidemics, famines, and annual crop yields. The database has been used in several studies on kin effects, including the impact of having elder siblings (Nitsch, Faurie, and Lummaa 2013; Rickard, Lummaa, and Russell 2008) and grandparents (Chapman et al. 2021), costs of male offspring production (Helle and Lummaa 2013), and the trade-off between reproduction and survival (Nenko et al. 2018).

The Importance of Historical Family Reconstitution Studies in the Study of Kinship

The primary purpose of family reconstitution methods has been to estimate mortality, fertility, and marriage patterns in historical populations. Such classical historical approaches have provided information on historical living situations and standards, debunking common misconceptions. For example, the popular idea that members of historical European

9 http://human-life-history.science/datasets. Last accessed March 18th, 2021).

populations married young and produced many children has been relatively widespread. However, individuals in pre-industrialized Europe usually married in their late twenties with an average family size generally less than five (Western European marriage pattern (Hajnal 1965)).

Family reconstitution datasets have also proved immensely valuable to other social and natural scientists to researching questions related to kinship. Observing multiple generations of the same families through time allows for the study of intergenerational transmission and intergenerational accumulation or de-accumulation of effects such as the heritability of fertility and longevity (Pettay et al. 2005). Furthermore, in contrast to traditional individual-level survey datasets which tend to indicate only the number of siblings at the time of the survey, family reconstitution datasets provide information on outcomes for all or most members of the family surveyed. This information is vital if the status of the siblings or their own outcomes have a bearing on the dynamics of kinship effects. Dillon (2010), for example, finds the likelihood of marriage increased if the subject had married older siblings, and decreased with the presence of unmarried older and marriage younger siblings. For this population, the marital status of siblings seems to have been an important moderating effect in sibling interactions.

Historical family reconstitution studies can also complement information compiled at the individual, local, or regional level, and used to create a hierarchical dataset through time. Reconstitution studies have been combined with tax rolls and food prices (economic variables; e.g., Bengtsson 1993), climate and weather data (environmental variables; e.g., Willführ and Störmer 2015), medical records and reports on epidemics (epidemiological variables; e.g., Gagnon and Mazan 2009; Quaranta 2014; Tommy Bengtsson and Lindström 2003)), periods of war and social change (political variables; e.g., Kemkes 2006), and geographic information (Pantazatou et al. 2016). For some family reconstitutions it is possible to estimate the individual social-economic-status (SES) through information on occupation (see HISCO Historical Classification of Occupations (van Leeuwen, Maas, and Miles 2002)) or landownership (Bengtsson and Dribe 2011). Furthermore, the fertility and mortality response to economic fluctuations of the different SES groups can be studied if information on food and crop prices is available (Bengtsson and Dribe 2006; Willführ and Störmer 2015; Amialchuk and Dimitrova 2012). For reconstitution studies in which place(s) of residence is available, it is possible to compare kinship effects and outcomes across space. This can be of significant value, as mortality, especially child mortality, differed substantially between urban and rural areas. Increased pathogen risk due to higher population density, unhygienic conditions, and pollution associated with the beginning of industrialization all contributed to an urban mortality penalty. Additionally, trading by surface and sea regularly introduced infectious disease from other parts of the world. We note that in the contemporary developing world, the situation is the opposite. Urban residence is associated with access to better medical care and higher SES.

Observed and Unobserved Heterogeneity in Moderating Kinship Effects

The presence of kin is often correlated with other factors that impact outcomes of researcher interest, the effects of which may be difficult to disentangle. These factors may be either observed or unobserved, and can play a moderating role, thereby improving estimates and understanding of kinship effects, as well as create challenges to their identification. Historical family reconstitutions, with their focus on complete families observed over long periods of time, present unique opportunities in the identification of kinship effects, as well as those factors which may moderate their expression.

Historical family reconstitutions often combine individual vital data, such as date of birth, death, and marriage, with anthropometric, medical, socio-economic, or other statistics, and so provide information on individual, group, and environmental factors potentially correlated with kinship-related outcomes. Consideration of these observed statistics can show patterns of kinship effects across groups and how individual, group, and environmental factors can moderate kinship impacts. These observed effects provide information on how kinship effects may vary across occupation, geography, demographics, or other factors. The consequences of parental loss and parental remarriage (Willführ and Gagnon, 2012; Willführ and Gagnon, 2013), for example, varies in different geographic and historical groups. Using information on place of residence, it is possible to identify context-specific patterns of kin effects. This can be important when families migrate. Migration and kinship, especially as it related to siblings, are often interrelated processes as families delay childbearing until arrival in the destination (Andersson 2004, Wilson 2013). If migration is systematically associated with better or worse outcomes, then efforts to identify kinship effects would be confounded. This is an issue when families migrate from urban to rural areas or vice versa, who are immediately confronted with a different mortality regime and systematic differences related to disease risk. In addition, migration during pregnancy could result in a phenotype-environment mismatch, resulting in negative effects for infants born in the new environment (Willführ and Myrskylä 2013; Kuzawa 2005). Intra-rural and intra-urban migration can also result in notable environmental differentials. In the St. Lawrence Valley for example, although both the Northern and Southern shore areas are considered rural, there exist substantial differences in climate and farming conditions. These differences were meaningful enough to cause substantial differences in maternal and intrauterine condition (Gagnon 2012).

In addition to the effects of geographic variation, information on families over time can also help identify how kin effects may vary across different genetic groups. Biometric measurements (including genetic characteristics) and health conditions of individuals living in contemporary societies can be linked to information on their ancestors' living conditions and experiences to identify potential founder effects (Gagnon and Heyer 2001) or epigenetic effects (Bygren, Kaati, and Edvinsson 2001; Bygren et al. 2014).

Family reconstitutions, as well as other longitudinal datasets, can be used to control for unobserved effects correlated with outcomes and patterns of kinship. Unobserved effects may be jointly associated with the presence or absence of kin and individual outcomes and complicate efforts to identify kin effects. Since longitudinal datasets observe individuals over multiple time periods, they can be used to control for individual-level unobserved

effects through a fixed-effects modelling framework. Fixed-effects models, as referred to here, are models that allow for different intercepts (or the analogous concept for non-linear models) across individuals. Such techniques control for all individual-level factors constant through time, so rely on variable changes to identify factors of interest. The effect of sibling composition, for example, is then identified off changes in sibling composition through births or deaths. Given that many moderating factors related to kinship effects and of researcher interest are time-invariant (i.e. gender, race, maternal or paternal age at birth), individual-level fixed effects models are often impractical. This has resulted in a wide variety of kinship studies applying random effects models, sometimes referred to as hierarchical models or mixed models. Random effects methods parameterize the individual-specific effects as originating from the same overall distribution, and so can allow for the inclusion of time- invariant variables such as parental age at birth. If these individual specific effects are not correlated with any observed variables related to kinship composition, then random effects methods offer a method of estimating time-invariant kinship effects.[10] However, this is a critical assumption in non-experimental settings, and if not met, random effects methods produce biased estimates of kinship effects. We should mention that hybrid models utilizing fixed effects for those variables correlated with the unobserved effects and random effects for those which are not, are an alternative framework in which it is both possible to include time-invariant factors and obtain unbiased estimates of kinship effects (Laird and Ware 1982, Goldstein 1986, StataCorp 2013). These models require careful, and sometimes arbitrary, judgement regarding which variables are not correlated with the unobserved effects.

Family reconstitution datasets offer an additional advantage over the typical longitudinal datasets that track individuals. Many unobserved individual effects related to kinship occur at the family level, and so controlling for unobserved family effects facilitates identification of kinship composition on outcomes. Exposure to the disease environment, genetic effects, ecological fitness, parenting techniques, and socioeconomic status are all unobserved effects correlated with kinship and kinship-related outcomes, and plausibly common within families.

The same fixed and random effects methods applicable to individuals can be applied to families. Family-level random effects methods, often referred to as shared frailty methods, allow for correlation across individuals in the same family and so produce more efficient estimates of kin impacts. As such, tests of significance for estimates of kinship impacts have greater statistical power. Rickard et al. (Rickard, Lummaa, and Russell 2008), for example, include a random term for a common mother in their linear mixed effects models to show that older brothers tend to reduce the probability of reproduction for their younger siblings in preindustrial Finnish farming and fishing communities. Suanet and Bras (Suanet and Bras 2014) apply this method using Cox proportional hazard models to determine that sibling position became less important for marriage timing within Dutch provinces between 1840 and 1922.

10 Researchers deciding between the application of fixed or random effects methods should produce some statistical test of the consistency of the random effects estimates. The best known of these is the Hausman Test (Hausman 1978)

Non-linear shared frailty models, such as the Cox proportional hazard framework with family-level random effects, can often be computationally intensive for large populations. As such, researchers must balance the gains from statistical power with the computational intensity of these shared frailty methods.

As described in the discussion of individual unobserved effects, random effects methods such as shared frailty models parameterize the unobserved effects and, as such, require they are not correlated with the observed variables related to kinship. If this assumption is not credible, fixed effects methods are necessary to identify the effect of the kinship presence. Families should then be allowed their own intercept terms and kin effects are identified through comparison of kin within the same family (typically the same household or common parent). Families thus require at least two members, but the role of individual time-invariant factors such as gender and parental age-at-birth can be estimated.

Unobserved effects can obscure our ability to observe kin effects, but by controlling for the effect of the family and the unobserved effects associated with it, one is able to tease out a more precise role of kinship effects. This can help researchers both better identify the presence of kinship effects, as well as separate behavioural effects from those accompanying non-behavioural factors. As an example of the former, selection effects have tended to complicate the ability to identify the relationship between longevity and fertility. Theoretically, maternal depletion increases with the number of childbirths, however empirical estimates from proportional models suggest that many childbirths are associated with increased post-menopausal survival and longevity (Gagnon et al. 2009). The reason for this positive association, often described as phenotypic correlation, is that the less vulnerable and more robust females live long and produce many babies, even though they might live longer had they reproduced less. For instance, child mortality and morbidity tend to be elevated among families with many children in historical populations (Edvinsson and Janssens 2012).

Sibling rivalry and competition constitute social interactions which may contribute to elevated mortality in these larger families, while sibling cooperation may tend to reduce child mortality. Determining the relative strength of these two behavioural elements can be challenging, as family size is correlated with other risk factors. For example, the risk of cross infections increases with the number of siblings, especially older siblings. Illnesses lead to increased mortality during childhood, as well as elevated frailty through adulthood. As such, being part of a larger family may be associated with reduced fitness in adulthood due to increased risk of sickness rather than any active role of the different siblings (Barclay and Myrskylä 2014). Siblings must be present in the household to pose an infectious risk, but social interactions in the manner of competition and cooperation is not needed. Cox proportional models stratified at the family level control for family-level fixed effects through a likelihood function that includes separate terms for each of the different families in the dataset. As such, each family is allowed their own individual baseline hazard function and family-specific frailty is controlled for.

Evolutionary Versus the "Classical" Approach in Understanding Kinship Effects

Historical family reconstitutions are a powerful tool to apply in the study of kinship effects. By providing information on complete families over time, they can help clarify the role different observed factors have in moderating the impact of kinship, and they can help researchers control for a great deal of unobserved factors that may otherwise complicate identification efforts. However, researchers must remain cognizant of the context in which these kinship effects are estimated and how peculiarities in the population or environment may lead to differences in kin impacts. Different empirical strategies such as those outlined above and applied to the family reconstitution datasets can mitigate these peculiarities but are unlikely to eliminate them.

Historical family reconstitutions represent different slices of societies in different time periods, and kinship interactions can be sensitive to these different contexts. It is thus essential to weigh different models regarding kinship interactions and judge their relative importance within the different contexts. We find, however, too little of this process in empirical studies of kinship, and a reluctance to consider scientific approaches guided by Darwinian theory in favour of approaches more in line with the "classical" perspective on human behaviour. This despite that both approaches have relied on similar, and frequently the same, datasets and methods for their purposes. This issue is improving, but nevertheless remains. For example, recent papers that draw on from both approaches include Mattison, Moya et al. (2018) on age at last birth, Mattison, Seabright, et al. (2018) on mortality of adopted and biological daughters, and Macfarlan et al. (2020) on marriage dynamics.

One factor that may contribute to insufficient knowledge exchange is the different understanding of how individual life courses are shaped by the environment. Traditional approaches to family reconstitution studies investigate the socio-economic, political, and cultural contexts that shape demographic outcomes, and draw explanatory approaches from disciplines including economics, epidemiology, sociology, and political science. Although historical demography includes many concepts of different origins, a framework that connects these explanatory approaches in a theoretical manner is absent. Rather, the concepts that exist in the traditional approach tend to be solitary. While there may be multiple models which explain the same phenomena, since they often rely on different assumptions, it becomes difficult to evaluate which has the greater explanatory power. From a theoretical perspective it is impossible to tell whether one concept is superior or whether multiple concepts are simultaneously correct. For this reason, many alternative concepts in historical demography coexist.

Evolutionary theory, on the other hand, considers all concepts within the Evolutionary approach to share the same paradigm: humans stockpile behavioural strategies and environmental response mechanisms which have been shaped by their phylogenetic history. As such, scientific approaches within the evolutionary theory framework are more interested in the variability than in the generality of traits. The opposite is true for the "classical" perspective which views variability more as an issue of disturbance and does not require such an active creator role of the individual or its traits.

This is not to say that the "classical" perspective neglects the role of individual traits or that concepts of response mechanisms are absent. In fact, the impact of individual traits and

characteristics like gender, age, and social position on the life course have been traditionally important study topics, and there exist theories about the mechanisms for how individuals respond to their environment. The key difference to evolutionary theory approaches is that the "classical" perspective is agnostic towards the origin of these mechanisms apart from their social development. In contrast, all disciplines which are guided by Darwinian Theory are connected in the same framework. A unifying theoretical framework results in wellspring of hypotheses because a hypothesis successfully tested in one discipline can be transferred to another (Mattison and Sear 2016).

We illustrate this with the concept of cooperative breeding. Cooperative breeding originally derived from animal studies but has been introduced successfully into family demography (Kramer 2010). The trade-off between current and future reproduction is an essential concept for life history theory (Fisher 1930) and further explains cooperative breeding behaviour. Like any other resource, breeding places and breeding opportunities are almost always limited in the wild and therefore highly competitive. Young adult individuals might have trouble to find promising breeding places, because these are often occupied by older (and stronger) individuals. Consequently, young adults across many species relinquish their own reproduction for a breeding season (or more)[11] and instead support their parents as helpers-at- nest in raising new offspring (Lukas and Clutton-Brock 2012). The postponement or sacrifice of reproduction by the helpers may benefit them through increased reproductive success in the future or increased inclusive fitness. Cooperation thus is a strategy to achieve success beyond the limits of solitary enterprise.

Conclusion

Human life histories and demographic outcomes are impacted by kin in many ways, and human evolutionary theory is essential to understanding why these impacts may be sensitive to the kin relationship or the context in which the kin interact. However, because kin impacts may also be correlated with non-behavioural accompanying factors, it can be hard to disentangle evolutionarily driven effects from other factors correlated within a family. Historical family reconstitutions, with their coverage of family member life histories and often over several generations, provide a possible solution. Through controlling for the effect of the family, it may be possible to control for confounding unobserved factors and successfully identify the impact of kin.

We emphasize, however, that controlling for family effects does not eliminate researcher responsibility to consider the context of their results and the theoretical framework into which they fit. Other unobserved factors affecting kinship interaction and outcomes may remain. For example, kinship influences may still be environmentally dependent and require a comparative approach. Environmental factors such as the level of extrinsic mortality may not only directly affect the size and structure of kin compositions but might

11 It should be mentioned here that biology distinguishes between facultative and obligate cooperative breeders. Eusocial insects and mammals (e.g. naked mole-rats, *Heterocephalus glaber*) are obligate cooperative breeders, because they produce sterile helpers. These helpers are hereditarily predisposed to be helpers and are therefore not exposed to the trade-off between current and future reproduction as facultative cooperative breeders are.

also moderate the relationship between individual kin and, further, the population-specific family concept.

Placing kinship effects within a Darwinian framework illustrates how kinship interactions may be context dependent. In general, human family compositions are more complex when compared to other mammals and exhibit substantial variation between populations. There exist biological causes (among others) for this complexity and variety. For one, human life history is characterized by a long period in which children are not completely dependent on the care of others. Within this pre-reproductive period children can contribute to the family's resource pool. For another, female menopause results in a substantial period of post-reproductive life. Within these pre- and post-reproductive periods, individuals are predisposed to be helpers, but can exist as both breeder and helper. In case of pre- reproductive individuals, the helper role can quickly and unexpectedly turn into a breeder role which might affect the flow of support among a helper-breeder-network. This rationalizes the parental tendency across cultures to intervene in their children's start of reproduction (e.g., request for a parental permission to marry) and why there is a substantial conflict potential between siblings, especially of the same gender, who compete for parental permission to breed first (Beise and Voland 2008). The integration of both biological and social science explanations is thus essential to fully appreciate the complexity and variety of kin effects across different populations.

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12 Note this chapter has been posted on the Open Science Framework website since 15/10/2019, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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SECTION 8: EVOLUTIONARY DEMOGRAPHY OF POPULATION HEALTH AND HUMAN WELL-BEING

Demography is no stranger to bringing many perspectives and methods together. Indeed, the subject matter alone, how and why processes of birth and death change by age and through time, guarantees overlap with nearly all of social science and much of the biological sciences. In this section we highlight the ubiquity of demographic concepts with chapters that bring evolutionary and ecological concepts together in addressing applied topics in health and well-being.

The first of these chapters is by Jonathan Wells, an anthropologist of nutrition who also brings an evolutionary perspective to his work, and whose past work on the concept of the "metabolic ghetto" should be of wide interest in many circles of demography. Wells is well-known for explicitly considering power relations in his work, typically at the population-level; his "metabolic ghetto" concept concerns how socioeconomic hierarchies, both within and between populations, affect health through nutrition. Here, on this theme, Wells reviews and identifies linkages among social hierarchies in mammals, primates and contemporary humans, demonstrating that nutrition is the ultimate context through which hierarchies are developed and maintained. This finding is fleshed out using classic models from ecology developed to understand general social relationships among members of a population or species, which Wells ties with concepts of maternal capital developed by economists. The implications of this approach are fascinating and far-ranging.

From ecology we know that the density of individuals in an area has a huge impact on all kinds of social interactions and these in turn affect resource distributions, mortality rates, behavioural diversity and many other factors. Arguably, the mechanisms of density dependence have been less thoroughly studied among human populations (but for any species, one could probably say not enough is known about how their demography is shaped by density dependence). To help initiate a wider appreciation for the importance of density in contemporary demography, DeLong identifies five pathways of density dependence, akin to five horses of a density-driven apocalypse, one of which is nutrition, the focal consequence of social hierarchy in Wells' chapter. This chapter is timely given the call for re-igniting interest in density dependence in the human life history literature, but it goes much further in efficiently covering a lot of conceptual ground and calling attention to key population-level issues facing our fairly immediate future. In this broad overview DeLong also presents an ecological view on some of the positive checks referred to by Kreager, via Malthus, from Chapter 2.

Lawson and Gibson articulate the largely untapped potential of human evolutionary demography in international development, where the goal is specifically to improve human health and well-being, including providing guidance for more effectively communicating research perspectives to a general public. Lawson and Gibson are a team of evolutionary anthropologists who have been vigorous and pioneering advocates for "applied evolutionary anthropology", using this discipline as a tool for actively improving population health, for some time. In targeting three pressing and challenging topics in this chapter, polygynous marriage, marriage at early ages ("child marriage") and female genital mutilation/cutting, they show how evolutionary scientists can responsibly inform the policy discussion by identifying novel understandings for why such behaviours occur. A reviewer not familiar with evolutionary approaches commented on this chapter that they "enjoyed reading it because it challenged my thinking", which is exactly the kind of reaction interdisciplinary work should evoke.

Gurven and colleauges give an overview of their team's long-term work, known as the Tsimane Health and Life History Project, one of the most important and influential projects in contemporary anthropology. Here they focus on the related topics of lifespan, aging (changes in well-being with age), infectious disease and chronic disease. Evolutionary theory shapes the nature of a research perspective that is empirically driven and illustrates the value of long-term field research. They make an especially strong case for how an evolutionary-informed approach can contribute to understanding human health more broadly. This work should be required reading in any health demography or epidemiology course; recent work by Gurven and colleagues has demonstrated that medical research is almost as "WEIRD" as psychological research (i.e. that medical research is very heavily biased towards samples from countries that are "WEIRD", meaning Western, Educated, Industrialized, Rich, and Democratic). This means that an understanding of health and disease in a non-WEIRD context is vital to rebalance our perspective on these issues. They also point out a need for more long-term studies akin to theirs, which are critical for evolutionary demography, of course, but also for public health, epidemiology and medicine.

Lastly, Pavard and Metcalf, two tried-and-true "evolution-first" evolutionary demographers, conclude the volume with a tour-de-force of applied evolutionary demography that brings a novel view to the evolution of aging patterns based on cancer mortality. Their chapter ties in analysis of age-specific mortality to long-standing issues in life history and the biology of aging. It is an exhilarating read that makes a truly original contribution to thinking on both cancer and how it may have shaped human aging patterns via life-history trade-offs. We chose it as the "final chapter" because many of the concepts developed in earlier chapters are brought together here to a truly exemplary degree in terms of theory, analysis and originality.

27. The Impact of Social Dynamics on Life History Trajectory and Demographic Traits: Insights from the "Producer-Scrounger" Game

Jonathan Wells

Evolutionary demography applies models and theories from evolutionary biology to understand variability in fertility and mortality patterns. Many important ecological influences derive from the natural environment, such as the burden of infectious disease, or the availability of energy and other nutrients. However, human society is itself a source of diverse stimuli and stresses that may generate profound impacts on demographic traits. On this issue, much attention to date has focused on the *benefits* of social interaction, in particular "cooperative breeding" through which the costs of reproduction are shared among kin or others. In contrast, this chapter will use a simple model of social inequality, based on the ecological "producer-scrounger" game, to shed light on how social hierarchy, through the key medium of nutrition, can shape diversity in life history trajectories. Life history trade-offs shape both physiological and behavioural characteristics of individuals, which in turn affect both fertility and mortality profiles. In every society, it is ultimately through relationships embedded in the context of nutrition that different groups within social hierarchies interact. The key insight from the producer-scrounger game is that in social hierarchies, the life history strategies of producers and scroungers are structurally inter-related. This results in contrasting phenotypes and demographic outcomes between the two groups. Those lower in social hierarchies have higher risks, and fewer opportunities to acquire resources, and may adapt through trade-offs that favour immediate survival and reproduction over growth and long-term health maintenance. In contrast, those with priority access to resources may demonstrate trade-offs that favour growth and long-term health maintenance, leading to greater longevity, a lengthier reproductive career and higher quality offspring. These contrasting life history strategies may emerge through the direct control of subordinates by high-ranked individuals, or through indirect control over the resources that subordinates struggle to access. This simple conceptual approach can help understand both contemporary variability within and between populations in demographic traits, and also their historical divergence or convergence over time.

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Introduction

Demography is the study of the size, structure, and distribution of populations, and the variability that they exhibit in association with patterns of births, migration, ageing, and deaths. Until recently, the discipline drew little insight from evolutionary theory, and instead focused on describing demographic patterns, and unravelling the proximate mechanisms that underlie variability in fertility, ageing and mortality. Such mechanisms include behaviours, cultural values and the hormonal regulation of reproductive function.

The sub-discipline of *evolutionary demography* emerged specifically to improve understanding of *why* individuals exhibit variability in demographic outcomes. Patterns of reproduction represent the consequences of a series of decisions (Sear, Lawson et al. 2016), some made consciously, others effectively "made by the body" through hormone-regulated mechanisms of physiological plasticity, through which traits relevant to reproduction or survival respond to ecological conditions (Wade, Schneider et al. 1996, Schneider 2004). Patterns of ageing, and hence mortality risk, can be considered through the same lens. Ultimately, these mechanisms are assumed to have been shaped by the action of natural selection on ancestral populations, so that both the pattern of producing offspring, and their characteristics (including reproductive potential and likely lifespan), have evolved to enhance the odds of parental genes passing to future generations.

It is well recognised that human reproduction is inherently social, perhaps best expressed in the phrase that "it takes a village" to raise children (Hrdy 2009). In recent decades, many researchers have considered how cooperative behaviours can help spread the various costs of reproduction over a larger pool of individuals, often kin, thereby reducing the pressures facing individual mothers. For example, several studies have evaluated potential support from grandmothers, as discussed in more detail below.

However, less attention has been paid to the impact of human relationships that are far from cooperative, and might therefore pose challenges for survival and reproduction. Some work has focused on the absence of individuals such as fathers who, if present, would likely have been beneficial for their offspring (Webster, Graber et al. 2014, Sear, Sheppard et al. 2019). This approach still assumes that fathers generally contribute beneficially to the development of their offspring. Others have considered whether parents-in-law, who do not share genes with the mother, may prioritise paternal interests at the expense of maternal outcomes (Leonetti, Nath et al. 2008).

Overall, aspects of the social environment that may prove unsupportive of women's reproduction remain relatively unexplored, and there is no over-arching framework through which different types of antagonistic relationships may be investigated. In this chapter, I aim to provide such a framework, by showing how hierarchical relations can affect biological and behavioural traits with relevance to demographic outcomes. I will consider two types of hierarchical relations: those within a social group, and those between social groups (whether defined at the level of class or caste, ethnicity or nationality). In order to integrate my approach with the work of others, however, I begin by reviewing several theoretical approaches used in evolutionary demography.

Theoretical Perspectives in Evolutionary Demography

Evolutionary demography draws heavily on life history theory (Stearns 1992, Hill 1993), which assumes that energy is a limited resource for every organism, and must be allocated across four competing functions (maintenance, growth, reproduction, and defence against pathogens or predators). Allocation "decisions" or trade-offs between these functions are the means through which individuals can respond to stimuli or stresses to maximise fitness. A key principle of evolutionary medicine is that natural selection shapes organisms to maximise survival and fitness, at potential costs to health or longevity (Nesse and Williams 1994). Accordingly, allocation trade-offs between maintenance and reproduction shape both longevity and fertility, and these trade-offs intensify in harsh conditions.

Using this framework, several important issues have been addressed. For example, fertility and mortality patterns have a complex connection. Reproduction is metabolically costly for the mother and may accelerate the physiological rate of aging, potentially shortening her lifespan (Westendorp and Kirkwood 1998, Penn and Smith 2007). However, greater investment in reproduction may either increase or decrease the risk of specific maternal diseases, depending on the underlying physiological mechanisms (Jasienska 2017). Counterbalancing such risks is the possibility that adult offspring might eventually care for their parents in old age, prolonging the lifespan of those who have reproduced. Overall, these inter-generational trade-offs vary in association with ecological and social conditions.

Another key issue is that kin may cooperate to share the costs of reproduction (Hrdy 2009). "Pooled energy budgets" help distribute costs that would otherwise fall entirely on the mother (Kramer and Elison 2010), thereby reducing the magnitude of inter-generational trade-offs. Grandmothers may be particularly important in this context, both reducing child mortality rates (Sear and Mace 2008) and promoting child growth (Gibson and Mace 2005, Meehan, Helfrecht et al. 2014), though in general this applies more to maternal than to paternal grandmothers (Sear and Mace 2008). The notion that human reproduction is typically a "cooperative enterprise" is now well established, and this strategy may have been an important factor favouring both encephalization and greater longevity in the genus *Homo*, though different explanatory theories have been presented (Isler and van Shaik 2012, Wells 2012).

Another approach rapidly gaining momentum is the "developmental origins of adult health and disease" (DOHaD) hypothesis (Barker 2004). This focuses on how ecological exposures early in the life-course shape the quality of adult phenotype. Pioneering work showed that early growth patterns predict the risk of conditions such as diabetes and cardiovascular disease in adulthood, thus contributing to variability in longevity (Barker 1992). This approach can be broadened to encompass other demographic outcomes, including maturation rate, fertility, migration and the rate of aging.

For example, a high infectious disease burden during infancy is associated with delayed menarche, indicating reduced availability of energy for early growth (Ellison 1981). In chronically under-nourished Indian populations, growth continues past the age of 20 years (Satyanarayana, Nadamuni et al. 1981), yet Indians still remain one of the shortest populations globally (N. C. D. Risk Factor Collaboration 2016). In this scenario, growth proceeds slowly and ceases late in terms of "time on the clock" but early in terms of the "final size attained". In settings with high vaccination rates and low infection risk, however, rapid infant weight gain is associated with earlier menarche (Dunger, Ahmed et al. 2006, Ong, Emmett et al. 2009), while

high mortality risk can also favour earlier maturation provided that there is adequate energy for growth (Walker, Gurven et al. 2006). In this scenario, growth ceases early in terms of the "time on the clock". Both of these scenarios are nonetheless consistent with life history theory, by showing that life history trajectory is sensitive to cues of both energy supply and mortality risk (Figure 1).

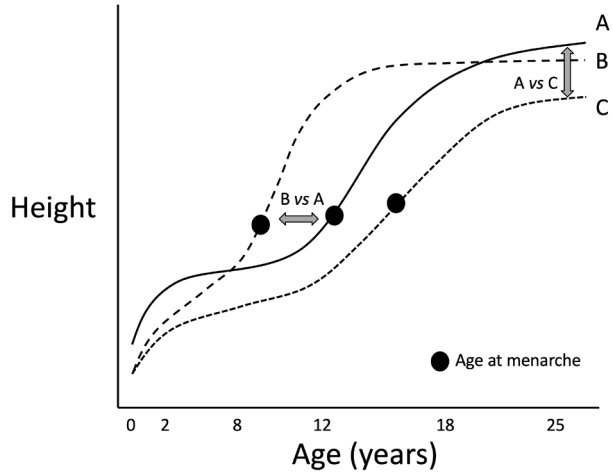

Fig. 1 Schematic diagram, illustrating three contrasting patterns whereby developmental trajectory response to ecological cues. Relative to a healthy growth trajectory in a benign environment (A), high mortality risk, in association with adequate energy supply, favours earlier maturation (B), whereas inadequate energy supply, in the absence of high mortality risk, can favour delayed maturation (C). The arrows highlight contrasts between individuals on these trajectories on one axis, while showing similar values on the other axis.

The "DOHaD" approach can be extended to an inter-generational time-frame, as variability in developmental trajectory is associated with parental nutritional status (Barker 1992, Monaghan 2008). Below, I discuss how these insights can be developed further, through the lens of "maternal capital", allowing me to develop a new perspective on the association between demographic traits and social hierarchy.

The Maternal Capital Model

Mothers maximise their fitness by allocating some of their resources to their offspring, a scenario known as "parental investment" (Trivers 1972). Consequently, life history trade-offs in each generation are initially shaped by many components of maternal behaviour and biology, that determine the level and timing of investment (Wells 2019) Each offspring passes through a succession of "critical periods" during development, and a given level of maternal investment during foetal life may have different effects on the offspring, compared to the same level of investment at a later stage of development (Wells 2018).

The concept of "embodied capital" provides a broad framework with which to investigate how individuals accumulate diverse fitness-enhancing characteristics through the life-course

(Kaplan, Lancaster et al. 2003). Building on this approach, I defined maternal capital as "any aspect of maternal phenotype, whether somatic or behavioural, which enables differential investment in offspring" (Wells 2010). This approach was developed to help integrate evolutionary approaches with the DOHaD hypothesis, by emphasising maternal phenotype as the key niche experienced by the offspring during its most plastic period of development, the "first thousand days of life".

Much attention has focused on physical components of maternal capital, such as energy stores, body size, micronutrient status, and the burden of infections. Across countries, for example, maternal height is inversely associated with the risk of stunting, underweight and early mortality in the offspring (Ozaltin, Hill et al. 2010). Many other components of maternal nutritional or physiological status show similar associations (Wells 2010).

However, other types of resource are also relevant, though they may all ultimately impact the offspring via nutritional pathways. For example, education in its broadest sense (social learning) can enhance parenting success, and can help improve the success of reproductive behaviours such as lactation, which in humans is not instinctive (Wells 2006). Social capital may be equally important, ameliorating the costs of producing and nurturing offspring as discussed above. Beyond grandmothers, other beneficial groups include siblings who may act as "helpers at the nest", whereas the benefits of fathers for child survival are more variable (Sear and Mace 2008). In Ethiopia, support from maternal grandmothers was associated with better growth of grandchildren through reducing the mother's workload (Gibson and Mace 2005). Beyond the contribution emanating directly from these helpers, we should also recognise the agency of mothers in nurturing such supportive networks.

Similarly, material capital may contribute to physical shelter, the capacity to produce food directly, or in market economies the ability to purchase food, accommodation and medical care. These resources are typically influenced by other members of a woman's household, such as brothers, parents, uncles, husbands or parents in law. In patrilocal and patrilineal societies, women have little or no agency in selecting the characteristics of the household in which they will produce their offspring, or how material resources are allocated, as these choices tend to be made by other family members. Nonetheless, the resulting resources still have major implications for maternal fitness, and hence the phenotypic quality of offspring.

The availability of maternal capital also depends on reproductive scheduling, and can vary in association with maternal age, parity, inter-birth interval and the sex of the offspring, as well as time-varying factors such as household composition, financial assets and so on. A broad conceptual model of maternal capital is presented in Figure 2.

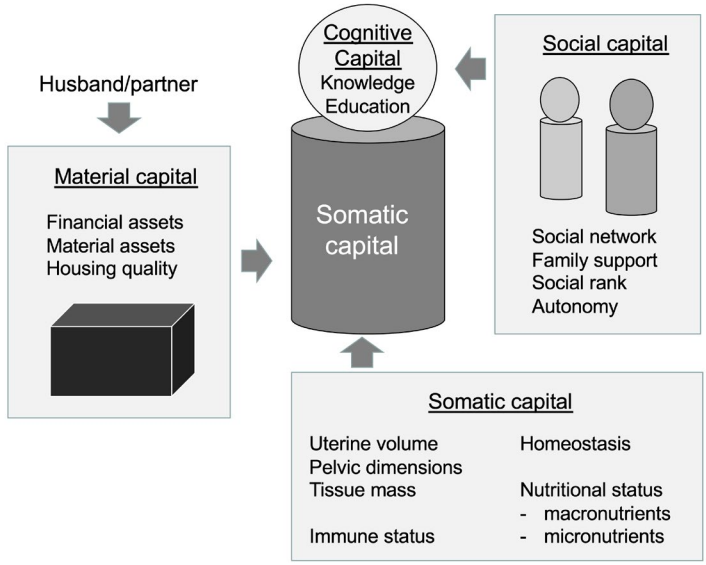

Fig. 2 Components of maternal capital, a composite trait that maternal promotes investment in the offspring. The central cylinder represents the maternal body, through which diverse tissues, organs and skeletal structures contribute to a wide range of types of somatic capital. The upper circle represents the brain, and cognitive capital, through which mothers can also access social capital, in the form of supportive individuals and networks, and material capital, such as housing or financial support.

It is no coincidence, I have argued, that the primary window of plasticity in each life-course — the first thousand days — coincides broadly with physical exposure to maternal capital through placental nutrition and lactation (Wells 2003, Wells 2014). The offspring benefits from substantial buffering against ecological stresses during its most sensitive periods of development, though this protection is imperfect. However, this benefit is obtained at a cost: offspring phenotype is strongly influenced by maternal phenotype, and such effects may benefit maternal fitness, potentially at the expense of offspring fitness. In this way, maternal capital is the *critical* determinant of the life history strategy initially adopted by the offspring (Wells 2016). While some maternal-offspring correspondence in life history strategy is likely to reflect shared genes, there is ample evidence that plastic maternal traits also exert strong effects on the phenotype of the offspring.

The way in which maternal phenotype imprints the *composite* life history strategy of the next generation was recently shown by a study of South Asian women living in the UK (Wells, Yao et al. 2016). Birth weight, a simple proxy for maternal nutritional investment in early life, was associated with a series of life history traits, indicative of life history "decisions" or trade-offs, in the daughters: lower birth weight predicted earlier menarche, shorter adult stature, higher levels of body fat, and higher blood pressure (Figure 3). Collectively, therefore, lower maternal investment in early life obliges the daughter to shunt energy towards rapid maturation and reproduction, at a cost to somatic growth and the long-term capacity for homoeostasis.

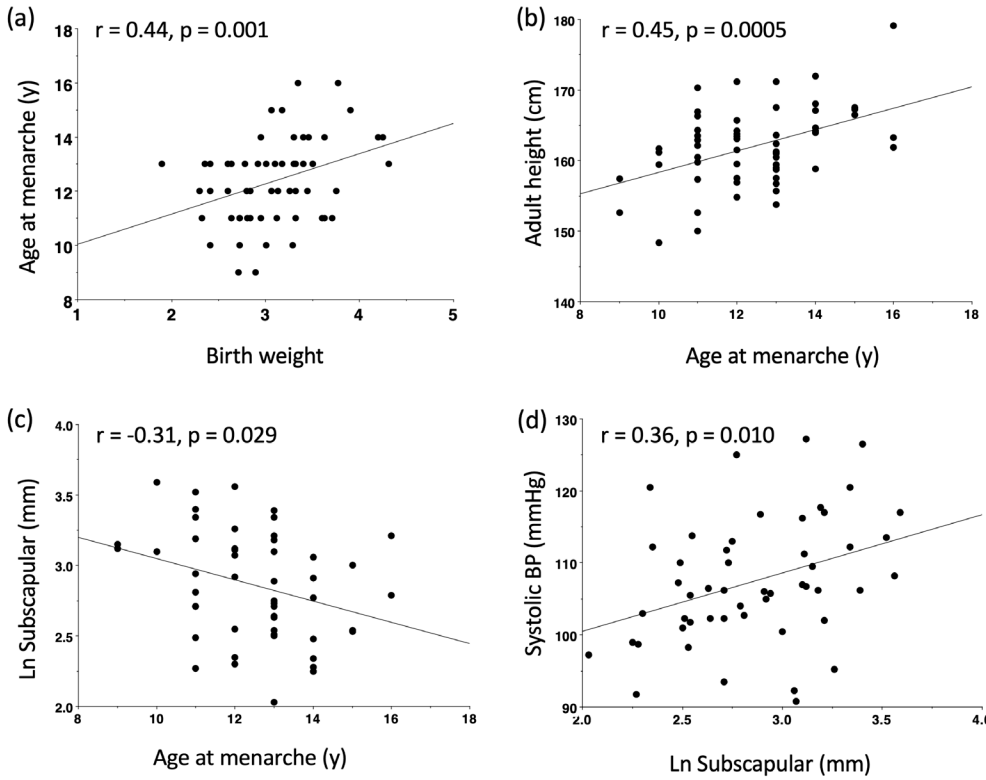

Fig. 3 A chain of life history decisions in South Asian women living in the UK. (a) Birth weight (indicating maternal investment) is inversely associated with age at menarche. (b) Earlier menarche is associated with lower adult stature. (c) Earlier menarche is associated with higher adult body fat. (d) Body fat is positively associated with systolic blood pressure. Reproduced with permission from (Wells, Yao et al. 2016).

Similar findings emerged from a larger study of mothers and their adult daughters in southern Brazil. Maternal capital was assessed using a composite index, integrating data on height, nutritional status, education and household income. Mothers with low capital had daughters who grew poorly in height from foetal life onwards, and who instead developed a more central (unhealthy) distribution of body fat by adulthood. In this setting, daughters receiving low maternal investment did not undergo early puberty, but were still more likely to have reproduced by 18 years, in comparison with those receiving high maternal investment. Once again, therefore, low maternal investment in early life induced trade-offs in the daughter that favoured short-term survival and reproduction at a cost to somatic growth and homeostasis, highlighting that the overall trade-off between health and fitness has its basis in developmental plasticity (Wells, Cole et al. 2019). As yet, it remains unclear whether sons would show similar or contrasting trade-offs.

We can therefore see two key periods of developmental plasticity in the offspring. The first component enables the adjustment of early growth trajectory to the magnitude of maternal investment, while external stresses are buffered. The second component allows the reorganisation of life history strategy in response to the external resources and stresses

encountered in postnatal life (Wells 2019). Trade-offs enacted during the second period are strongly shaped by those occurring in the first (Wells 2010, Wells, Yao et al. 2016).

Once we understand the crucial role of maternal capital on life history trajectory of the offspring, we gain new insight into the association between social hierarchy and demographic outcomes. Most human populations show diverse forms of social inequality, so that the magnitude of maternal capital is inherently associated with the mother's place in the social order (Wells 2010). The more severe the hierarchy, the greater the imprint of society on maternal capital, and the more strongly development of the next generation is imprinted by maternal social rank. If high-ranked mothers can direct more resources to each offspring, then those of low-ranked mothers are exposed to depleted maternal capital.

Precisely because maternal rank is relatively stable, offspring are consistently exposed to its metabolic correlates throughout early development. Thus, the very buffering systems that reduce exposure of the offspring to ecological stresses such as food insecurity and infections increase the exposure to maternal rank (Wells 2010, Wells 2016). To understand these associations, it is helpful to draw on evolutionary models that explicitly acknowledge the dynamic relationships that characterise hierarchy and the mediating role of nutrition.

The Producer-Scrounger Game

The physiological mechanisms through which hierarchical relations impact life history strategies can be examined through the lens of the "producer-scrounger" game (Wells 2016). This dynamic game was developed by ecologists, to understand how social interactions result in unequal distributions of food among organisms. The approach may be particularly valuable in humans, because nutritional dynamics lie at the heart of all forms of human hierarchy and inequality (Wells 2016). The fundamental insight of game theory is that the best strategy for one individual depends on what others in the population are doing.

One of the first games used to study interactions over resources was the Hawk-Dove game (Maynard Smith 1982). In this approach, hawks use aggressive tactics to obtain resources, whereas Doves avoid aggressive interactions. When Hawks compete with Doves, Hawks almost inevitably obtain the resource, but when Hawks compete with each other (regardless of winning, losing or sharing the pay-off) they have a high risk of physical injury. In a population of hawks and doves, the crude pay-offs of Hawk-Hawk and Dove-Dove interactions are equal, but paired Doves ultimately do better than paired Hawks because they do not experience physical injury.

The Hawk-Dove game can be applied to many social interactions in humans. One way or another, power imbalances always involve unequal access to resources, which ultimately resolve to energy. In social species, therefore, power relations are directly relevant to life history theory as they mediate the conversion of energy into fitness (Wells 2016). Nevertheless, foraging would be a very costly activity if a large proportion of individuals regularly indulged in overt aggression to obtain their energy supply. A more suitable game for modelling access to resources is therefore the "producer-scrounger" game (Barnard and Sibly 1981), which addresses more subtle forms of competition, and unlike the Hawk-Dove game takes into account how resources are obtained in the first place.

Producing and scrounging are considered discrete and incompatible tactics: an individual cannot engage in both simultaneously (Morand-Ferron, Giraldeau et al. 2007). Directly producing a resource may lead to an immediate payoff, known as the "finder's share". However, unless

the resource is immediately consumed, it may be stolen by another individual, a scrounger. At the simplest level, the potential payoff for either strategy depends on the proportion of the population engaged in each activity. The fewer the producers, the fewer the opportunities for scrounging, and the lower the average payoff. As producers increase in number, so do the returns the scrounging, but if there are too many scroungers then the equilibrium tips back in favour of producing. This frequency-dependent interaction thus shapes the distribution of the two strategies in the population (Figure 4).

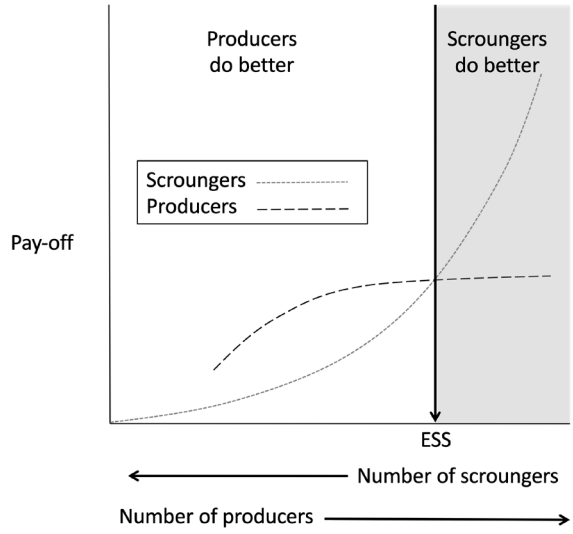

Fig. 4 Schematic diagram illustrating a simple dynamic model of two types of foraging strategy, producing and scrounging. The pay-off from scrounging increases as the relative number of producers rise. Redrawn with permission, Anim. Behav. Vol 29, Barnard and Sibly, 'Producers and scroungers: A general model and its application to captive flocks of house sparrows' pp. 543–50, Copyright (1981) with permission from Elsevier.

Producing and scrounging could be addressed entirely at the level of behaviour, representing alternative tactics to obtain a food package. Whether producers or scroungers obtain a higher pay-off depends on the nature of the resource. For ubiquitous resources, such as a common food plant, producing may far more efficient. Where substantial effort is needed to locate and access resources, however, scroungers can potentially outsource the costs of foraging by exploiting producers, hence increasing their own pay-off.

Producing and scrounging can represent alternative tactics for every individual, and each could select the behaviour offering the best returns on a moment-by-moment basis. Scrounging is predicted to be favoured when producers are unable to prevent it, when food packages are visible and high quality, and when a given resource has greater value to the scrounger (for example, if they are hungrier than the producer, and prepared to take more risks to obtain the resource) (Brockmann and Barnard 1979).

However, if individuals were to commit systematically to producing or scrounging, persistent variability in the supply of energy among individuals could emerge. In a study of bats, for example, individuals consistently produced or scrounged from other animals over several months, and individual scroungers repeatedly targeted specific producers (Harten, Matalon

et al. 2018). On this basis, the two life history strategies of the two types of forager would be predicted to diverge. Producing and scrounging would no longer be *ad hoc* behavioural tactics, but more fundamental strategies that become "locked" into components of physical phenotype such as growth rate, adult size, body composition and sexual signalling (Wells 2016). The next sections consider evidence for this scenario in primates and humans.

Hierarchy and Life History Trajectory in Non-human Primates

Evidence from primate research supports three relevant hypotheses — that primate societies typically demonstrate significant social hierarchies; that a mother's social rank affects her maternal capital, and therefore shapes the life history trade-offs demonstrated by her offspring; and that individual primates within a population may act either temporarily or more consistently as producers or scroungers.

In mammals in general, the effects of high social rank on fitness vary by sex. Theoretically, the fitness of male mammals is limited most strongly by mating opportunities, whereas that of females is constrained by the costs of pregnancy and lactation. On this basis, females are expected to prioritize access to nutritional resources, whereas males are expected to prioritize gaining access to fertile females. Within any species, we would expect higher-ranked members of social groups to align more closely with these ideal strategies than those of lower rank. In other words, high-ranked males might use their status to achieve greater body size and thereby increase access to fertile females, while high-ranked females might use their status to obtain the best quality foods or reliable support networks. In each case, high status would promote fitness, and this prediction is increasingly supported. Across mammals in general, for example, dominant females are younger at first conception, have shorter birth intervals, produce more offspring, have better offspring survival rates, and may even suppress the reproduction of lower-ranked rivals, thereby diverting resources towards themselves (Ellis 1995).

In primates, female rank appears as expected to be relatively independent of body size, and arises through coalitions and alliances, which may be inherited through the matriline. This means first that social skills are essential for attaining high rank, (Chadwick-Jones 1998), and second that social networks are a key conduit through which the pay-offs of high rank emerge. Among Chacma baboons, for example, higher-ranked females with greater social capital live longer and produce more surviving offspring (Silk, Beehner et al. 2009, Silk, Beehner et al. 2010).

In general, high-ranking mothers across diverse primate species transmit more nutritional resources to their offspring, often supported by priority access to the best foraging areas (Wells 2010). The greater nutritional investment results in faster offspring growth, and accelerated maturation of female offspring (Pusey, Williams et al. 1997). Overall, therefore, there is substantial evidence from non-human primates that maternal position in the hierarchy generates major inter-generational effects, through the medium of greater maternal capital.

Specific application of the producer-scrounger game in primates remains rare, however in studies of baboons, females were more likely to scrounge from co-feeding neighbours of subordinate status (King, Isaac et al. 2009), and low-ranked animals tended to experience lower food intakes when resources were scarce (Marshall, Carter et al. 2015). These studies therefore provide early evidence that variability in maternal capital may relate to competitive foraging dynamics, through which some animals obtain more energy at the expense of others.

Hierarchy and Life History Trajectory in Humans

In humans, there is likewise substantial evidence that social hierarchy is associated with variability in life history traits and demographic outcomes. Many studies have shown that children from more advantaged backgrounds are larger in size at birth, and remain so in adulthood (Eveleth and Tanner 1976). For example, family income was strongly associated with birth weight and infant weight gain in a population from southern Brazil (Victora, Barros et al. 1987). These differences attenuate only slowly when secular changes in living conditions occur (Kuh, Power et al. 1991), indicating powerful inter-generational effects (Wells and Stock 2011). Constraints on social mobility mean that maternal capital runs in families, propagating height inequality over time. Already by birth, therefore, the mother's position in the hierarchy has profoundly impacted the life history strategy of her offspring.

As economic development occurs, nutritional constraint during foetal life and infancy may be followed by compensatory catch-up growth. Sometimes this results in larger size in adulthood (Siervo, Stephan et al. 2011), but in other cases catch-up represents an acceleration only in tempo, resulting in adulthood being achieved sooner but at smaller body size. An extreme version of this scenario is shown by Indian girls adopted in early life into Swedish families. Substantially smaller than the Swedish girls at birth, the Indian girls underwent precocious puberty without resolving the growth deficit, leading to short adult height (Proos 2009). A similar pattern on a broader scale is evident in many middle/higher income populations, where age at menarche is decreasing over time while adult height remains relatively static (N. C. D. Risk Factor Collaboration 2016, Wells 2016).

Collectively, these studies show that life history traits vary in association with social rank in a range of settings, but that the nature of the association depends on the risks and resource availability of the setting.

The Fundamental Role of Nutrition in Hierarchy

The nature of social hierarchy has varied substantially across time and geography, but as I argue below, the producer scrounger game can be applied to many different types of human society, and to many different types of behavioural interaction. If this single ecological game has such widespread application, it is because nutrition and power are fundamentally connected (Wells 2016).

In contemporary societies, position in the hierarchy is often assessed in terms of the capacity to participate in markets, reflecting financial wealth. However, nutrition is the ultimate context in which all hierarchies are generated and maintained (Wells 2016). Reflecting the emphasis of life history theory on energy dynamics, I use the term nutrition to refer not only to food intake but also to physical activity patterns and the condition of the body in terms of its growth, composition and ability to resist infectious diseases. Likewise, while the producer-scrounger game has been primarily applied to feeding interactions, its potential for understanding the consequences of social inequality is much broader. According to life history theory, the resources that are subjected to scrounging should not be limited in concept to discrete food parcels, but rather to energy dynamics in general. In non-human apes, for example, we could apply this lens to activities such as parental care and allo-mothering, while for humans the remit could

be extended to a huge range of activities, such as work, taking risks, material resources and so on (Wells 2016).

It is generally accepted that the least hierarchical form of human society is foraging. Ethnographic studies indicate that foragers actively suppress hierarchy, in part by sharing food and other resources on a routine basis (Kelly 1995, Wiessner and Schiefenhovel 1997). Foragers never know who will fail to find adequate food on any given day, hence the available food tends to be redistributed relatively equally, and over time everyone is a net contributor, i.e. a producer, whilst those experiencing shortfalls are merely temporarily scroungers. Foragers tend to use several "levelling strategies" to reduce the emergence of social differences, and promote mutually supportive relations with neighbouring groups. Overall, therefore, systematic scrounging is suppressed, though subtle forms may persist including differences in status and gender inequality. Simple horticultural societies also show modest hierarchies, for the role of human labour in producing food limits the production of resources available for scrounging (Gurven, Borgerhoof Mulder et al. 2010).

Pastoralist societies contrast with foragers in maintaining tangible assets, in the form of animal herds that are potentially more susceptible to scrounging. In one sense, animals now represent the primary producers, and humans are generically the scroungers. Beyond that, certain social groups often attempt to raid the herds of other groups. Such competition may lead to long-term inequality between groups, since power and status are closely associated with the size of the animal herd (Borgerhoff Mulder, Fazzio et al. 2010). Again, nutrition is central to this relationship, since animals are the primary food source for pastoralists.

With the emergence of intensive agriculture, crops and stock animals became resources more susceptible to scrounging. Agriculture also encompasses a high element of risk, due to the possibility of harvest failure. This results in farmers regularly borrowing from each other, in order to recover from ecological shocks. Should they fail to repay their debt, they are often obliged to forego access to the land, and become tenant farmers or other forms of servile worker (Graeber 2011). Landowners and tenant farmers thus emerge as a new form of hierarchical society, in which food production remains central to the unequal status. This transition is associated with the emergence of major differentiation in body size and other life history traits (Shenk, Borgerhoff Mulder et al. 2010, Wells 2016).

In early industrial society, many of those low in the hierarchy shifted from agricultural production to manufacturing, in part through their displacement from common lands that terminated their capacity for direct food production. Those higher in the hierarchy could exploit this scenario by offering low wages, so that landless workers must work long hours in the new factories in order to purchase adequate food through markets (Hobsbawm 1968). Differential control of nutrition was thus a key element in the transformation to early industrial society, which saw the producer-scrounger relationship "reinvented" in the context of markets without negating its underlying logic. Ever since, the market has remained the primary medium of access to food in high-income countries, introducing a new interface between life history strategy and wealth inequality.

Thus, human hierarchy always involves groups that emerge in the context of each other, operationalized primarily through the medium of nutrition. The brief summary presented in this section does not discuss the heterogeneity that manifests within each broad mode of subsistence, or the variable associations that social inequality may show with life history traits

in different settings. My broader argument is that it is through systematically obtaining food and other resources from producers that scroungers acquire and maintain their dominant status, with implications for life history traits and demographic outcomes (Wells 2016). Whilst this allows us to explore social hierarchy *within* populations, the same approach can be applied to relations *between* populations.

Social Hierarchy Within Groups and Demographic Outcomes

According to the notion of "evolutionary stable strategies", the frequency of producers and scroungers stabilises when the average pay-off of each strategy is the same (Barnard and Sibly 1981). In human societies, however, scroungers may use various forms of power to coerce the producers. Consequently, the greater the degree of hierarchy, the greater the trade-off between health and fitness in the lower ranking group.

Figure 5 illustrates this scenario in a hypothetical population, in which a small number of dominant scroungers co-exist with a large number of subordinate producers, who provide the food requirements of the entire population. Over time, the size of each sub-population may remain constant, such that the average fitness of producers and scroungers is equal. However, this apparent equality may co-exist with substantial differences in demographic outcomes and health status. Producers may show high levels of mortality in early life, which translates into shorter average lifespan. Those that survive to adulthood may compensate for the higher juvenile mortality by having higher fertility rates. In combination, exposure to high-risk environments in early life and the elevated metabolic burden of reproduction (and labour) in adult life is expected to accelerate metabolic markers of ageing. This will result in poorer metabolic health and shorter life-span in those producers who reach adulthood.

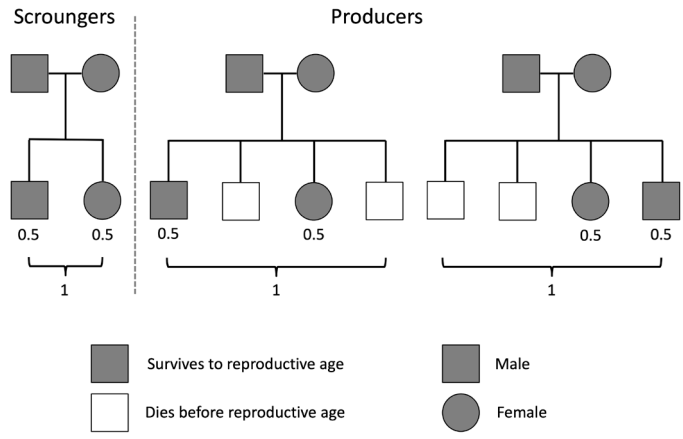

Fig. 5 Schematic diagram illustrating fitness and demographic outcomes in a hypothetical population of producers (common and subordinate) and scroungers (rare and dominant). Scroungers have negligible juvenile mortality and produce small numbers of high-quality offspring. Producers have high juvenile mortality, and compensate through higher fertility rates. The total fitness of producers and scroungers is similar, but producers have shorter average lifespan.

Given their superior health and longevity, one might ask why the scroungers do not also show high fertility. However, it is well established that greater height, wealth and education, all markers of higher social rank, do not typically drive higher fertility, but rather result in a higher quality of offspring.

Recent data from the UK provide support for this model. As argued above for human societies in general, the living standards of wealthier and poorer groups in the UK are structurally related. Comparing across levels of deprivation, poorer groups show higher levels of infant mortality (Kershenbaum, Fu et al. 2017), and shorter total life-span as well as shorter healthy life-span (the years spent free of chronic disease) (Office for National Statistics 2014), while the age of first reproduction also tends to decrease with the level of deprivation (Chipman and Morrison 2013). Collectively, these data support the over-arching hypothesis that demographic patterns emerge through variability in life history trade-offs, that in turn reflect the position of a given individual in the social hierarchy.

Social Hierarchy Between Groups

The producer-scrounger game can also be applied to relationships between populations, whether these are defined as castes, ethnic groups or countries. In each case, dominant groups acquire resources from, and in the process maintain power over, subordinate groups. Once again, the consequence is expressed in health inequalities that impact demographic outcomes such as fertility and life expectancy.

At the broadest level, the producer-scrounger game can be applied at an international level, thus helping understand demographic differences across countries. In the modern globalised market, unequal trading relations that emerged on the back of past relations of imperialism and colonisation maintain some countries as producers of cheap food, where large sections of the population continue to suffer poverty, high burdens of infectious disease, food insecurity and chronic under-nutrition. High-income countries have priority access overall to high quality foods and low burdens of infectious disease, though they may also maintain high levels of social inequality within the country. In turn, this helps explain why "producer countries" have higher average fertility than high-income countries, and lower average longevity.

Once again, the key point is that the demographic outcomes of different countries are structurally linked through their unequal access to resources, and the resulting life history trade-offs that emerge depending on their position in the international hierarchy.

Conclusion

In this chapter, I have explored the utility of a dynamic game theory model of social inequality for integrating across several fields of academic enquiry, in order to improve understanding of life history variability within and between populations. The resulting conceptual approach can in turn help understand contemporary variability within and between populations in demographic traits, and also their historical divergence or convergence over time. I propose that this framework offers new opportunities to explore the demographic consequences of relations within and between populations that are not cooperative, but rather exploitative. To date, interest in sociality and life history theory has focused almost exclusively on cooperative relationships, which has produced an unbalanced perspective on the relationship between human society and biological and demographic outcomes.

I give particular emphasis to nutrition because it is the key link between the various components of the over-arching framework. First, the central component of nutrition is energy supply, which fits well with the focus of life history theory on energy allocation strategies, which incorporate developmental adjustments in early life. However, my definition of nutrition is very broad, and relates to diverse forms of energy use and storage as well as energy income. Second, I have argued that all human hierarchies ultimately reduce to unequal access to energy as the most fundamental resource, that underpins all human activities. Third, I argue that it is because nutrition is the primary medium in which human hierarchies are generated and maintained, as recognised explicitly in the producer-scrounger game, that nutritional health is also where the primary benefits and costs of social inequality manifest at the level of individuals and populations (Wells 2016). This theoretical framework is illustrated in Figure 6.

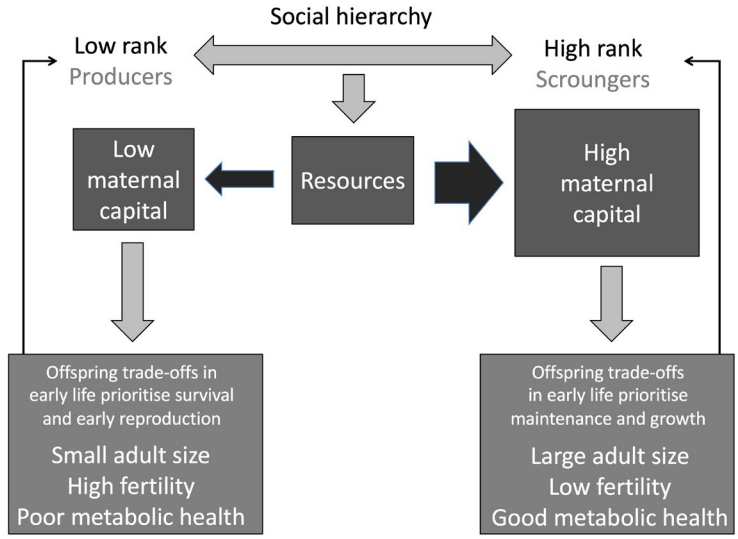

Fig. 6 The over-arching framework linking the interactions of the producer-scrounger game with variability in maternal capital and hence with life-history trade-offs in successive generations. Producers acquire lower levels of resources and hence lower levels of maternal capital. This leads to trade-offs favouring immediate survival and reproduction in the next generation, at a cost to growth and health. Smaller adult size then renders the next generation susceptible to becoming producers too. In contrast, scroungers gain more resources and acquire more maternal capital. This results in trade-offs favouring growth and health in the next generation, leading to large body size and a high chance of becoming scrounger.

Recent studies provide empirical evidence for links between position in the hierarchy, variability in maternal capital, life history trade-offs in the offspring and metabolic and demographic outcomes (Wells, Cole et al. 2019). However, the dynamic game may also be used directly in future, to test *in silico* how the life history trade-offs of producers and scroungers interact, through the medium of contests over energy availability. A strength of this approach is that it may be applied to any form of human society across time, geographical space, subsistence mode and culture.

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28. Pathways of Density Dependence and Natural Selection in Modern Humans

John P. DeLong

Living things depend on a flow of energy and materials to grow, reproduce, and maintain their bodies. Populations are aggregations of individuals, so they too depend on resources. Humans use many fuels derived from the Earth's photosynthetic energy, which in turn support a population that often occurs at unusually high densities for a mammal. Like most populations, growing human populations may experience negative feedbacks from population size unless the socio-economic system in which the population lives grows fast enough to maintain resource flows to individuals and to limit the downsides of high density. I map out a simple view of the pathways of density dependence through five main causes of negative feedback: poor nutrition, increased disease, increased toxins, altered life history strategies, and violent conflict. The pathways trace the different ways in which increasing population size can cause lower birth rates or higher death rates and set the stage for selection on contemporary human populations. Some of the pathways are not traditionally viewed as density-dependent, but since they all depend on a tension between population size and the ability of the socio-economic system to generate positive feedbacks, they are all a form of density-dependence. These pathways are also dependent on changes to the global environment, including warmer and more variable climates, and the way people respond to the feedbacks by altering socio-economic expectations or technology.

Introduction

Changes in the size of human populations are generated by births, deaths, and migration. All three of these processes arise from a combination of biological traits (e.g., age at first reproduction and longevity) and environmental pressures (e.g., disease, climate, and predators). Understanding these pressures, and how they drive long-term population growth, is key to fostering a sustainable society. This is because the same factors that might stabilize population growth play a role in stabilizing the economy, human health, and political systems (Frisch 1978; DeLong and others 2010; Brown and others 2011).

As with most natural populations (Sibly and others 2005), human populations may experience negative feedback from the environment that can limit population growth (Zhang and others 2007). When that feedback is mediated by the size of the population, it is known as density-dependence. Here I will consider abundance and density equivalent, because in today's world, the global population and country-level populations exist within fixed (or at least infrequently changing) political boundaries. Unlike most non-human populations, human populations also may exhibit

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positive density dependence. For example, super-exponential growth during the early twentieth century required a relaxation of negative density-dependence, or more precisely, positive feedbacks that more than compensated for whatever negative feedbacks were in place (Cohen 1996, 2003; DeLong and Burger 2015; Burger and DeLong 2016). An indication of this positive feedback is the observation that the total amount of energy used by human populations has on average kept pace with or exceeded population growth over time (DeLong and Burger 2015), presumably due to technological or social advances (Bettencourt and others 2007; Weinberger and others 2017).

Density-dependence in human populations has not been well studied empirically (but see Lee 1987; Lutz and others 2006; DeLong and Burger 2015; Burger and others 2017), perhaps in part because rapid population growth in many countries over the last few centuries seems to suggest that negative feedbacks are somewhat unimportant (Lee 1987). There has been plenty of interesting theoretical discussion about the role of density-dependence and resource constraints on the ultimate size of the human population (Foerster and others 1960; Bettencourt and others 2007; Hamilton and others 2009; Kaack and Katul 2013; Burger and others 2017; Malthus 1798). However, density dependence in human population growth is empirically detectable in time series data (Wrigley 1983; Lutz and others 2006; Bettencourt and others 2007; DeLong and Burger 2015), indicating a real need to try to understand it.

Here I lay out five pathways of negative density-dependence in human populations that could slow human population growth either now or sometime in the future. They are not mutually exclusive. These pathways are caricatures of more complex phenomena and are meant to illustrate the likely causal pathways from increasing human population size back to decreasing birth and/or increasing death rates. I take a deliberately ecological view, as population growth always can be reduced to the mechanisms of births, deaths, and migration, and all of these mechanisms are driven by the way humans interact with their environment (i.e., ecology). In this view, economics, society, and technology are functionally all components of human population ecology. I also consider how such negative density dependence may set the stage for different forms of natural selection in human populations. Finally, I consider the implications of density dependence and trait evolution for understanding and predicting human population growth.

I begin with a simplistic overview of how the human population is embedded in a socio-economic environmental system, itself embedded in the biosphere, that influences human life (Figure 1). People live within a system that provides (to varying extents) the things people need including food, shelter, safety, and health care (Daly 1977; Hall and others 2001; Burger and others 2012). The system requires contributions in time, skills, and money from people to actually function, as well as energy and raw materials to power work and with which to make products. The extent to which the socio-economic system can provide the services people require (or want) depends on the capacity of people to operate the system and extract the necessary energy and materials from the environment. It also depends on disruptions in the environment, particularly climate change (including both human-caused climate change and natural climate variation), as this type of change affects the overall productivity of natural systems at a large scale (O'Reilly and others 2003). As human populations grow, the socio-economic system must expand. This expansion is the vehicle of the positive feedback that increases access to resources and mitigates the downsides of high density (Boserup 1965). Whenever the socio-economic system fails to keep up with population size, however, negative density dependence should arise (Butler 2004). The ability to provide services also may depend on links among countries that can trade for goods and services and, in effect, extend the socio-economic system beyond its borders (Suweis and others 2013).

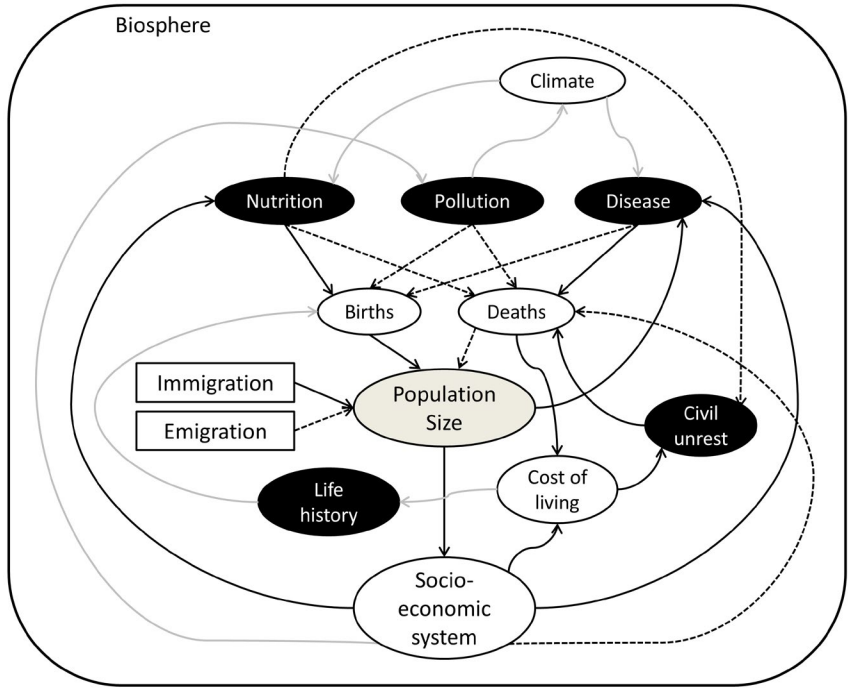

Fig. 1 Schematic of the causal links from human population size back to births and deaths. Population size is in the center, and the five pathways run through the key processes of nutrition, pollution, disease, civil unrest, and life history, all shown in black ovals. Solid black lines indicate positive effects, dashed black lines indicate negative effects, and gray lines indicate more complex effects that do not have an inherent sign. Immigration and emigration are included but not explicitly considered as an alternative pathway, since at the global level they cancel out.

The Pathways of Density Dependence

The nutrition pathway: *Population size → socio-economic system → nutrition → births and deaths*. The nutrition-based pathway is in essence the standard ecological pathway where individuals compete for a limited amount of nutritional resources. As a population grows, available resources must be divided up further and further among individuals, and at a certain point, individual nutrition is poor enough that birth rates decline (i.e., the nutritional requirements of pregnancy are not being met) and death rates increase (i.e., the nutritional requirements of maintaining bodies and fighting off disease are not being met), causing population growth rate to decrease. The population stops growing when the growth rate is zero and birth and deaths are in balance, and this population size is known as the carrying capacity (Cohen 1996; DeLong and Burger 2015). In human populations, as with other populations whose population growth is accompanied by spatial expansion, food and energy resources typically have increased along with population size. This increase in food availability has been made possible by expansion of the socio-economic system that provides the technology to produce and distribute more food to more people over greater spatial extent. Now that human populations have occupied much of the globe, however, further growth of human populations may not be accompanied as easily by increasing the area used to acquire food or other essential

resources, setting up the possibility that limits to the human population could arise through the nutrition pathway.

The disease pathway: *Population size → socio-economic system → disease → births and deaths*. The disease-based pathway traces the effects of increasing population size through the socio-economic system because of the increased disease caused by, among other things, increased stress, declining nutrition, contact among individuals, travel, and drug resistant pathogens. This pathway requires that something about the socio-economic system makes it somewhat incapable of taming certain risk factors for earlier death. For example, as the socio-economic system grows and individual contributions to that system become more constant and demanding, stress levels will increase, setting the stage for a variety of physiological disorders, including heart disease (Kivimäki and others 2006) and sleep disorders (Kalimo and others 2000). Similarly, increased density of people can increase transmission of pathogens, as people come into close contact with more and more people, potentially selecting for more virulent pathogens (Anderson and May 1982). For example, MRSA (methicillin-resistant Staphylococcus aureus) is a type of bacteria that has evolved resistance to numerous antibiotics and is transmitted through contact (Centers for Disease Control 2016). Thus, although growth of the socio-economic system makes health care more available, increasing population size can in some settings set the stage for increased transmission and rapid evolution of pathogens, creating a negative feedback pathway from population size to mortality. A sub-pathway here might skip over disease straight to the mortality risks associated with things like workplace mishaps and car accidents.

The toxins pathway: *Population size → socio-economic system → toxins → births and deaths*. This pathway follows again the increasing activity of a socio-economic system due to increasing population size to the waste products produced by the system (Dietz and others 2007; Rosa and Dietz 2012; Burger and DeLong 2016). Although many waste products are recyclable, isolatable, or transformable by biotic agents into non-toxic products, many other waste products have direct health effects by, for example, altering hormone pathways or causing cancer, leading to lowered fertility and increased mortality. These toxins include those used in agriculture to control weeds, insects, or fungi, some of which have hormone-disrupting effects on people (Richard and others 2005; Mnif and others 2011). Other potential toxins may be present in household cleaners, emitted as a by-product of electricity generation, automobile exhaust, biomass combustion, or manufacturing (Bell and others 2004), and hundreds of such chemicals have been detected in human samples (Centers for Disease Control and Prevention 2009). Thus, as populations grow, and more and more chemicals are used in food production and preparation, to clean bodies, equipment, and buildings, and to manufacture a widening array of products, the potential for toxins to influence births and deaths increases.

The life history pathway: *Population size → socio-economic system → cost of living → births*. The life history pathway traces changes in the allocation of time and energy associated with expectations of living longer and different lifestyle choices made in a larger socio-economic system to lower fertility. This pathway recapitulates the Demographic Transition, which is the shift from low survivorship/high fertility life histories to high survivorship/low fertility life histories that occurs with economic development in most countries, but there may be other types of life history changes involved. The evolutionary benefits of lowering fertility are still somewhat unclear and/or contested (Borgerhoff Mulder 1998; Burger and DeLong 2016), but it

seems likely that people are giving up additional offspring in exchange for some other (real or perceived) benefit, whether it be greater investment in each child (in the form of time, money, education) (Smith and Fretwell 1974), self-allocation to improve the parent's lifespan or health, or the use of resources for greater parental involvement in the socio-economic system (more work, travel, recreation). Although not generally thought of as a form of density-dependence, this pathway does lead through population size, as it is the larger population size, and the associated larger socio-economic system, that sets the stage for different allocation decisions as people engage in the system in different ways.

The warfare pathway: *Population size → socio-economic system → cost of living → civil unrest → deaths*. This pathway leads through civil unrest because if the socio-economic system cannot expand fast enough, individual needs (or expectations) will not be fully met. For example, the cost of goods and real wages varied wildly with population growth in England in the eighteenth century, indicating variability in the ability of the socio-economic system to keep up with population growth (Wrigley 1983). If the cost of living exceeds the ability of people to pay, or at least for some people to pay if income inequality is high, it also may lead to civil unrest and potentially violent conflicts. This pathway may be augmented by lower nutrition that generates both economic and medical distress. Although clearly complex, an empirical relationship between population size and the amount of civil unrest can be detected, depending on geography and other factors (Raleigh and Hegre 2009; Thayer 2009). Thus, growing populations may experience more violent population regulation, depending on the capacity of the socio-economic system to mitigate these effects.

These five generalized pathways are not mutually exclusive and are likely to be operating at the same time. If this is true, then it will be very difficult to empirically identify the relative importance of each pathway in driving future changes in human population size, or for that matter to even detect them without controlling for multiple causal variables. Furthermore, if all of these pathways are important, along with the potential positive feedbacks that can mask negative density dependence, then predicting the dynamics of human populations will require modelling many hard-to-detect and interacting processes.

Many countries today are rather fixated on economic growth. Whether explicitly acknowledged or not, the underlying goal of any effort to grow the socio-economic system is the reduction of the negative effects of increasing population size. That is, the growing socio-economic system may alleviate the negative effects of larger population size, obscuring the underlying density-dependence operating in the population. Thus, whenever a society can expand the socio-economic instrument by increasing energy and material inputs, creating greater efficiencies in providing services, or eliminating threats from disease or toxins, it can minimize density dependence (Boserup 1965). Whenever it struggles to do this, the effects of density dependence — through any or all pathways — will inevitably arise (Butler 2004). Which pathway the density dependence will take, however, will depend on the specific nature of the society and the set of challenges it faces. Thus, there is an element of unpredictability to density-dependence in human populations.

A complicating feature of density dependent pathways in human populations are the recent and projected increases in average global temperatures and climate variability (IPCC 2014). Because the human socio-economic system is embedded within the biosphere, changes to the broad patterns of temperature and precipitation will change the distribution of ecosystem

productivity in space. These changes are likely to have impacts on the nutrition and disease pathways because they may disrupt the functioning of the socio-economic system in at least two ways. First, temperature and water availability play an overriding role in determining crop production, so crop production may be altered (for better or worse depending on the location) (IPCC 2014), altering the magnitude of the nutrition feedback. Second, the risk of contracting tropical diseases may spread to higher latitudes from the tropics, as increasing temperatures in temperate areas can open these areas up to tropical diseases whose vectors require less severe winters, altering the magnitude of the disease pathway (Patz and others 2005). For example, while very complex, the civil war in Syria was preceded by a severe drought that exacerbated the economic challenges the country already faced (Gleick 2014). Thus, the already complex set of potential negative feedbacks from population density to population growth rate is made more complex by spatial heterogeneity and unpredictability of the effect of climate change on the feedback mechanisms.

Selection on Human Populations

It has long been understood that populations experiencing the negative feedbacks of density dependence can evolve due to genetic variation in traits that influence the ability of individuals to reproduce and survive (Darwin 1859; Lee 1987; Nekola and others 2013). As with any population, traits that reduce survival in humans will be selected against, and traits that favour successful reproduction will be selected for. For example, earlier maturation is associated with higher fitness. Selection for earlier maturation should arise, then, barring opposing costs and trade-offs with other traits. In an isolated pre-industrial population on Ile aux Coudres island in Canada, exactly this was observed. Selection favoured an advance of maturation by four years over roughly seven generations (Milot and others 2011), prior to industrialization and a change in the fitness landscape (i.e., the set of relationships between traits and fitness). Even with strong fitness gradients, however, selection may be limited by gene flow, low heritability of fitness-linked traits, and both genetic and ecological pleiotropy (Williams 1957; Barton 1995; Futuyma 2010; DeLong and Gibert 2016).

With five different pathways, density-dependence in humans has the potential to generate natural selection in human populations depending on what negative feedbacks arise. These negative effects could be on mortality, which may limit lifespan and the potential for reproduction depending on the age of death. Thus, traits that enable people to tolerate stress, process toxins, resist diseases, avoid accidents, and escape violence might all be under selection to a greater degree as population size grows. Negative effects of stress, toxins, and disease might also influence reproduction, and thus traits that maintain fertility despite the negative effects of larger population size would also be favoured by natural selection. Although evolution has been viewed historically as too slow to influence ecological process such as the population feedbacks arising through density dependence, more recently it has become clear that rapid evolution can occur in ecological time for a wide range of organisms including humans (Hairston, Jr. and others 2005; Schoener 2011; DeLong and others 2016; Milot and others 2011; Byars and others 2010).

What kinds of traits could be involved here? Many of these traits could be physiological traits associated with the allocation of energy and materials within our bodies. For example, a genotype that allocates more resources to immune function would likely be favoured along

the disease pathway, depending on the costs of that allocation to other competing ends such as growth or reproduction. Similarly, allocation to greater toxin processing might enable some people to tolerate the higher body burdens of toxins that we carry today (Centers for Disease Control and Prevention 2009). Interestingly, there is evidence that contemporary human populations are under selection for lower blood pressure (Byars and others 2010), which could enable greater tolerance of stress and maintain fitness in current socio-economic systems. A variety of potentially interacting fitness gradients are likely present in contemporary human populations, making future evolution complicated and hard to predict without more information.

Given the many ways that increasing density can lead to negative feedback on birth and death rates and thus on population growth, it might be surprising that these feedbacks are rarely considered in models of human population growth. For example, the regular U.N. population projections do not explicitly consider any form of density dependence in their models (Lee 2011). The *effects* may be included in some cases, for example estimates of mortality from HIV/AIDS are included in estimates of survival for countries with high prevalence of this disease (United Nations 2011; Gerland and others 2014). Nonetheless, the mechanism (i.e., a link between population density to disease-induced mortality) is not considered, even though it is clear that providing preventative healthcare services and treatment requires a socio-economic system that has sufficient energy, materials, and skills to function. I argue that when a socio-economic system cannot keep up with diseases that are having real negative effects on people that the population living in that system is experiencing a form of density dependence. It would be useful to have a generalized understanding of how a society's energetic and economic capacity per capita translates into the potential for dealing with density-dependent effects such as disease, toxins, malnutrition, and violence.

Possible Issues for the Future

It has now been well more than a century since Malthus wrote about the inevitability of limitations on human populations through density-dependence (Malthus 1798). Malthus argued that populations grow exponentially and food production grows linearly, because food production was thought to be just a multiple of the amount of land in production. Therefore, population needs eventually would exceed the food supply, causing the population to stop growing or possibly decline. This idea has been both embraced for recognizing the obvious limits on global food production (i.e., there is only so much land and sunlight) and disparaged for making incorrect predictions (the dire warnings have not [yet] come to pass — notably this is more true primarily for countries with expanding socio-economic systems) (Lam 2011; Allendorf and Allendorf 2012; Nekola and others 2013). Malthus was incorrect not in recognizing the links between resources and population growth but in the assumptions he made. Populations only grow exponentially when there are expanding resources to support it, so when resource limits engage, population growth will slow down and be tempered by the growth of food production. Thus, exponential growth cannot occur as a population approaches its resource limitations. Second, food production can increase faster than the simple expansion of area used for farming. This latter assumption was shown to be very plainly false as the Green Revolution took hold, and even further increases in per area production are possible with revolutions in genetics, soil management, and integrated pest management. Nonetheless,

spraying pesticides to control insects and fertilizing crops may not help much when food production systems are increasingly challenged by extreme weather events such as droughts and floods. So the potential for increasing per area yields in the future remains unclear.

Switches from slower to faster growth are clearly visible in the pattern of world population growth through history (DeLong and Burger 2015). Such switches suggest that negative density dependence was relaxed, leading to increased birth rates or decreased death rates (Wrigley 2013; Kaack and Katul 2013; DeLong and Burger 2015). In other words, some aspect of the expanding socio-economic system facilitated access to new resources, better utilization of existing resources, or both, and that the benefits of this carried through to the processes controlling population growth. Should we expect further increases in the resource base for humanity through technological innovation or fundamental changes in the needs of people? This is difficult to say. It may depend on which type of resource ends up being the most limiting. There are many different ways to produce electricity, and many different types of food to eat, but there is nothing that can substitute for water. Although water can be used more efficiently and can be extracted from the ocean (at high cost), water could be the most important limiting nutrient generating negative density-dependence in humans, even if our global socio-economic system can accommodate considerable trade and generosity (Suweis and others 2013). Another candidate limiting nutrient could be phosphorous, which is both a pollutant when it runs off into water bodies and is globally limited in minable quantities (Elser and Bennett 2011). Regardless of the limiting nutrient, however, it is clear that our socio-economic system and its ability to function is the core mediating structure moderating density-dependent feedbacks in human populations. Whether future negative effects of increasing population size pass through the nutrition, disease, toxins, life history changes, or warfare pathways, however, is impossible to predict at this time.

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1 Note this chapter has been posted on the Open Science Framework website since 05/07/2019, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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29. Evolutionary Approaches to Population Health: Insights on Polygynous Marriage, "Child Marriage" and Female Genital Mutilation/Cutting

David W. Lawson and Mhairi A. Gibson

An evolutionary perspective offers remarkable insight into the roots and current drivers of human behavioural diversity, not least with regard to the study of demographic and population health phenomena. It also holds considerable, yet largely untapped, potential to inform the actions and priorities of international development sector. In this chapter, we contrast the ways in which questions of human diversity and wellbeing are approached by evolutionary behavioural scientists and population health scholars, and highlight exemplary evolutionary research addressing applied topics of contemporary policy relevance. We concentrate on three case studies: polygynous marriage, early or "child marriage", and female genital mutilation/cutting. Each of these behaviours is now targeted by global efforts to achieve gender equality and promote female wellbeing. However, policy aiming to change behaviour remains poorly informed by an understanding of why such ostensibly harmful behaviours occur. Here, we outline rival theoretical models, their supporting evidence, and potential implications. In an effort to encourage dialogue between evolutionary scientists working on population health issues and the international development sector we also consider the challenges of doing applied research, including how best to navigate disciplinary boundaries and engaging with, and influencing, policy-makers, stakeholders and the general public.

Introduction

As this book testifies, there is a growing enthusiasm for the added value of an evolutionary approach in the social sciences, not least in demography and population studies. In this chapter, we consider the application of evolutionary theory to population health issues in the international development sector. As a field of study, population health encompasses the study of health inequality within and between populations, including health outcomes, health determinants and the design and critique of measures that may be taken to improve health and reduce inequality (Kindig and Stoddart 2003; Young 2005). Researchers working under this umbrella definition do not share a singular theoretical framework, but rather draw on theory and methods from fields such as (social) epidemiology, demography, economics and psychology. As such, forging new connections with evolutionary behavioural science extends

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a pre-existing commitment to interdisciplinary exchange and synthesis. Here, we focus specifically on drawing out novel contributions to population health science from the emerging field of *applied evolutionary anthropology*, drawing on its key foci of human diversity and adaptation, context-dependency in the drivers and consequences of behavioural strategies, and a strong aversion to ethnocentrism i.e. the tendency to judge other cultures by the values and standards of one's own (Gibson & Lawson, 2014; Gibson & Lawson, 2015; Tucker & Rende Taylor, 2007).

In what follows, we offer our own observations and reflections on the contrasting "worldviews" taken by evolutionary anthropology and population health science, and the, still largely untapped, potential for collaboration across these fields. In doing so, we highlight recent exemplary research in applied evolutionary anthropology and identify priority areas for future study. Rather than offer an exhaustive review, we focus on three case studies where we believe the contribution of evolutionary ideas holds great potential: the drivers and wellbeing implications of polygynous marriage, early or "child marriage" and female genital mutilation/cutting (FGMC). Each of these phenomena have been labelled "harmful cultural practices" by those working in international development, and are typically viewed as inherently damaging to child and adult wellbeing, including reproductive and sexual health.

While our immediate focus is narrowed by the use of these case studies, we also invite interested readers to consider the wider range of ways in which evolutionary behavioural scientists are tackling applied themes in population health. Other notable foci include the wellbeing consequences of intervention programs, including the impact of labour-saving technologies on birth rates (e.g. Gibson, 2014; Kramer & McMillan, 2006), indigenous health and livelihood shifts (e.g. Gurven et al., 2017; Page et al., 2016; Tucker, 2007); socioeconomic disparities in health behaviours (e.g. Pepper & Nettle, 2017); child care practices, including fostering and adoption (e.g. Lawson et al., 2017) and biased parental investment (e.g. Alvergne, Faurie, & Raymond, 2009; Du & Mace, 2018); the timing of puberty (e.g. Kyweluk, Georgiev, Borja, Gettler, & Kuzawa, 2018); intimate partner violence (Stieglitz and others 2018); the implications of biased population sex ratios (e.g. Schacht, Rauch, & Borgerhoff Mulder, 2014; Uggla & Mace, 2017), and more (Gibson & Lawson, 2015).

Understanding "Harmful Cultural Practices"

Evolutionary behavioural scientists and population health scholars approach questions of human diversity and wellbeing from very distinct starting points. Most obviously, there is a difference in core objectives. Evolutionary behavioural scientists share an overarching objective to generate and test theoretical models, derived from evolutionary biology, to better understand human behaviour and the world we live in. They ask ultimate and proximate questions about *why we do what we do*. By default, they have no stake in questions of *what should we do*, which cannot be predicted on the basis of evolutionary theory alone (to do so would be to commit the naturalistic fallacy i.e. inferring what ought to be, from what is deemed natural).

Population health scholars working in the international development sector, on the other hand, have the overarching objective of improving human experience. Interest in explanations of current or historical behavioural variation is limited by the extent to which it holds value in predicting future trajectories of development, and is frequently absent altogether. This objective necessitates explicit and shared value judgments about what constitutes desirable behaviour. It

also requires the comparative measurement of populations on generalized metrics to deduce the extent to which a population is "developing" or "developed". Historically, attention has fixated on crude economic (e.g. Gross Domestic Product) and demographic indicators (e.g. child mortality rate, total fertility rate). Today, international or "human development" is used in a more holistic and multidisciplinary sense to include general improvements in quality of life, and increasingly issues of gender equality (Coles and others 2015), which, by extension, has led to increased focus on cultural practices viewed as impeding successful development.

For anthropologists, the notion of placing value judgements on behaviour and the idea of singular dimension of progress on which humanity can be measured have long been met with scepticism. While few disagree that reductions in child mortality or a lowered infectious disease burden can be considered progress, other development targets are vulnerable to ethnocentrism. This is perhaps most obvious with respect to so-called "harmful cultural practices" (HCPs) or "harmful traditional practices", terms used interchangeably to refer to customs ostensibly damaging to wellbeing. This terminology was initially developed by the United Nations (UN) to name and combat seemingly blatant forms of male domination of women (Winter and others 2002). The concept originated in UN circles as early in the 1950s, gathering momentum over the following decades with increased global focus on women's human rights, and culminated in the mid-90s in a UN factsheet devoted to "*Harmful Traditional Practices Affecting the Health of Women and Children*" (UN 1995). Initial emphasis was placed on FGMC (which continues to dominate discussions of HCPs), son preference and its negative repercussions for daughters, female infanticide, child marriage, early pregnancy, nutrition taboos, and unfavoured practices related to childbirth and violence against women (Longman & Bradley, 2016). Today it remains the predominant framework in international development guiding current efforts to abolish a range of behaviours deemed harmful to women and children among low and middle-income countries.

Cultural anthropologists in particular have long raised concern that the HCP framework falsely implies that subordination of women is limited to populations in the Global South and that "modern" cultural practices are exempt from a potential to harm (Winter and others 2002). Concerns have also been raised that a focus on HCPs provides fresh respectability to a long tradition of casting low-income nations as "primitive"; effectively blaming poor wellbeing on the moral failings of local people. Furthermore, by emphasising the cultural determinants of wellbeing, we may stifle investigation of broader socioeconomic and structural drivers of seemingly harmful behaviours e.g. poverty and lack of viable alternatives (Hart 2009; Walley 1997; Pot 2019). Humanitarian efforts to abolish HCPs may thus, in some cases, inadvertently fuel wider patterns of "ethnocentric disdain", adversely influencing socio-political interactions between societies; influencing factors such as aid budgets, immigration regulation, trade negotiations and justification for violent conflict (Hart 2009). Moreover, as discussed below, evidence, or indeed sound supporting theory, that HCPs are best understood as truly harmful varies from practice to practice and across contexts. Yet programs to discourage certain behaviours commonly proceed on the basis of good intentions alone. This leaves wide open the possibility that efforts to improve wellbeing in some instances could be ineffective or even detrimental, no matter now well-intentioned.

Building on these concerns, evolutionary anthropology is well positioned to respond to the growing need to better understand allegedly HCPs. Taking up this challenge involves bold new

steps to engage and collaborate with other applied social scientists to draw out explicit policy implications. Most importantly, an evolutionary approach offers a rich theoretical framework to not only better predict the wellbeing consequences of purportedly harmful behaviours, but also to understand the motivations driving their maintenance across time and space, including the potentially conflicting motivations of men and women, and of parents and offspring. Natural selection, for example is understood to have "designed" the human organism to deploy behaviour which maximises the production of genetic descendants, not health, financial gain or other measures of personal or societal wellbeing. This insight explains why, in any society, humans so often appear to act against their own self-interest (Hill 1993). Behavioural variation observed across cultures is furthermore understood to exist in large part because the pay-offs to alternative behavioural "strategies" are highly dependent on local circumstance (Nettle and others 2013; Kaplan and others 2009). This focus on contingency and adaptation leads to an *a priori* scepticism of broad-based interventions applied cross-culturally with little regard for local context; and instead favours targeted programs designed to address local conditions and specific needs. As a scientific and primarily quantitative discipline, evolutionary (unlike cultural) anthropology also offers new opportunities to reinforce stronger standards of evidence, while still remaining vigilant to the pitfalls of ethnocentric and confirmation bias common in less culturally-sensitive disciplines.

Together these insights offer considerable promise to inform policy, particularly with respect to predicting otherwise unforeseen consequences of interventions and the design of policy aiming to encourage positive behaviour change (Gibson & Lawson, 2014; Gibson & Lawson, 2015). In the sections below, we examine each case study HCP highlighting instructive contributions from classic and more recent evolutionary anthropological research. These discussions draw on key evolutionary concepts such as inclusive fitness, life history theory, parental investment, parent-offspring conflict, mate choice, sexual selection and sexual conflict, which we assume the reader has some basic familiarity with. More detailed introductions to such concepts can be found elsewhere (e.g. Barrett and others 2002).

Polygynous Marriage

Over 80% of preindustrial societies in the ethnographic record permitted polygynous marriage (Murdock and White 1969). Today the practice is most common in Africa, particularly in West Africa, and in rural areas regions within national boundaries (Timæus and Reynar 1998; Westoff 2003). Polygyny is defined as the simultaneous marriage of one man to multiple wives. Yet this simple definition is deceiving; polygynous experience varies widely across and within cultures (Lawson & Gibson, 2018a; White, 1988). For example, the proportion of men and women polygynously married differs, as do the categories of men and women in polygynous marriages, e.g. in some contexts relatively wealthy men are more likely to be polygynous, while women from advantaged backgrounds may be more likely to assort into monogamous unions (e.g. Gibson & Mace, 2007). In sororal polygyny, co-wives are usually sisters or close relatives and share the same residence. In non-sororal polygyny, co-wives are not close relatives and generally live in distinct dwellings, semi-independently from their cowives. Substantial variation is further introduced by religious and legal codes restricting wife number; whether levirate marriage acts as a source of polygyny; the extent and type of wife "ranking"/differentiation; the legal status and rights of secondary wives; the degree of formality of marriage; the opportunity

for and accepted grounds for divorce; the extent of individual choice in marriage partners; and the presence and type of marriage payments (White 1988, Lawson and Gibson 2018a). Circumstances may also differ for a woman who enters an initially monogamous marriage with a period of exclusivity, compared a "junior" wife who enters a marriage with a cowife already in place.

With such variation in what it means to be polygynous, an anthropological view (even without the added insights of an evolutionary perspective) dictates that it makes little sense to anticipate polygyny to have a singular uniform consequence for any aspect of wellbeing across time and space (Lawson and Gibson 2018a). Instead, we should anticipate varied consequences of polygyny corresponding to the multitude of forms of polygynous experience. Indeed, it is possible to anticipate negative (e.g. effects of household resource dilution, co-wife competition) or positive impacts of polygyny (e.g. via marriage to a higher status male, co-wife cooperation) depending on the context (Omariba and Boyle 2007; Madhavan 2002; Strassmann 1997; Jankowiak and others 2005). Polygyny via levirate marriage (whereby a woman marries a male relative of her late husband) requires exceptional consideration because the only feasible alternative to some widows may be to remain single which may leave a woman and her children vulnerable (Palmore 1987). Nevertheless, polygyny has long been regarded unfavourably in western thought, particularly on theological grounds (Witte 2015), actively discouraged by missionaries throughout Africa (Fenske 2015), and is currently condemned by the UN Convention on the Elimination of All Forms of Discrimination Against Women, which states that polygynous marriage "*contravene[s] a woman's right to equality with men and can have such serious emotional and financial consequences for her and her dependents that such marriages ought to be discouraged and prohibited*" (Gaffney-Rhys, 2012: pp. 53). This statement however, predates dedicated empirical investigation into the wellbeing implications of polygynous marriage, treating the practice as effectively "guilty until proven innocent".

Building on models of animal mating systems, evolutionary anthropologists have predicted that both monogamous, polygynous, and even polyandrous marriage (one wife, multiple husbands), can suit both male and female interests in particular socioecological settings, while in others there may be a conflict of interest (Fortunato 2015). An important insight is that polygynous marriage is predicted to be beneficial or at least relatively inconsequential for female and child wellbeing in contexts where women lack direct resource control and males differ substantially in status, so that sharing a wealthy husband is favourable to marrying a low status male (see Borgerhoff Mulder, 1990; Orians, 1969; Verner & Willson, 1966). A number of evolutionary anthropological studies have reported evidence consistent with this hypothesis (referred to as the "polygyny threshold model", Figure 1). Supporting data, for example, comes from studies of polygynous marriage in rural Ethiopia (Gibson & Mace, 2007; Uggla, Gurmu, & Gibson, 2018) and Tanzania (Lawson et al., 2015). In these studies, polygynous men were found to be typically wealthier than monogamous men, and female reproductive success or child health equal or superior in polygynous marriages, at least for first wives.

In other contexts, women may be better understood as coerced into marital arrangements that are not to their benefit. A number of studies have characterized polygynous marriage as the outcome of sexual conflict, with women effectively losing the conflict under patriarchal regimes. This explanation has been argued to better fit, among others, a rural Malian population where polygynous marriage is associated with elevated child mortality (Strassmann, 1997; see

also Chisholm & Burbank, 1991; Sellen, 1999). However, distinguishing between female choice and male coercion as drivers of polygynous marriage is not straightforward. There is some disagreement across the literature on the exact predictions of alternative models (Fortunato 2015; Borgerhoff Mulder 1990), and data limitations, such as a reliance on cross-sectional analysis and a limited range of wellbeing measures, have raised important methodological concerns (Lawson & Gibson, 2018a). In some studies, wellbeing and/or reproductive costs of polygynous marriage have been suggested, but appear limited to, or are more pronounced among, the poorest households, suggesting that polygyny is only costly if there are insufficient resources to go around (Borgerhoff Mulder, 1992; Lawson & Gibson, 2018a). Future research further delineating alternative explanatory hypotheses for polygynous marriage would be valuable. Moreover, the literature needs to engage more directly with considerations of women's wellbeing and measures of livelihood resilience, rather than the use of child outcomes like survival or growth as all-encompassing proxies for maternal and child health. Nevertheless, these considerations imply that polygyny is not likely to be harmful in all circumstances. Indeed, if there are large differences in male status then prohibiting polygyny will logically be disadvantageous to some women by restricting marital options and consequently increasing risk of poverty (see also Dessy and others 2021).

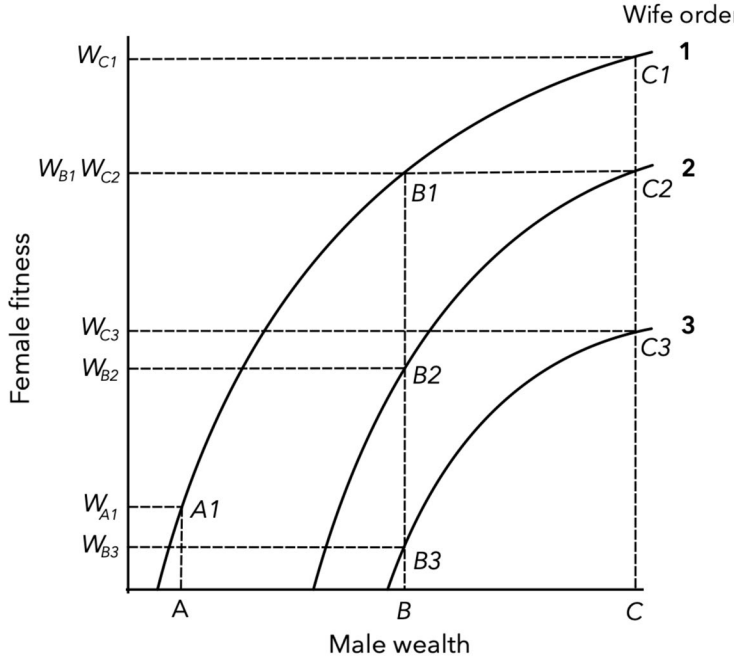

Fig. 1 The polygyny threshold model. Female fitness (W) is determined by the wealth of their husband and by wife order or 'wife rank'. In this particular scenario, first or only wives (wife order = 1) benefit from greater shares of male-owned wealth than later wives (wife order = 2 or 3). The poorest male (A) can only afford one wife, while relatively wealthy men (B & C) may have multiple spouses. Polygyny is adaptive for women in any state where sharing a husband leads to a greater or equal share of male wealth compared to monogamous marriage with an alternative male. For example, being the second wife of wealthiest male C leads to equal female fitness as being the first wife of male B, and much greater fitness than being the only wife of the poorest male A. This graphical depiction of the "polygyny threshold model" is redrawn from Smith & Winterhalder (2006).

How might these insights from evolutionary anthropology be useful to those working to improve women's lives in countries where polygyny is practiced? Most obviously, they present a challenge to the dogmatic view of the universal harms of polygyny across much of development community (Lawson & Gibson, 2018a). By extension, this insight also encourages policy solutions beyond marital reform, be that via enforced new marriage laws or penalties for polygynous families. Indeed, such initiatives not only come at the potential cost of restricting individual agency, but are likely to be ineffective in communities where most marriages are recognized only by customary law. Instead, given the predicted ecologically and individually contingent consequences of polygyny, policy would arguably be better focused on need-based support for vulnerable families (e.g. those experiencing chronic food insecurity), irrespective of individual marital status. Policy could also be targeted to ensure that when polygyny does occur it is most likely to be consistent with women's (and children's) interests. This will require an improved understanding of the *root* socioecological drivers of low female autonomy and resource control. Initiatives to improve women's wellbeing may therefore be better focused, for example, on female land-owning rights, and the support of widows and single-mothers to ensure viable and sustainable alternatives to polygynous marriage.

A focus on context-dependency in anthropological studies of polygyny has also raised important methodological critiques of population health research. Large-scale national datasets, such as the Demographic and Health Surveys (DHS), play a central role in informing the priorities of development sector (David and Haberlen 2005), and patterns established with such data are generally prioritized over the results of small-scale anthropological studies (Lawson & Uggla, 2014). However, there is considerable potential for these datasets to mislead analysts when considering the wellbeing implications of cultural practices.

This issue is illustrated by a study of polygyny across an ethnically diverse sample of 56 rural Tanzanian villages (Lawson et al., 2015). Polygyny was predictive of poor child health when data were aggregated across all villages, consistent with a number of cross-national studies based on African DHS data (e.g. Adedini & Odimegwu, 2017; Omariba & Boyle, 2007; Smith-Greenaway & Trinitapoli, 2014). However, polygynous households had equal or better child outcomes than monogamous households when contrasted specifically to neighbouring households within their own communities, a pattern driven by the greater relative wealth of polygynous households. Furthermore, at the village level, a negative association between polygyny prevalence and child health was accounted for by underlying socioecological differences between villages. In this case, polygyny was most common among ecologically vulnerable (e.g. low rainfall) and socioeconomically marginalized (low service provision, low education) Maasai villages compared to surrounding villages inhabited primarily by other ethnic groups (see also Lawson et al., 2014). This study highlights that the common method of aggregating data across heterogeneous regional units to infer the wellbeing implications of alternative family structures, even across relatively small geographical ranges, can be misleading. Population scientists dealing with large-scale datasets, such as the DHS, should thus be vigilant that apparent negative relationships between "traditional" customs and wellbeing may rather reflect nothing more than the tendency of such behaviours to be most common in already marginalized population sub-groups.

Child Marriage

"Child marriage" is defined within the development sector as the marriage of any individual under the age of 18 years, and is most common in South Asia and Sub-Saharan Africa (Hodgkinson 2016). The last decade has witnessed a dramatic increase in attention to girl child marriage by international organisations, NGOs, and governments (Lawson and others 2020). This includes the founding of "Girls Not Brides" in 2011, a global partnership of now over 1,000 civil society organisations committed to the issue. In 2015, the Sustainable Development Goals marked a global pledge to eliminate child marriage within 15 years, a hugely ambitious target given its current high frequency (Hodgkinson 2016). Humanitarian concern centres on the lack of meaningful consent to marry, and on purported negative consequences on the physical, mental and economic wellbeing of girls and young women, especially via early pregnancy, school dropout and an elevated risk of sexual violence, along with poor outcomes for the offspring of "child brides" (Boyden and others 2012; Gaffney-Rhys 2012; Hodgkinson 2016). Within policy discussions, child marriage is often assumed to always be forced marriage, occurring against a young girl/woman's will. This assumption is also common among the general public within higher-income nations, such as the United States (Lawson and others 2020).

Existing discussions of child marriage are driven largely by moral concerns over "the right to childhood", a perspective assuming the universal applicability of a specific western vision of a clear boundary between childhood innocence and adult responsibility. This vision implies that young people can be shielded from risks and responsibilities of adulthood by abolishing early marriage. Anthropologists have countered that a strict barrier between "childhood" and "adulthood" depends on material, social and cultural conditions that are absent in many settings (Hart 2009). Indeed, remaining unmarried does not guarantee safety and shelter from risk, and young people and their parents may view marriage as mitigating against certain hazards during adolescence (see below). Moreover, the widely-held notion that 18 years universally delimits harmful from healthy marital age lacks an adequate theoretical or empirical basis. Many studies report that those who married under 18 have worse wellbeing compared to those who married over 18 (Hodgkinson 2016). However, analysts near universally impose an 18-year cut-off *a priori*, rather than explore what age categorizations emerge from the data. This approach is more definitive of "policy-based evidence" rather than "evidence-based policy". Consequently, the possibility that alternative age thresholds are more meaningful in some contexts, or indeed that no clear single threshold may be apparent, has been left largely unexplored. Dixon-Mueller (2008) for example, emphasizes the widespread failure of the development community to acknowledge the possibility that "*… late adolescence may be an ideal time for some young people to marry and start a family…*", warning that "*…defining all unions in which one partner is younger than 18 as child marriages and calling for their elimination… could be construed as a denial of their freedom*." (Dixon-Muller 2008, p. 258).

This state of affairs is especially problematic because the majority of "child brides" in the developing world are in their late teens (16-17 years of age). This contrasts with the use of images of pre-pubescent girls by advocacy organizations to represent the plight of so-called child brides, and stifles consideration that teenage and very early child marriage may have distinct drivers and wellbeing implications. The hypocrisy of current international attention on child marriage is further underlined by the fact that "child marriage" over the age of 15 years remains legal with parental consent in the United States and many European nations

(Lawson and others 2020). In one instructive study on the closely related phenomena of "teen pregnancy", anthropologists Kramer & Lancaster (2010), examined the evidence across cultures that 18 years presents a meaningful distinction between harmful and healthy pregnancies. They conclude that when teens are considered as an overall category the evidence for health costs of early pregnancy is mixed. Only by isolating those under the age of 16, is there consistent evidence that early pregnancy is harmful to health. They also note the role of weak social support in exacerbating the costs of teen pregnancy, implying that if social support is available then early pregnancy need not be harmful. These observations highlight the arbitrary nature of an externally drawn boundary between childhood and adulthood at 18 years, and the role of context dependency in determining wellbeing implications of early transitions to adulthood.

Current policy could also be better informed by an understanding of the strategic motivations and/or conflicts of interest accounting for the high prevalence of early marriage, despite its purported costs to wellbeing. Here, evolutionary models of parental investment, parent-offspring conflict and life history theory provide some alternative hypotheses. For example, an evolutionary perspective predicts that early marriage could be optimal for parents and their daughters in some circumstances, in terms of both wellbeing and fitness. Life history theory predicts that optimal age of marriage and reproduction will be sensitive to variation in mortality rates (Nettle 2011; Kramer 2008). In contexts of low life expectancy and high infant and maternal mortality, parents may be keen to ensure early marriage to maximize opportunities for successful reproduction and the availability of kin support during their daughters' vulnerable child rearing years (see also Geronimus, 2003). A lack of income for women may also lead early marriage to be the most feasible means for ensuring a girl's economic security. Archambault (2011), for instance, reports that among Kenyan Maasai, parents deem the much-touted economic returns of delayed marriage for the sake of educational attainment to be locally absent for most girls, while early marriage is viewed as more likely to lead to economic security. Similarly, qualitative research in rural Ethiopia (Boyden and others 2012) suggests that economic security is contingent on collective effort and group solidarity, not individual entitlement. As such, while early marriage may infringe on female autonomy, it can also be vital to consolidating collective and individual interests that are ultimately protective e.g. cementing alliances between marrying families as a means to social security. Early marriage can also be a social mobility pathway, advancing female wellbeing by utilizing the high value of female youth on the marriage market (Voland and Engel 1990). Finally, marrying early may reduce the risk of pre-marital sex, and by association sexually-transmitted disease and the production of illegitimate children. Indeed, while very early marriage has been associated with HIV transmission, particularly when combined with large spousal age gap (Hodgkinson 2016), later marriage can also increase transmission risk due to an increased number of premarital sex partners (Marston and others 2009).

On the other hand, early marriage, particularly at the youngest ages, could truly be best considered harmful in some contexts. But why would parents willingly sacrifice their daughters' wellbeing? Evolutionary theory predicts that detrimental parental behaviours can proliferate if they are the product of conflicting strategic interests between parents and offspring. Most obviously, the optimal share of parental investment will differ between parents and offspring, predicting that parents will be willing to sacrifice the wellbeing of one child if it serves the family unit as a whole (Trivers 1974; Figure 2). Marriage may be a key site at which

such conflict plays out (Wiessner 2009), with scope for conflict further escalated in cultural contexts where parents are the beneficiaries of bridewealth transfers at marriage, or where marrying young reduces the cost of dowry transfers (Schaffnit and others 2019a). Supporting this perspective, child marriage rates have been shown to increase in response to extrinsic economic shocks (drought) in African regions where bridewealth is commonly practiced, with parents presumably marrying daughters early in order to access capital (Corno and Voena 2016). Furthermore, as hypothesized by Apostolou (2010), in order to control the mate choices of their children, parents may prefer them to marry earlier than their children themselves prefer (see also Wiessner 2009).

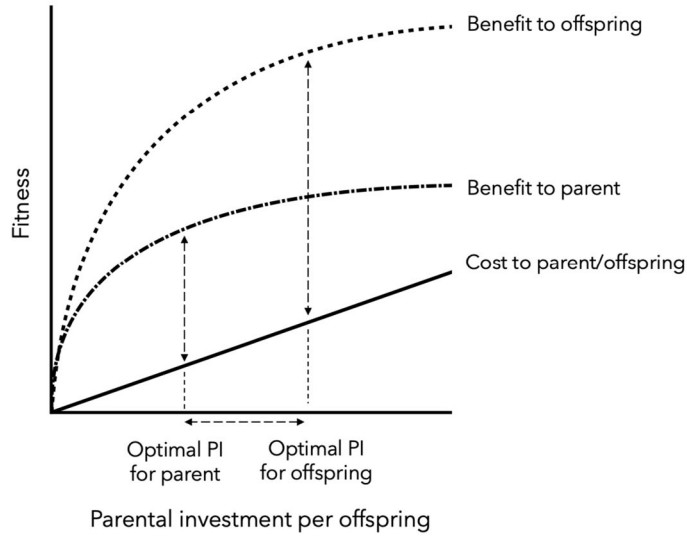

Fig. 2 Parent-offspring conflict. Optimal allocations of parental investment must balance the benefits accrued to an individual offspring vs. costs of reduced investment in alternative offspring (i.e. siblings). For both parents and offspring, increased parental investment has diminishing fitness returns as it saturates an offspring's need. Parents and offspring also experience equivalent costs to continued investment since, assuming full siblings, parents are equally related to offspring as siblings are to each other. Yet for offspring the benefits of continued parental investment are amplified because, while a parent only shares half its genes with each offspring, offspring are fully related to themselves. The optimal level of parental investment is thus lower for parents than for any individual offspring (Trivers 1974). This scenario could apply to 'child marriage', with adolescents preferring to remain under the care of parents for longer, while parents benefit from their earlier marriage and economic independence. Financial incentives to marry daughters early via larger marriage payments could also escalate the scope for conflict (Schaffnit and others. 2019a). Figure redrawn from Lazarus & Inglis (1986).

Lastly, we must also recognize that it is also possible that early marriage is not motivated by wellbeing concerns of either parent or daughter, but rather is to be understood as a strategy that promotes high fertility at the expense of wellbeing. A key insight from an evolutionary perspective is that any behaviour can evolve provided it maximizes inclusive fitness via direct or indirect reproduction (Hill 1993). It seems intuitive that early marriage would lead to higher fitness; with women who marry earlier ultimately going on to have more children than those who marry later, although very early reproduction can pose a maternal mortality risk

(Kramer and Lancaster 2010). Even if those that start reproduction early do not have more children in total, all else equal, a propensity for earlier reproduction can be favoured by natural selection due to shorter generation times (Jones and Bird 2015). This hypothesis suggests that if we wish to reduce the high prevalence of early marriage then this needs to grounded in an understanding of the process by which individuals in low-income countries come to adopt low and delayed fertility norms — a topic of active study by evolutionary demographers covered in detail elsewhere in this book (Sear and others 2016).

These considerations suggest a need to interrogate simplistic, but politically expedient, narratives concerning "child marriage", and produce more culturally-sensitive, evidence-based policy that places emphasis on contextual drivers. Within the development sector, the primary narrative concerning early or "child marriage" is that it is a product of parental coercion driven by financial incentives via marriage payments. Following this observation, Schaffnit et al. (2019), set out to evaluate the fit of a parent-offspring conflict model to the high prevalence of marriage under 18 years in a rural Tanzanian population. Consistent with parent-offspring conflict, bridewealth transfers were highest for younger brides i.e. parents benefit economically from daughters marrying younger. However, self-reported autonomy in partner choice was very common at all ages, relationships between age at marriage and women's wellbeing were mixed and largely equivocal, and women who married early achieved relatively higher reproductive success (Schaffnit and others 2019a, 2019b). The results of this study imply that, in contexts where adolescents have autonomy in marriage choices and where marriage promotes economic and social security, early female marriage may, in many cases, be best understood as serving the strategic interests of both parents *and* daughters. Consequently, "child marriage" may be better conceptualized as product of environments that offer limited alternatives for adolescent girls and young women, rather than as a root determinant of poor female wellbeing. Qualitative research with the same population confirms that early marriage was often viewed as risky for female adolescents, but that it remained desirable because structural constraints, like poverty, limit feasible alternatives and because similar risks, like pregnancy, occur outside of marriage (Schaffnit and others, 2021). Crucially, these results suggest that criminalizing child marriage could increase adversity in some contexts if feasible alternatives are not simultaneously presented for girls and young women (Schaffnit and others, 2021).

A parent-offspring conflict model may fit better to alternative contexts, so that campaigns to eliminate early marriages via criminalization or other means are more likely to yield direct and immediate benefits for girls and young women. For example, arranged and forced marriages are more common and divorce less acceptable in South Asia than in sub-Saharan Africa (Petroni and others 2017), making it more likely that early marriages are costly to women because they cannot remove themselves from marriages, and beneficial to parents because they control the process. In Schaffnit's study in north-western Tanzania, very few marriages also took place under 15 years, and spousal age gaps were rarely extreme, limiting the potential costs of early marriage when weighed against locally available options. More detrimental impacts of early marriage may be anticipated when marriage occurs closer to puberty (e.g. via reproductive health consequences), pregnancy rarely takes place prior to marriage and/or when spousal age gaps are more pronounced (e.g. via heightened power differentials between spouses). As with polygynous marriage, an evolutionary anthropological approach cautions that the drivers and consequences of child marriage are context-dependent, and thus require context-dependent development policy and programmatic considerations.

Female Genital Mutilation/Cutting (FGMC)

FGMC is the partial or total removal of the external genitalia of girls and women for non-medical reasons. The age at which it is conducted varies from days after birth to early adulthood. The practice is most common in African populations, but also found in parts of the Middle East and Asia. It is estimated that 100–40 million girls alive today have undergone some form of FGMC, and a further 15 million may be subjected to it by 2030 (UNFPA 2015). Unlike polygynous marriage and "child marriage" there is little doubt that FGMC is inherently harmful to individual wellbeing across contexts. Health consequences depend on the nature of the procedure, but can include chronic pain, susceptibility to recurrent infections, obstetric complications, fatal bleeding and sexual and mental health consequences. Campaigns to abolish FGMC are now widespread, and elimination of the practice by 2030 is a key target of the UN Sustainable Development Goals (Figure 3).

Fig. 3 An anti-FGMC campaign billboard in Uganda. Source: https://ar.m.wikipedia.org/wiki/ملف:Campaign_road_sign_against_female_genital_mutilation_(cropped)_2.jpg

Both evolutionary and cultural anthropologists have addressed the challenges and morality of eliminating FGMC behaviour (Shell-Duncan and Hernlund 2000; Ross and others 2016; Gibson and others 2018), including contributing insights into the underlying drivers motivating the practice, for example in signalling sexual fidelity and paternity certainty (Howard and Gibson 2019) or in promoting group identity between women (Shell-Duncan and others 2011) or between men (Wilson 2008). Evolutionary behavioural scientists have tended to focus on exploring how and why FGMC could first evolve, but then be maintained (or discontinued) within populations. For example, one prominent view on the origins of male and female circumcision is that it is linked to the emergence of social stratification both within and between groups (Wilson 2008; Sosis and others 2007). Other evolutionary studies have revealed how cultural evolutionary forces like conformity bias (the tendency to copy the behaviour of others) could explain the persistence of FGMC (Howard and Gibson 2017; Ross and others 2016), contributing important ideas on why FGMC persists in some groups, but has declined in others.

A common view among development practitioners and evolutionary scientists is that the procedure controls women's sexuality, which benefits men, resulting in a preference for women with FGMC at marriage. Many evolutionary explanations for female genital cutting have focused on the extent to which the practice provides men with an honest signal of sexual

fidelity. By controlling women's desire for extra-pair sex, it has been suggested that FGMC provides inclusive fitness benefits for men by enhancing their paternity certainty and avoiding the allocation of resources to raising unrelated offspring (Hartung and others 1976). Given the risks to women's health and well-being, the benefits to women (and their kin) seem less clear. It may be that higher paternity confidence leads to greater paternal investment in offspring, resulting in their improved survival and growth; and/or that FGMC may permit women to marry up and into a higher status family (hypergyny) under conditions of resource inequality. Many of these evolutionary ideas remain either untested, or have provided inconclusive results.

Analysing large-scale DHS data from over 70,000 West African women, Howard and Gibson (2019) find that FGMC is not associated with reductions in women's reported sexual activity (extra-pair sex). In other words, cutting does not appear to be an "honest" or reliable signal of a women's sexual fidelity. However, women with FGMC get married at a younger age than those without FGMC (Reason 2004; Howard and Gibson 2019), indicating that FGMC does influence women's marriage opportunities. It may be that health costs of the procedure for women are "tolerated", because there are greater potential evolutionary fitness gains from early marriage (which is directly linked to earlier age at first birth and higher lifetime fertility). Alternatively, in contexts where marriage payments are higher for cut than for uncut daughters, then parents may also be incentivized by the higher economic returns from cutting daughters, leading to resource and fitness gains for the younger siblings of cut women and girls.

Among development policy circles there is a growing interest in the influence of social networks, in particular in how social norms are transmitted between individuals, and how social information could be used to promote the abandonment of FGMC specifically, and HCPs more broadly. One prominent view is that FGMC is maintained by a social coordination game linked to marriage, an idea sometimes referred to as "social convention theory" (Mackie, 1996). The central premise is that maintenance of FGMC in a population depends on a critical number of families who cut and demand cut daughters for their sons. If numbers drop below this point in the marriage pool, the probability of finding a husband without cutting your daughter is sufficiently high for the practice to disappear (Mackie and LeJeune 2009). The policy implication of this account is that if development practitioners convince a critical number of families to abandon FGMC (e.g. via public declarations at large community events) then, with FGMC no longer serving the interest of the few remaining cutting families, it will rapidly disappear.

This approach has been influential among policy-making circles, with considerable resources being invested in development intervention schemes based on mass abandonment ceremonies. There is, however, mixed empirical evidence in support of FGMC being a coordinated practice, or demonstrating the universal efficacy of related interventions (Shell-Duncan and others 2011). Cultural norms, particularly those relating to marriage, do appear to be important in maintaining FGMC practice in some contexts. For example, Hayford (2005) found that Kenyan women's decisions to circumcise their daughter was influenced by community norms (after controlling for individual circumstances). However, Efferson et al. (2015) report no evidence that cutting was coordinated within 45 Sudanese communities, rather they identified substantial levels of variation in attitudes and cutting behaviour between individual families. Howard and Gibson (2017) similarly were unable to find the predicted discontinuous distribution of cutting versus non-cutting conventions across 47 ethnic groups in West Africa, rather that people are disproportionately more likely to copy the FGMC norm of their group. This is consistent

with evolutionary models of conformity bias (specifically a frequency dependent distribution) predicted by Ross and others (2016). The implication of this finding is that rather than allocating limited resources to mass abandonment ceremonies to force numbers below a tipping-point, any intervention which reduces even small numbers of cutters could contribute to a cumulative reduction in FGMC overtime.

Alternatives to the idea that signalling sexual fidelity to secure a better marriage drives FGMC behaviour, have also been proposed by evolutionary behavioural scientists. For example, the practice may signal information not only to men, but to other women, reducing same-sex reproductive conflict and increasing social trust between cut individuals (Wilson, 2008). Evidence that FGMC facilitates the accumulation of social capital by younger women, and power and prestige by elder women in Senegambia provides support for idea that the practice creates important networks between women (Shell, Duncan and others, 2011). However, the notion that FGMC reduces female reproductive conflict remains untested. It seems likely that there will be considerable heterogeneity in the drivers of FGMC, which will be shaped by local context and history of each community (rather than one overarching explanation for the practice).

Evolutionary studies have also focused on the cultural forces which lead to the persistence of FGMC within populations, indicating that there may be strong incentives, including reproductive advantages in conforming to (even harmful) local norms. Howard and Gibson (2017) find that West African women with FGMC have higher numbers of surviving offspring, than women without FGMC, but only in high frequency FGMC ethnic groups. In the low FGMC contexts, women without FGMC have relatively higher reproductive success (Howard and Gibson 2017). Mechanisms which underpin the reproductive benefit of conforming to the FGMC norm of your group may include: simply gaining entry into marriage, and/or having an earlier age at first birth; both of which increases fertility (Reason 2004; Howard and Gibson 2017). Alternatively, conformity may provide access to social and economic resources and skills, for example, access to women's support networks and groups in a high frequency FGMC environment (Shell-Duncan and others 2011) or public institutions like health-care and education in a low FGMC environment; which may increase child survival. In support of the latter, communities perceived to be at high risk of FGMC in the UK, report how "heavy handed" and stigmatizing approaches to safe-guarding children have led to disengagement with vital public services (Karlsen et al., 2019).

A number of questions remain unanswered in evolutionary studies of FGMC, but perhaps the most critical is establishing whether cutting is a reliable signal of group solidarity, sexual fidelity or indeed any other factor. Given that FGMC is not externally visible to a social group and ceremonies increasingly occur in secret (Camilotti 2016), there is considerable potential for families to defect by not cutting their daughters and hiding their uncut status from the group. This raises the question of whether there are possible conflicts of interest between men and women, kin and non-kin regarding the continuation of FGMC. Evolutionary studies of alloparenting have emphasized the fitness benefits of ensuring health and well-being of daughters (versus daughters in law), due to their daughter's children genetic relatedness being more certain, but also their daughter's capacity to raise their relatives' children as well as their own (Perry and Daly 2017). While informative data is scarce, to date there is no conclusive evidence of conflict between men and women or among their kin on FGMC preferences,

indicating that the costs and benefits of cutting may be equivalent (Gibson and others 2018). Parents may indeed be more concerned with controlling the sexual fidelity of daughters-in-law (than daughters), but they also seek to ensure their daughters gain entry to the marriage market (in contexts where marriage is tied closely to women's future economic security). Indicative of this, the Arsi Oromo of Ethiopia express equal levels of support for cutting daughters-in-law versus daughters, however men are more inclined to openly admit their support of FGMC in daughters, possibly because they are signalling their marriageability to potential in-laws (Gibson and others 2018).

Obtaining accurate data on FGMC prevalence and support for the practice represents an ongoing methodological challenge for studies in this area. In the absence of clinical data, most FGMC data is collated from self-report demographic surveys which are exposed to reporting biases. As a sensitive topic, people may be inclined to hide their true attitudes and practice when questioned directly. Evolutionary behavioural scientists are well-placed to address both the theoretical and methodological challenges of FGMC research, and a number of recent studies have started to do this by applying indirect questioning methods (e.g. unmatched count techniques, and implicit association tests) (Vogt and others 2016; Gibson and others 2018; 2019). Such methodological innovations hold great potential to provide new insights on hidden attitudes and intentions which may underpin the practice.

The Challenges of Applied Research

In this chapter, we have identified how evolutionary anthropological scholarship on population health issues is relevant to international development sector policy. While we have selected HCPs as a case study, the potential applied value of evolutionary research to population issues is far-reaching (Gibson and Lawson 2014, 2015; Tucker and Rende Taylor 2007; see also Wells and others 2017). To make research truly "applied", however, requires transforming policy relevance into real impact, a formidable challenge shared by other branches of applied social science. How best can rise to this challenge? In attempt to answer this important question, we conclude with three recommendations for evolutionary researchers who wish to make their work more applied.

First, we must make bold new steps to improving communication and collaboration with appropriate decision-makers, including national policy-makers, research think tanks and non-governmental charities. These organisations can help to guide our research towards the most pressing human issues, but also have the power to implement and, most importantly, effectively evaluate our recommendations. This can be achieved via arranging working groups and interdisciplinary conferences/workshops (e.g. Lawson and Gibson 2018b), but also in presenting our work in policy reports and other non-academic forums (for example to policy-makers in government (Gibson, 2018) and multilateral UN organisations (Gibson 2019). Direct access to the key policy decision-makers, government officials and other influential people is never easy, therefore we must find ways to ensure our results are accessible. We can do this by being more explicit in drawing out the policy implications and recommendations arising from our work (particularly in writing abstracts and concluding statements); using plain language in our key summaries; and, when possible, ensuring that this work is freely available online. Effective use of social media, and online platforms, including research blogs and editorials where content is produced directly by researchers, also offer novel opportunities to reach broader audiences and

network with key policy-makers, advocacy groups and NGOs (e.g. Lawson & Schaffnit 2019). These platforms can furthermore be used to address and correct any misreading of our work. Throughout academia there is an increasing emphasis and interest in communicating to the wider public via journalists in the press. This can present particular challenges for evolutionary-based researchers due to common misunderstandings of evolutionary approaches (Gibson and Lawson 2015). To avoid the oversimplification and sensationalism of work within the media, we need to be actively involved in communicating directly with journalists, either in writing press reports or answering queries in press briefings, through social media, radio or television. Policy (like science) is ultimately not only driven by research, but also governed by dominant ideas, agendas and politics of the time.

Second, evolutionary behavioural scientists must do more to prioritize equitable collaboration, not just with study communities via participatory research methods, but with national research centres and academics in lower and middle-income countries (Urassa and others, 2021). Despite long-standing commitments to decolonizing research within anthropology, research within the global health sector, where "nothing about us, without us" has become a popular sentiment, is ahead of the game in this respect (Boum and others, 2018; Abimbola and others 2019). Several prominent global health journals now actively discourage manuscript submissions where primary data has been collected without explicit collaboration and co-authorship with local researchers; a stance, to our knowledge, not shared by any social science journal (Urassa and others, 2021). As such, avoiding "parachute" fieldwork is not just a vital matter of research ethics, but entirely necessary if applied evolutionary demographers and anthropologists want to engage fully in contemporary debates of international development policy and practice. There is also a scientific case for equitable collaboration, since diverse and inclusive research teams stimulate innovation and open up greater pathways for research impact (Alshelbli and others 2018). Global research partnerships also promise the benefits of a robust international research ecosystem, more capable of responding to unexpected change — as illustrated by disruption of many field-based research programs following the travel restrictions of the COVID-19 pandemic (Urassa and others, 2021).

Appropriate actions will vary by context and topic of study, and are not limited to issues of co-authorship, which may be challenging to achieve in some contexts (Urassa and others, 2021). Researchers must also avoid "token" assignment of authors to manuscripts in place of growing truly equitable partnerships defined by genuine intellectual collaboration. Inundating researchers with requests to collaborate will be counterproductive if requests are not both backed financially and approached flexibility. Nevertheless, authorship, and access to it, is a discernible indicator of collaboration and provides required opportunities for professional networking, career progression, and academic independence for those included (Abimbola and others 2019). More generally, we strongly encourage researchers to campaign for institutional change (e.g. greater opportunities for international-level grant sharing), and to commit to individual actions prioritizing good practice. This includes increasing awareness of appropriate local institutions and scholars in the countries we work, teaching students about the dangers of extractive research, developing cross-national mentoring arrangements, and rewarding the time and effort required to forge equitable collaborative partnerships as reviewers in recruitment, promotion and funding allocation decisions. We emphasize that ostensibly altruistic activities, like paying for schoolbooks, medical care or aid in times of shortfall, are all important and

beneficial activities characteristic of many long-term field programs managed by high-income country researchers. Yet we must be attentive to the fact that such activities rarely foster national academic capacity and research infrastructure, which are too often left dependent on external expertise with limited opportunity for independent growth (Urassa and others, 2021).

Third, we encourage that more evolutionary-based research is directed towards transitional populations, particularly those most vulnerable to the effects of rapid and recent changes in society and health. Indeed, transitional populations are among those facing the greatest social and health challenges, linked with rising inequality, climate change, and growing demands for food, employment, and public services. Evolutionary anthropology has historically prioritized the study of populations considered similar to those of the evolutionary past (foragers and small-scale, high-fertility, high-mortality subsistence economies). A focus on nonindustrial populations has proved essential for testing evolutionary predictions about human behaviours; providing important insights into how our ancestors lived, and data to test hypotheses about the processes underpinning important behavioural shifts across human history, such as the agricultural revolution. Yet, while essential, a focus on the most "traditional" of human societies in today's world, also means a focus on the most exceptional and non-representative societies from a policy perspective. To maximize our relevance and potential to draw conclusions with wide-ranging policy implications, we must expand our repertoire to transitional populations and consider reactions to market integration, dietary and lifestyle shifts and related changes.

Focusing on contemporary communities in transition also enables us to develop a clearer understanding of important and often controversial issues in evolutionary studies, among them adaptive lag (i.e. when adaptations become outdated by rapid environmental change), decision-making in uncertain environments, and the dynamics of cultural change (Mattison and Sear 2016). Indeed, this review has focused on functional approaches in evolutionary anthropology (i.e. drawing primarily on the framework of human behavioural ecology, Nettle and others 2014) — but evolutionary scholars are also interested in how norms and behaviours are socially transmitted (Mesoudi 2011; Ross et al., 2016; Boyd and Richerson, 1985). Fully understanding the origin, spread and persistence of cultural practices, in particular those that have harmful consequences, will necessarily require an appreciation of the dynamics of social influence. A major strength of an evolutionary approach is that it provides a uniquely holistic framework capable of working across different levels of explanation (proximate and ultimate). By now turning our attention to applied topics, there is great potential to inform population health science and bring fresh insights to the design, critique and evaluation of contemporary development policy.

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30. The Biodemography of Human Health in Contemporary Non-industrial Populations: Insights from the Tsimane Health and Life History Project

Michael Gurven, Hillard Kaplan, Benjamin Trumble and Jonathan Stieglitz

The life history of human hunter-gatherers is characterized by an extended post-reproductive lifespan, prolonged juvenile growth, high fertility with multiple dependents and biparental care, and extensive intra- and inter-generational resource and information transfer. Long-term, in-depth study of contemporary non-industrial populations offers important glimpses into how these traits operate, and insights into how they may have evolved. The Tsimane Health and Life History Project is a large-scale bio-behavioural study of the human life course designed to help understand the bidirectional connections between life history, health and social behaviour in a high fertility, kin-based population lacking amenities of modern urban life. It seeks to document the epidemiology of health and mortality across the lifespan, and to understand how growth and investment, social structure, sharing networks and behaviour impact health and aging. It focuses on how pathogen burden influences health and well-being during development and adulthood, and addresses how modernization affects health and sociality. We reflect on the implications of current findings and highlight the need for more joint ethnographic and biomedical studies of subsistence populations to address unresolved questions not only in evolutionary anthropology or biodemography, but in public health, epidemiology, gerontology and medicine.

Introduction

Average human life expectancy has increased by almost three months per year over the past 160 years, surpassing 70 years well into the twenty-first century (UN, 2015). Improvements in sanitation, nutrition, and public health account for much of this change (Riley, 2001, Oeppen and Vaupel, 2003). Reductions in infant and child mortality have greatly increased life expectancy, and chronic degenerative diseases have become the main sources of morbidity and mortality in industrialized populations. In fact, it is commonly reported that chronic diseases of aging were rare or absent throughout much of human evolutionary history. These afflictions of industrialized society are viewed as examples of evolutionary "mismatch" due to rapid environmental and lifestyle changes (i.e. "modernization") outpacing our evolved genetic

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heritage. According to this view, the widespread prevalence today of cardiovascular disease (CVD), type 2 diabetes mellitus (T2DM), Alzheimer's Disease, and other degenerative diseases (e.g. osteoporosis) result from our being "Stone Agers in the fast lane" (Eaton et al., 1988).

Here, we introduce a detailed case study to help improve understanding of the processes that shaped the evolution of the human life course, with a focus on health and aging. What does physical condition, health, biological aging and disease look like in non-market subsistence contexts more similar to how we lived prior to industrialization? How can the study of aging, health and social lives in remote rural populations provide insight into what ails (or does not ail) us in contemporary urban settings? While it would be useful to study multiple populations over evolutionary time, comparable fine-resolution data on health and aging do not exist for past populations, and thus data from extant modern groups offer an imperfect lens to view the past. The demography and life history of extant foraging populations was spearheaded by pioneering research among Dobe !Kung of Botswana and Namibia (Howell, 1979, Howell, 2010, Lee and DeVore, 1976), the Ache of Paraguay (Hill and Hurtado, 1996) and Hadza of Tanzania (Marlowe, 2010, Blurton Jones, 2016). These and other vital empirical studies conducted among non-industrial populations identified universal features of the evolved human life history: compared to other mammals, and even primates, an extended post-reproductive life span, high fertility with multiple dependents, delayed juvenile growth, extensive intra- and intergenerational resource transfers and cumulative culture — in concert with the co-evolution of a highly encephalised brain (Kaplan et al., 2000, Hill et al., 2009, Kaplan, 1997) (see Kramer, in this volume). The exact timing and context for the evolution of these traits remain difficult to ascertain, but the long-term study of contemporary non-industrial populations with limited access to modern amenities (e.g. sanitation, electricity) is one approach to better understand this human adaptive complex.

The Tsimane Health and Life History Project (THLHP) is one such endeavour designed to integrate traditional ethnography with advances in methods and concepts from other disciplines, including demography, biomedicine, gerontology, epidemiology, economics and psychology in a subsistence population of forager-horticulturalists in lowland Bolivia. By examining changes in physical growth, health, development and aging in relation to economic productivity, resource transfers and social networks, we seek to test competing models meant to explain the evolution of our long human lifespan and associated traits (Hawkes, 2003, Kaplan et al., 2000). This task, by nature, demands an inter-disciplinary, mixed method approach. A classic expectation from the branch of evolutionary biology known as life history theory is that low exogenous mortality acts as a prime driver shaping a slower life history — that is, prolonged maturation, greater energetic investments in somatic maintenance, and longer lifespan (Stearns, 1992). A more realistic approach treats mortality as endogenous and co-evolving with other life history traits, such as the role of learning in development (Kaplan and Robson, 2002). The learning-intensive human foraging niche shifted an already slow life history further in this direction, but numerous questions remain: How did increased investments in learning and mortality reduction co-evolve, and how are they related to mortality-reducing effects of human sociality, risk buffering and cumulative culture? How did ecological shifts impact human food production and sharing, risk preferences, and mating patterns? How do different sources of morbidity, such as exposure to a diverse array of pathogens and co-morbidities, impact growth rates, somatic maintenance costs, and aging? To what extent are human-specific traits a coordinated and coevolved bundle, versus a mix of adaptations and by-products? Addressing these questions requires careful

study of *directly observable* patterns of behaviour, health and human-environment interactions, which is only possible by studying contemporary, small-scale subsistence populations.

Joint behavioural and biomedical inquiry among contemporary non-industrial societies like the Tsimane aids in reconstructing ancestral patterns of human aging, health, life history and sociality. It also provides insight into the respective roles that changing diets and other lifestyle characteristics (e.g. physical activity) play in affecting health. A broader range of societies is required to understand evolved human reaction norms across different environments over recent millennia. Subsistence horticulturalists like the Tsimane share many similarities with existing full-time hunter-gatherers: they also hunt, gather, and fish, exhibit natural fertility, have minimal access to modern sanitation or medicine, and show limited group size. The differences between foragers and horticulturalists like the Tsimane, however, can shed additional light on the impacts of plant and animal domestication on health and life history-relevant traits, including parasite burden, nutritional status, fertility, mobility, residence patterns and social structure. Bottlenecks and expansions of human populations during the advent of agriculture also had profound impacts on human population genetics (Hawks et al., 2007, Fumagalli et al., 2011, Karmin et al., 2015), further highlighting the importance of non-foragers when considering health and disease in contemporary populations.

"Modernization" (defined here as a trend toward greater participation in the market/cash economy) affects health and reshapes aspects of social ecology, but the mechanisms by which these changes occur are not well understood. Contemporary non-industrial populations like the Tsimane are experiencing rapid social, political, economic and cultural change. Socioeconomic transformation due to increasing access to cash economies, wage labour, schooling, sanitation, access to modern medicine and other amenities (e.g. savings accounts) adds layers of complexity to understand how changing conditions alter health, risk management and life histories. Careful study is necessary for evaluating whether chronic diseases of aging, such as CVD, T2DM, osteoporosis and Alzheimer's disease, were common during human evolutionary history or are fairly novel, resulting from an evolutionary mismatch between our evolved heritage and rapid environmental and lifestyle change. Addressing complex questions of how modernization influences health and sociality requires long-term study of an appropriate reference group, in this case, small-scale subsistence populations with relatively limited access to modern markets.

In this chapter, we first describe the Tsimane population, and then introduce the goals and organization of the Tsimane Health and Life History Project (THLHP). The Tsimane are a useful case study to describe various aspects of physical and mental health in a subsistence population, in relation to other life history, economic and demographic patterns, and in marked contrast to industrialized, urban populations. The Tsimane are now beginning to experience an epidemiologic transition, where the overall burden of disease currently dominated by infectious sources of morbidity, may soon be replaced by chronic, degenerative diseases. We thus highlight the role of infection in shaping different aspects of health, including several chronic diseases.

The Tsimane of Bolivia

Bolivia is home to 36 indigenous groups which together constitute over 60% of the population (INE, 2012). Of the 30 groups inhabiting the tropical lowlands, the Tsimane are among the most isolated (along with the Yuqui, Araona and Siriono). The Tsimane are forager-horticulturalists of the Bolivian Amazon who subsist largely from slash-and-burn horticulture, including plantains, rice, sweet manioc and corn (Figure 1). They also fish in rivers, streams and lagoons, hunt a

large array of neotropical mammals, and engage in seasonal gathering of fruits and other foods (e.g. honey, nuts). They inhabit over 90 villages numbering from 50 to 500 individuals along the Maniqui, Quiquibey and Mato Rivers and interfluvial *terra firma* (Figure 2). While early censuses in the late 1990s estimated a population size of 6,000 Tsimane, the most recent complete THLHP census in 2015 suggests closer to 16,000 (also see INE (2012)) and a population growth rate of over 3.5%. Unlike most extant foragers, the relatively large Tsimane population size provides the opportunity for study of all stages of the human life course, including late adulthood, which is essential to study competing models of human life history evolution and diseases of aging.

Fig. 1 The Tsimane of central Bolivia. A central feature enabling delayed childhood, high fertility and long life span is extensive sociality — manifesting in cooperative production, distribution and childcare. Photo credits: Michael Gurven.

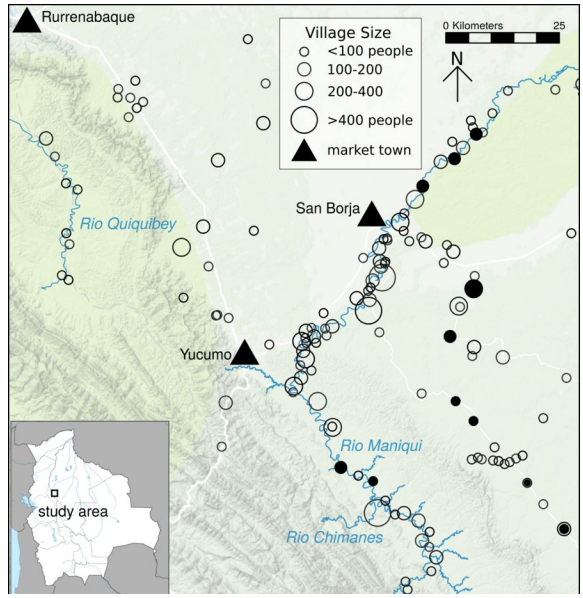

Fig. 2 Map of Tsimane territory and study villages. Solid circles signify "core" villages where relatively long-term study has occurred, empty circles are other villages visited by the biomedical team. Triangles reflect towns. Size of circles are proportional to village census size.

Throughout the first half of the twentieth century, Tsimane maintained a traditional lifestyle due to the relative absence of navigable roads in their territory. While road and river access has improved due to logging, development projects and new technologies (e.g. the recent boom of *pequi* outboard boat motors in the last five years), market access remains somewhat limited for many villages, especially during certain periods of the year when heavy rains wash out bridges and dirt roads, and make river travel dangerous. This variable access to the market and associated non-traditional cultural influences serve as a quasi-experiment — an opportunity for examining effects of socioeconomic change on health, fertility and social behavior (Gurven, 2012, Trumble et al., 2015, Gurven et al., 2012, Gurven et al., 2013).

Production and Reproduction

Total fertility rate is higher than most hunter-gatherers (nine births per woman), and the production and sharing network is multi-generational. Tsimane produce less food than they consume until late adolescence (Gurven et al., 2012, Hooper et al., 2015). Thus, the caloric burden on families can be substantial, especially for younger parents with multiple highly dependent offspring. For example, a married woman age 33 is at her peak dependency of 4.3 expected children younger than age 15. Food production efficiency peaks in the 30s-40s, especially for hunting and other difficult, skill-intensive activities (Gurven et al., 2009). Peak productivity extends long beyond peak strength (Gurven et al., 2006), suggesting the importance of skills-based practice and learning. Though delayed productivity is clear for hunting (Gurven et al., 2006, Walker et al., 2002), expertise in a wide range of production, manufacturing and other tasks (e.g. childcare, conflict mediation) is reported most frequently among middle-aged or older adults (Schniter et al., 2015). Nuclear families provide much of the daily calories, with older adults, including

parents, grandparents and siblings providing substantial amounts of food to younger kin (Hooper et al., 2015). As strength and functional ability decline in later adulthood, Tsimane shift emphasis toward low strength and high skill subsistence and political activities, including hook-and-line fishing and horticulture, conflict mediation, village leadership roles and storytelling (Schniter et al., 2015). Older adults are also actively involved with various forms of pedagogy. Though caloric production declines at later ages, cognitive abilities remain relatively intact through the seventh decade of life (Gurven et al., 2017), helping to facilitate knowledge transfer.

Food sharing is widespread within extended families, but is more limited in scope than typically described among foragers. Kinship and relative need, as determined by recipient age, productivity and family size, and health status, largely determine the magnitude and direction of resource flows (Hooper et al., 2015, Gurven et al., 2012). Informal exchange networks help Tsimane manage multiple risks like sickness and injury, in addition to those from daily food shortfalls (Gurven et al., 2012, Jaeggi et al., 2016). The "prices" implicitly negotiated in these informal exchange networks partly reflect individual differences in supply and demand, which itself relates to household needs and abilities.

Whereas our nearest primate relatives, chimpanzees, show a rate of reproductive decline that is more closely linked to somatic decline and increasing risks of mortality, human reproductive aging precedes somatic senescence by roughly 20–25 years (Wood, 1994). After ceasing to reproduce, both men and women provide net economic transfers to children and grandchildren. By the time they reach their 70s, Tsimane rarely give food away, and so contribute less to sharing networks (Figure 3). The time delay between unproductivity due to physical deterioration and death appears to be short. During periods of productive decline, older adults may help in other ways. However, given this pattern of productivity and transfers, further delays in the age at menopause would produce net economic deficits within families because older adults would not be able to produce enough food for their own offspring (Kaplan et al., 2010).

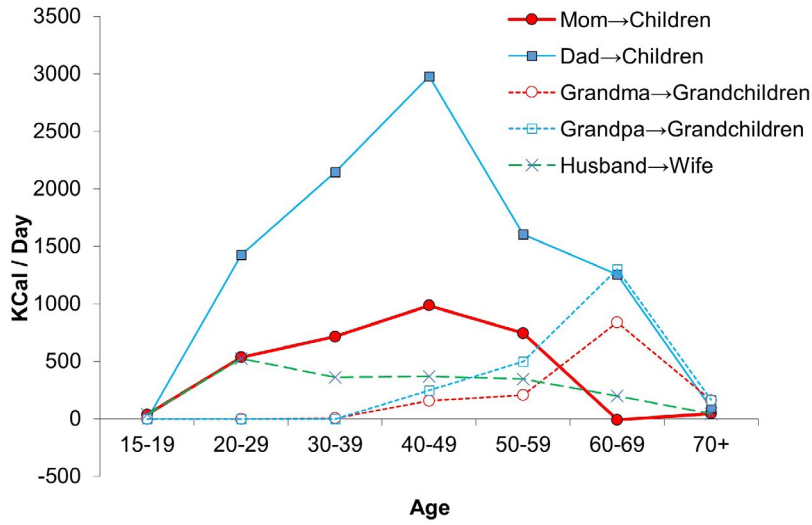

Fig. 3 Kin-based resource flows. Net contributions of food (measured in kilocalories transferred per day) from parents to children, from grandparents to grandchildren and husband to wife by age. Data come from over a thousand interviews on production and sharing behavior, and spot observations of consumption patterns.

Tsimane Health and Life History Project (THLHP)

From its inception in 2002, the research design of the THLHP has included a mobile biomedical team comprised of Bolivian physicians, biochemists, and Tsimane research assistants that visits each sampled village annually or once every other year to provide broad snapshots of physical condition and health, demography and socioeconomic life (see Gurven et al., 2017). A reduced "core team" composed of a few anthropologists and Tsimane research assistants complements this mobile team through more focused, longer-term sampling and more intensive socioeconomic data collection in core villages. These extended field sessions in core villages provide rich ethnographic study of economic and social behavior, and health. Our initial sample included 18 villages, expanded to 23 by 2005, 85 by 2009, and 90 by 2015 (Figure 2).

Our holistic bio-behavioral approach has the advantage that we can link information on multiple phenotypes for the same individual over time to better understand factors influencing aging, health and sociality. Our team has collected systematic baseline data at the individual level on many traits: demographic, behavioral, morbidity, biomarkers related to health and aging, infectious exposure, inflammation and other indicators of immune function, and measures of physical and functional status (see Gurven et al., 2017). The measures are derived from observation, surveys, medical exams, and biospecimens (blood, feces, urine, saliva). Several THLHP protocols were modified from prior life course epidemiological studies in high income countries (e.g. NHANES, Mexican Health and Aging Study, MacArthur Aging Study), permitting direct cross-cultural comparisons.

New THLHP findings replicate those from earlier studies of foragers and forager-horticulturalists: Tsimane exhibit a skill-intensive economic niche with all the hallmarks of our evolved life history, including a long learning period of juvenility and adolescence, biparental care, socially-mediated risk-buffering, multi-generational resource transfers by parents and grandparents to dependent young, high productivity of post-reproductive adults until the 8th decade of life, and a long adult lifespan. New research has enriched our understanding of the life history of production, consumption and cooperation, and shed new light on a variety of health-related themes: (a) lifespan, aging and psychological well-being, (b) the role of infectious disease in shaping different life history components including growth, senescence and fertility, (c) chronic disease and lifestyle change. In what follows, we discuss each theme in turn.

Lifespan, Aging and Well-being

From the period 1950–89, life expectancy at birth (e_0) among Tsimane was 43 years; by 2002, e_0 increased to about 53 years (Gurven et al., 2007) (Figure 4a). Despite recent improvement, Tsimane death rates at all ages are similar to those observed in Europe in the 1800s (Gurven et al., 2008). Infection is the largest cause of mortality, responsible for about half of all deaths, and a loss of almost 12 years of life expectancy at birth (Figure 5). Unlike many patterns observed historically, where initial increases in lifespan are largely due to reductions in infant and child mortality, the improvement in e_0 from 1990–2002 was more a result of reduced death rates in adulthood than among infants or children (Figure 4b). We suspect that this is due to differences in access to medical interventions for adults and older children, since they have a greater ability than infants or young children to seek and survive treatment. Despite recent improvement in access to health care facilities, Tsimane cultural beliefs about sickness and death, coupled with some ethnic discrimination in town may still deter people from seeking treatment. Modal age of adult death

is 70 years (SD=6.3), similar to that observed among hunter-gatherers and other horticulturalists, and 15 years earlier than that observed in high-income countries (Gurven and Kaplan, 2007).

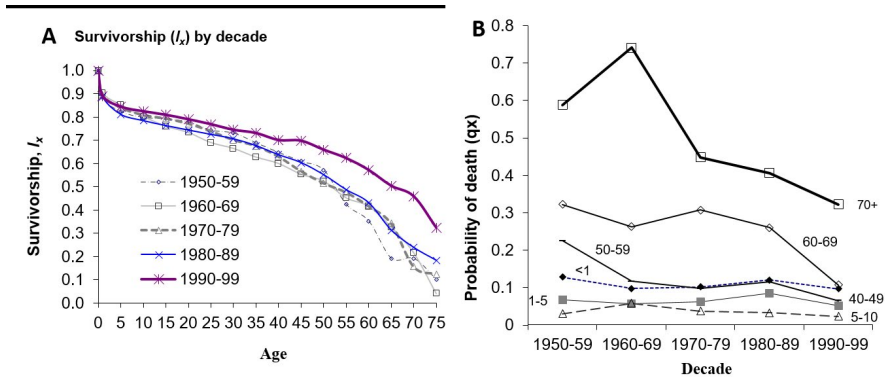

Fig. 4 Tsimane period mortality. (a) Tsimane survivorship (l_x) by decade, spanning period from 1950–1999; (b) Mortality rates (q_x) by decade for same time period for selected age groups. Late age mortality declined more substantially over the period 1950–99 than infant and child mortality. For details on mortality methodology, see Gurven et al. (2007).

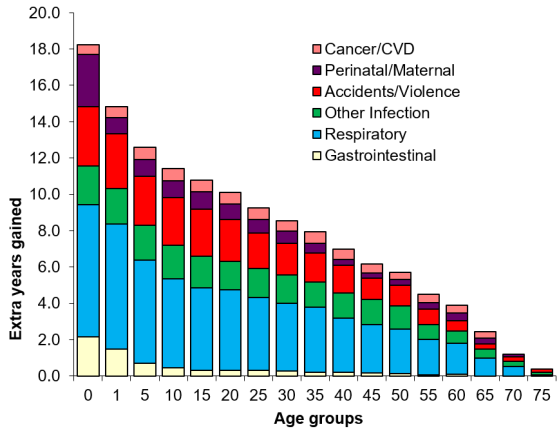

Fig. 5 Effect of mortality cause elimination on remaining life expectancy at age x (e_x) during the period 1950–89, when e_0 was 42 yrs. Using single decrement life tables offers a glimpse of the absolute increase in e_x expected by eliminating six macro-causes of death. Causes of death were assessed by verbal autopsy.

By age 60, Tsimane show evidence of significant physical disability. Physical strength declines continuously by the fourth decade of life (Gurven et al., 2006). Over 60% of Tsimane over age 60 complain about hearing loss, over 80% have trouble seeing close distances, and over 70% can no longer chop large trees in their fields. About 50% of men and 70% of women over age 70 can no longer walk long distances, and complain frequently about painful arthritis in their legs, back, and hips. Over 70% of men no longer hunt by age 70; these men complain about weakness, lethargy, and having poor eyesight and hearing. Functional disability is a strong predictor of Tsimane depression: adults aged 50+ in the top decile of a composite disability measure score 14% higher on a depression scale than those in the bottom decile after controlling for

multiple potential confounders (Stieglitz et al., 2014). We find that depression increases with age as disability increases and limits production and sharing ability (Stieglitz et al., 2014). This observation runs counter to the common claim that human depression is a modern mismatch disease, or that it universally peaks in mid adulthood (i.e. "mid-life crisis") (Weiss et al., 2012).

Fluid cognitive abilities related to reasoning and processing speed also appear to decline in late life from their peak in early adulthood, whereas crystallized abilities based on cumulative experience and knowledge increase throughout the lifespan (Gurven et al., 2016, Trumble et al., 2015). While this pattern has been widely documented in Western contexts, it had never before been assessed systematically in a non-literate or non-industrial population. While the decline in fluid abilities seems to mirror changes in physical abilities with age, non-declining crystallized abilities are consistent with the functional role of middle-aged and older adults as mentors, instructors, and caregivers in Tsimane society. We also find that older Tsimane with the apolipoprotein E4 are protected against decline in cognitive performance, but only among those with heavy parasite burden (Trumble et al., 2017); E4 carriers without parasites and non-E4 carriers with parasites showed lower cognitive performance (Figure 6). These findings suggest that the E4 allele, the strongest risk factor for Alzheimer's Disease and cognitive decline in industrialized populations, might have fitness-relevant advantages in a more infectious environment.

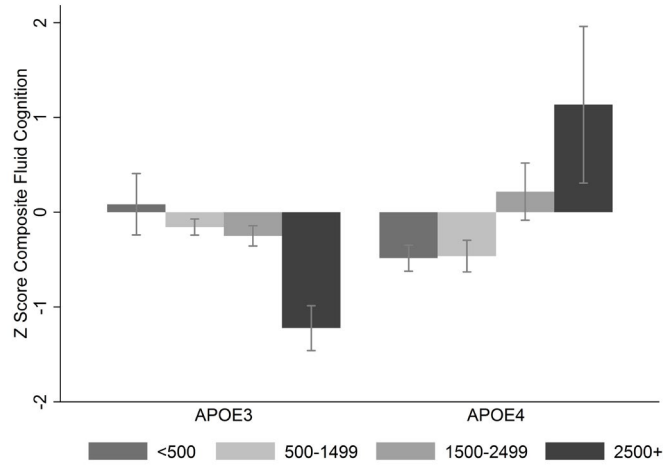

Fig. 6 Cognitive performance, apolipoprotein-E (APOE) allelic variation and parasitic infection. Predicted z-scores for composite fluid cognitive performance (n=242 adults aged 30+). APOE alleles include E3 and E4; parasite infection proxied by four levels of eosinophil count ("normal" <500/uL, "mild" eosinophilia 500–1499/uL, "marked" eosinophilia 1500–2499/uL, "very high" eosinophilia ≥2500 /uL). Model controls for age, sex, years of schooling, Spanish fluency, and community ID as a random effect. See Trumble et al. (2017) for more details.

Despite the belief that depression is largely a modern ailment unique to industrialized populations characterized by high inequality, intense social competition and eroded family ties (Nesse, 2000), Tsimane adults frequently report symptoms of persistent sadness that interferes with routine daily functioning. We find that depression in older adults is associated with reduced energetic status, greater physical limitations, reduced subsistence involvement and greater social conflict (Stieglitz et al., 2014), consistent with a human life history perspective emphasizing the importance of adult economic production surplus and downward net

transfers. We also find that emotional, cognitive and somatic symptoms of depression are each associated with greater immune activation (i.e. pro-inflammatory cytokines), both at baseline and in response to *ex vivo* stimulation (Stieglitz et al., 2015) (Figure 7). This result is consistent with depression serving as a type of "sickness behavior" geared towards conserving energy to aid in immune defenses against infection. In Western populations, the association between immune activation and depression has instead typically been interpreted to reflect reduced cellular immunity, and immune dysregulation due, in part, to reduced pathogen exposure during child development.

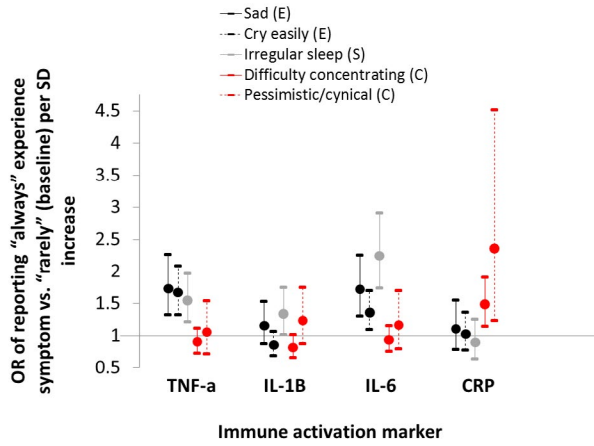

Fig. 7 Depression symptoms and immune activation. Odds ratio (OR with 95% CI) of reporting always vs. rarely experiencing a symptom per standard deviation unit increase in cytokine or lnCRP concentration. Symptoms are categorized by whether they include emotional (E, black), somatic (S, gray), or cognitive (C, red) components. Estimates derived from multinomial logistic regression controlling for age, age2, sex, body mass index and geographic region of residence. See Stieglitz et al. (2016) for more details.

In summary, mortality rate increases in late adulthood are linked to changes in physical condition due to aging and associated declines in muscularity and strength, aerobic fitness, sensory acuity, cognitive performance and immune function. Those changes, in turn, are linked to changes in economic productivity and psychological well-being. The productivity of Tsimane adults supports net economic transfers to descendants until the eighth decade, coinciding with the modal age of adult death observed among Tsimane and other subsistence populations (Gurven and Kaplan, 2007). These findings demonstrating declining fitness-related utility at late ages are compatible with the "disposable soma" theory of aging, which views aging as the result of compromised energy allocation favoring investments providing fitness benefits earlier in life (e.g. direct reproduction) in light of somatic maintenance costs that increase with age (Kirkwood and Westendorp, 2001). However, despite speculation, it has still not been clearly determined why humans live as long as they do, but not longer. One possibility is that the fitness benefits of costly investments in grandchildren outweigh the fitness costs of slowing down the aging process, but that these benefits diminish once grandchildren are past the high mortality period early in life. Given dispersal, migration and the dilution of genetic relatedness with each successive generation, the sum of fitness effects over all descendants may be too small to favor further delays in aging.

Pathogens and Life History

Throughout history, human populations were exposed to an array of pathogens, many of which were common to other wild primate species (Nunn et al., 2004). Ancestral humans may also have been exposed to additional pathogens due to the consumption of meat and fish (Finch and Stanford, 2004). Phylogenetic evidence for several pathogens, including smallpox, *Plasmodium falciparum*, and *Mycobacteria tuberculosis* suggests a pre-agricultural history of exposure (see review in Pearce-Duvet, 2006, Bos et al., 2014). Sexually transmitted diseases also likely have a long evolutionary history among humans (Donovan, 2000). Antibodies to viral infections, such as herpes simplex, Epstein-Barr and varicella-zoster virus (VZV) have been documented in isolated Amazonian groups, along with cytomegalovirus (CMV), intestinal helminths, herpes simplex viruses, hepatitis B and arboviruses (Black et al., 1970, Salzano and Callegari-Jacques, 1988).

Helminths, or intestinal worms, have coexisted with humans for millennia and represent a major feature of early human disease ecology (see Hurtado et al., 2008 for review). Non-human primates are widely infected with helminths, and infection with multiple species of soil-transmitted intestinal parasites has been documented in remote Amerindian populations (Lawrence et al., 1980, Confalonieri et al., 1991, Hurtado et al., 2008). Macro-parasites such as *Enterobius vermicularis* (pinworm) and hookworms (*Necator americanus* and *Ancylostoma duodenale*) have been discovered in coprolites from 7–10kya (Fry and Moore, 1969, Gonçalves et al., 2003). Throughout human history, helminth burdens have fluctuated, but it is likely that the absence of helminths is a very recent occurrence specific to industrialized, urban environs. Helminths have complex life cycles within human hosts, passing through numerous host tissues, and with intricate survival strategies that involve not only thwarting host immunity, but also competing with other helminths for host resources and creating a favorable niche by host manipulation (Maizels et al., 2004, Blackwell et al., 2013). This long history suggests that human immune systems have co-evolved with helminths and may occasionally produce maladaptive outcomes under the novel, mismatched conditions introduced in recent human history.

Tsimane exhibit high rates of diverse infections. Over 66% of Tsimane have at least one intestinal parasite, the most common being hookworm (*Ancylostoma duodenale* or *Necator americanus*, prevalence 56%), roundworm (*Ascaris lumbricoides*, 15%) and whipworm (*Trichuris* sp., 4%) (Blackwell et al., 2011). Protozoan infections are also common, including *Giardia lambia* (30%), and *E. histolytica* (5%). About half of men and women have anemia, with children and adolescents showing the highest risk (56% of girls, 63% boys). Polyparasitic co-infections are common. Several helminth species co-occur, whereas helminths such as hookworm and roundworm appear to have protective effects against giardia infection (Blackwell et al., 2013). Helminths also affect Tsimane fertility: hookworm is associated with reduced fertility, while roundworm is associated with shorter interbirth intervals, perhaps by increasing maternal immunological tolerance for a fetus (Blackwell et al., 2015). Given the high transmission rate of multiple pathogens in the Tsimane environment, the prevalence of several parasites and energetic investments in immune defenses against them (e.g. immunoglobulin-E, IgE production) peak earlier in childhood than in populations with lower transmission rates (Blackwell et al., 2011). This finding is consistent with the "peak shift" hypothesis, which suggests that in more infectious environments, immune defenses develop earlier and thus peak incidence rates occur at younger ages.

Perhaps as a consequence of earlier and consistent immune responses to diverse pathogens over the life course, several components of adaptive immune function show evidence of rapid

senescence. Adaptive immunity refers to humoral and cell-mediated immune components that help build immunological memory to specific pathogens in order to mount an effective response upon re-exposure. Naïve CD4+ helper T-cells, essential for mobilizing immune defenses against unfamiliar pathogens, are considerably depleted by age 50, while natural killer cell counts are substantially elevated (Blackwell et al., 2016). Consistent with these patterns, a measure of epigenetic age acceleration — based in part on the estimation of immune cell counts — is higher among Tsimane relative to other populations (Horvath et al., 2016).

Indeed, infections are the main source of Tsimane morbidity and mortality over the life course (Gurven et al., 2007). Gastrointestinal illness and respiratory infections are frequent diagnoses: 30–40% of infants and young children suffer from each; 30–40% of adults suffer from gastrointestinal illness and 20–30% from respiratory infections. Living in a pathogenic environment likely favors pro-inflammatory (e.g. C-Reactive Protein [CRP], Interleukin-6 [IL-6]) alleles (Vasunilashorn et al., 2011), and higher levels of inflammation than in more hygienic environments. Levels of one indicator of inflammation, CRP, are higher than among Americans, especially in childhood (Blackwell et al., 2016). Cross-sectional estimates of life lived with high CRP indicate that by age 34, Tsimane have spent an average of 15 years (42% of life) with high CRP, compared to 6.8 years (19%) in the US. Tsimane CRP levels in early life are higher than those sampled among diverse populations, including Italians, Mexicans, Filipinos, and Native Americans in the USA (Gurven et al., 2008). CRP levels vary between and within individuals, with half of the total variance being between individuals; elevations thus likely do not represent only acute infections, and instead are moderately stable within individuals over time (Blackwell et al., 2016). In contrast to the geographical distribution of some sexually transmitted infections like trichomoniasis (where Tsimane living near town have higher prevalence than those living farther from town) (Stieglitz et al., 2012), Tsimane living farther from town show higher CRP levels than those living near town, suggesting higher exposure to other infectious ailments in remote villages. Other biomarkers also suggest high levels of immune activity throughout life: Tsimane have higher levels of white blood cells, erythrocyte sedimentation rate, B cells, and natural killer cells than Americans at all ages (Blackwell et al., 2016). On average, 20% of Tsimane white blood cells are eosinophils, consistent with high levels of parasitic infection, compared with the US reference range of <5%. Antibodies related to infection are also much higher among Tsimane: immunoglobulin-G (IgG) is about twice as high, and IgE, most relevant for helminthic infection, is about 100 times higher than typical US levels (Blackwell et al., 2011).

Perhaps as a consequence of high levels of infection and immune activation, Tsimane show elevated resting metabolic rates, with 10–15% of metabolism associated with immune activation (Gurven et al., 2016). The high prevalence of infection, and requisite energy shunted towards immune defenses, may help explain the slow somatic growth and stunting common among Tsimane and similar energy-limited populations experiencing high pathogen burden (Blackwell et al., 2017). Population differences in growth trajectories during childhood may reflect patterns of pathogen exposure and immune investment, since Tsimane show slower growth during periods of their early peak IgE production (Blackwell et al., 2011). The higher energetic cost of tolerating and/or defending oneself from parasites may be further offset by other shifts in energy use. Possibilities in the Tsimane context include lower physical activity, sickness behavior (Stieglitz et al., 2015), cachexia and osteopenia (Stieglitz et al., 2015), dyslipidemia and anemia (Gurven et al., 2016, Straub et al., 2010).

In summary, infectious disease has multiple phenotypic consequences. Not surprisingly, it appears to upregulate immune activity, resulting in greater energy expenditures throughout life and more rapid senescence of some immune cell populations. Variation in pathogen burden across human environments, and over time within individuals, seems to be associated with adaptive and plastic immune responses. The Tsimane, living in a warm, humid and tropical environment in relatively settled communities, may experience greater pathogen burden than other contemporary and ancestral human populations in drier, colder, or more mobile environments (Page et al., 2016). Yet their modal age at death is similar to that of subsistence-level populations living in drier environments (Gurven and Kaplan, 2007). Perhaps natural selection on human aging has resulted in a species-typical lifespan, despite sources of morbidity and death differing across populations.

Chronic Disease, Mismatch and Lifestyle Change

The THLHP provides an opportunity to test ideas about the role of environmental and socioeconomic change on health concerns believed to be either universal aspects of human aging, or consequences of an evolutionary mismatch between long-standing genetic adaptations and novel environments. We have found that several conditions common in urban areas of both high and low-income countries are rare or absent among Tsimane. As observed in other rural settings (Yazdanbakhsh et al., 2002, Rook, 2012), allergies, atopy and other auto-immune diseases are rare among Tsimane. This is to be expected according to the "hygiene" and "old friends" hypotheses, which propose that early pathogenic exposures, especially helminths, help promote improved immune regulation in ways that temper pro-inflammatory responses (Yazdanbakhsh et al., 2002, Rook, 2012). Benign prostatic hyperplasia is also rare, presumably due in large part to lower testosterone levels in early adulthood than those reported in Western populations (Trumble et al., 2015). Lower cumulative exposure to testosterone may be protective against benign prostatic hyperplasia. Reproductive cancers, such as endometrial, ovarian, breast and prostate cancers, are also often associated with high levels of cumulative exposure to reproductive hormones, and appear to be rare among Tsimane as suggested by our clinical data; other cancers of more infectious etiology, however, such as cervical cancer, are expected to be more common among Tsimane than in other populations due to higher infectious burden (Stieglitz et al., 2012).

Atherosclerosis, the main cause of CVD, also appears to be largely absent among Tsimane for several reasons (Figure 8). First, cardio-metabolic risk factors associated with greater heart disease and stroke risk in industrialized populations, such as obesity, high cholesterol, and hypertension, are rare among the Tsimane (Gurven et al., 2012). Even after adjusting for lower Tsimane body mass, rates of blood pressure increase in adulthood are lower among Tsimane than in 52 other populations from the INTERSALT study (Gurven et al., 2012). Second, despite living in semi-permanent villages with limited residential mobility, Tsimane are not sedentary; they engage in high levels of moderate physical activity, even at advanced ages (Gurven et al., 2013), and show strong cardiorespiratory fitness, as measured by VO_2max (Pisor et al., 2013) and high prevalence of bradycardia (resting pulse<60). Third, Tsimane diet is lean but calorie rich, and abundant in fiber and omega-3 fatty acids (Martin et al., 2012, Kraft et al., 2018). Regular cigarette smoking is rare (Gurven et al., 2009).

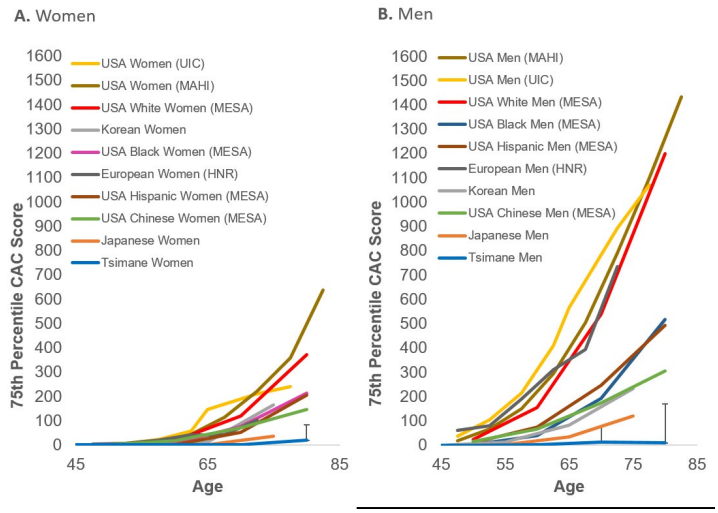

Fig. 8 75th Percentile of Coronary Arterial Calcification (CAC) in women (A) and men (B) across populations. Tsimane data are compared to U.S. (Multi-Ethnic Study of Atherosclerosis [MESA], Mid America Heart Institute [MAHI], University of Illinois at Chicago [UIC]), Germany (Heinz Nixdorf RECALL Study [HNR]), Japan and Korea. A bootstrapped 95% CI is displayed for Tsimane data. See Kaplan et al. (2017) for more details and references.

On the other hand, CRP and IL-6 — two biomarkers of inflammation that independently predict CVD morbidity and mortality in industrialized populations — are elevated throughout life among Tsimane, likely as a result of consistent pathogenic exposure (Gurven et al., 2008, Blackwell et al., 2016). In industrialized contexts, inflammation has been linked to every stage of heart disease, from atheroma and plaque formation to myocardial infarction (Buckley et al., 2009). Yet despite elevated levels of CRP, IL-6 and erythrocyte sedimentation rate, Tsimane present with exceptionally low levels of atherosclerosis (Kaplan et al., 2017). Tsimane high density lipoproteins (HDL, "good cholesterol") levels are also low (Vasunilashorn et al., 2010). However, peripheral arterial disease (PAD), a pre-cursor to fully developed atherosclerosis, is also not observed among adults, as assessed by ankle brachial blood pressure index; PAD increases with age in every other population studied to date (Gurven et al., 2009). Coronary artery calcification (CAC), a sensitive predictor of cardiovascular morbidity and mortality measured from thoracic computed tomography (CT) scans, is also extremely low among Tsimane (Kaplan et al., 2017) (Figure 8); only 8% of Tsimane by age 80 show evidence of moderate atherosclerosis, compared to 51% of "healthy" US adults. Several hundred "verbal autopsy" reports of recent and past deaths also reveal few cases of obvious cardiac or cerebrovascular events, and so mortality selection does not appear to be culling younger individuals with CVD. Ongoing cranial CTs will assess changes in cerebral morphology to help understand cognitive aging, dementia, and the link between CVD and dementia. Due to the relative absence of overt atherosclerosis and vascular disease, we expect to find lower rates of cognitive impairment and several types of cerebral atrophy in late adulthood among Tsimane than reported elsewhere. Alternatively, greater infection, inflammation and limited schooling may accelerate cerebral atrophy, cognitive decline and dementia.

Despite the systemic pro-inflammatory environment fostered by some bacterial and viral infections, other infections might offer protection against prominent chronic diseases of aging. A number of animal models provide evidence of protective effects of one type of parasitic infection on T2DM and CVD — helminths. These include mostly intestinal geohelminths such as hookworm and roundworm, but may also include water-borne helminths such as schistosomes and insect-borne filarial helminths such as *Wuscheria bancrofti*. The notion that helminths in particular might offer protection against atherosclerosis was first proposed in 2005 by the Israeli physician, Eli Magen (Magen et al., 2005). Exploring the role that pathogenic exposure, particularly helminths, plays in risk of atherosclerosis and T2DM is a relatively novel research direction that merits further attention.

As part of their own strategies to insure their own survival and reproduction, helminths have multiple effects on their host. They consume blood lipids and glucose, alter lipid metabolism, and modulate immune function towards greater T_H2 (T helper cell type 2) anti-inflammatory activity. In combination, these conditions can lower blood cholesterol, reduce obesity, increase insulin sensitivity, decrease atheroma progression, and reduce likelihood of atherosclerotic plaque rupture (Wiria et al., 2014, Gurven et al., 2016). Consistent with these expectations, we find that biomarkers of helminthic infection (e.g. IgE, eosinophils) are inversely associated with total cholesterol, LDL, HDL, and obesity (Vasunilashorn et al., 2010) (Figure 9). Total cholesterol is almost 10 points lower among those with elevated CRP and IL-6, and 19 points lower among those with elevated IgE controlling for potential confounders (Vasunilashorn et al., 2010). Other human studies are consistent with potential cardio-protective effects of helminths. There was minimal clinical atherosclerosis in patients with schistosomal hepatic fibrosis (Assaad-Khalil et al., 1991), lower levels of T2DM with lymphatic filariasis (Aravindhan et al., 2010), and lower blood glucose, glycated hemoglobin (HbA1c), insulin resistance, triglycerides and LDL with prior *Schistosomiasis japonicum* infection (Chen et al., 2012). The Indonesian ImmunoSPIN project has found that helminth infections are associated with greater insulin sensitivity (Wiria et al., 2015). Lastly, an autopsy study of cadavers in the Khanty-Mansiisk region of Russia measured both *Opisthochis felineus* worm burden and area of atherosclerotic lesions in the thoracic and aortic arteries (Magen et al., 2013). Fatty streaks, fibrotic plaques and complicated lesions were inversely related to the number of worms per infected liver and were most common in uninfected individuals.

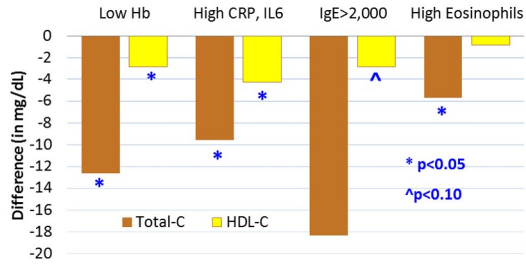

Fig. 9 Relationships between indicators of infection and immune activation on blood lipids. Low hemoglobin, high CRP and IL-6, high IgE and eosinophil count are all associated with lower total blood cholesterol, and to some extent with lower HDL cholesterol. Sample restricted to adults age 20+ from 17 Tsimane villages in 2004. Results based on multiple regression analyses of total-C (n=345) and HDL (n=318) that also control for age, sex, BMI. Low Hb refers to first quartile, high eosinophils refers to fourth quartile. Based on Vasunilashorn et al. (2010).

In summary, Tsimane have a very low frequency of chronic diseases typically found in Western populations. The relative roles of energetic expenditure, diet and pathogen burden in explaining differences in chronic disease risk among populations are still poorly understood due to the scarcity of detailed, longitudinal studies of appropriate populations in epidemiological transition, but each appears to play a contributory role. THLHP research shows that a more nuanced understanding of the role of infection-induced inflammation in the etiology of chronic diseases is needed. Perhaps high levels of inflammation are only atherogenic and diabetogenic in the context of high adiposity and minimal exercise. Alternatively, different sources of inflammation may have different effects on chronic disease, with infection in some cases actually lowering chronic disease risk.

Conclusion

Longevity is a feature of *Homo sapiens* living subsistence lifestyles, in direct contradiction to the Hobbesian view that human lifespan was "nasty, brutish and short" — a view also traditionally supported by paleodemographic lifetables of prehistoric populations. The THLHP provides an interesting case study with broad relevance to demography, and has contributed to critical debates in the social and life sciences. THLHP helps bridge theory with empirical data that establishes vital links between kin-directed cooperation, reciprocal exchange networks, age profiles of productivity and health, and human life history traits. Our research program thus attempts to advance a "whole organism" understanding of the aging process and health in environmental conditions more similar to the ones in which humans evolved, while also studying reaction norms and potential health-related mismatches as those environmental conditions change.

An ethnographic emphasis combined with an evolutionary and ecological focus has the ability to influence multiple disciplines by highlighting aspects of human diversity typically ignored by more conventional research traditions. For example, Tsimane findings support new ways of thinking about the role of inflammation on chronic disease etiology, and the potential benefits of helminth infection on immune regulation (as well as on cognitive performance, fertility and insulin resistance). Indeed, the long co-evolutionary history of helminths and other pathogens with humans highlights neglected potential mutualisms with beneficial effects on human health. The relative absence of many chronic diseases that afflict high income countries, including coronary heart disease, T2DM, osteoporosis, autoimmune diseases, prostate and breast cancer, provides new opportunities for study. A causal understanding of these and other mismatch diseases may be advanced by further study of groups like the Tsimane who are undergoing rapid changes in multiple ways that can be studied jointly.

Although the Tsimane represent a single case study, an important goal is to stimulate more cross-cultural comparative research using standardized methods. For example, a comparative approach will be needed to make broader inferences about the relative importance of infection, lifestyle factors and genetics in chronic disease morbidity. In this case, comparison of populations varying in parasite burden, diet and activity profile can provide insight into the role of immune dysregulation on chronic disease progression.

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31. Trade-Offs between Mortality Components in Life History Evolution: The Case of Cancers

S. Pavard and C. J. E. Metcalf

Little is known about the relative importance of different causes of death in driving the evolution of senescence and longevity across species. Here we argue that cause-specific mortality may be shaped by physiological trade-offs between mortality components, challenging the theoretical view that physiologically independent processes should senesce at the same rate, or that interactions between causes of death will make selection blind to the effects of specific causes of death. We review the evidence that risk of cancers trades off with risks of mortality from other diseases, and investigate whether this might explain two of the most puzzling paradoxes in cancer evolution. First, among species, cancer prevalence is not a function of species' size and longevity, despite the fact that cancer incidence is known to be a function of the number of cell divisions (and therefore of size) by unit of time (and therefore of longevity). Second, within species, despite the fact that genomic instability is thought to be the proximal cause of both cancer incidence and senescence, mortality rates rise with age while cancer incidence decelerates and declines at old ages. Building on a relatively novel theory from cellular biology, we construct a preliminary model to reveal the degree to which accumulation of senescent cells with age could explain this latter paradox. Diverting damaged stem cells towards a senescent-state reduces their risk of becoming tumorous; however, conversely, the accumulation of senescent cells in tissues compromises their rejuvenation capacity and functioning, leading to organismal senescence. Accumulation of senescent cells with age may then be optimal because it reduces cancer mortality at the cost of faster senescence from other causes. Evolution will drive species towards a balance between these two sources of mortality.

Introduction

For any organism, all fitness components cannot be maximized simultaneously. The result would be a so-called "Darwinian demon" (e.g. a species with both large survival and fertility, (Law, 1979)); which is improbable based on observations across species, and impossible based on logical considerations. Indeed, individuals are constrained by limited resources, which must be allocated to different functions of the organism at different times during the organism's life. The result is trade-offs across fitness components. Classic trade-offs in life history evolution include investment in current reproduction to the detriment of future survival or reproduction

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(the costs of reproduction, (Williams, 1966)), investment in fast growth during juvenile life to the detriment of adult fitness, or investment in the production of many offspring to the detriment of their survival (the quantity-quality trade-off, (Lack, 1947)). An alternative trade-off would be investment in a biological function linked to better surviving one cause of death, to the detriment of survival of another cause of death. Surprisingly, this is rarely considered.

Our aim here is to explore the question: do mortality components (potentially translating to specific causes of death) trade with each other? We start by briefly summarising the evidence for individual level trade-offs between mortality components, a requisite for allowing the evolution of varied strategies among species. We also critically review two major predictions that may have curtailed research into evolution associated with trade-offs between mortality components. We then narrow our focus to one of the most puzzling mortality patterns in evolutionary biology: **cancer mortality** defies prediction both within and between species. First, comparing between species, since genomic instability with age is thought to be an important proximal cause of senescence, and is known to be the key proximate driver of cancer, cancer mortality is expected to map closely to a species' actuarial senescence, and thus longevity. That this is not the case and is known as **Peto's paradox** (Nunney, 1999): cancer prevalence does not correlate with a species' size and longevity, suggesting that cancers are not "only" a by-product of senescence across species. Moving from comparison between species to comparisons within species, in humans, as in rats, incidence of cancer decelerates and even declines at older ages (reviewed below and in (Anisimov, et al. 2005)), decoupling cancer from senescence in these species. This lack of association of prevalence of cancer with longevity across species and senescence rates with cancer incidence by age within species suggests that the molecular and physiological mechanisms underlying cancer may be different or at least act differently on morbidity from those underlying other causes. Evolutionary theory is thus urgently needed to shed light on how cancer development at the level of organisms and species evolved together with lifespan and life-history (Casás-Selves and Degregori 2011). This is true even when species cancer prevalence is low in the wild (or large only due to recent human-driven changes, (Hochberg and Noble, 2017)) because, in most species, especially the large and the long lived, the puzzling question is how "the development and architecture of our tissues were evolutionarily constrained by the need to limit cancer" (Casás-Selves and Degregori 2011, DeGregori 2011).

Recent evidence for a negative correlation between cancer prevalence and that of other diseases further emphasizes the disconnection between cancer and other causes of mortality. Applying a competing risks model to data on underlying and secondary causes of death in the U.S. between 1968–2004, Yashin and colleagues (Yashin, et al. 2009) estimate a negative correlation between cancer and asthma (about –2.5%), Parkinson disease (ranging between −3% to −5%), Alzheimer's disease (ranging from −1 to −10%), diabetes (about −10%), cerebrovascular accidents (about −12%) and coronary heart disease (ranging from −25 to −15%). These intriguing negative patterns have been shown to also hold in the more finely resolved data from the Framingham Heart Study (Ukraintseva, et al. 2010). Importantly, negative correlations can emerge from mechanisms other than the physiological trade-offs required to drive evolutionary processes. However, molecular and cellular physiologists have several hypotheses of mechanisms that could drive these negative correlations (Ukraintseva, et al. 2010). Among them, the accumulation of cells in senescent-state in tissue may be the

physiological mechanism mediating negative correlations between mortality by cancers and from other causes, by impeding cell divisions and progress towards cancer. Despite its potential to illuminate patterns of cancer mortality both within and among species by capturing trade-offs between mortality components, this *senescent-cells theory of ageing*, has not formally been previously modelled.

In this chapter, after summarizing the general question of the potential for trade-offs between mortality components, and reasons for their relative neglect in life history evolution studies, we detail the core paradoxes around cancer mortality in the context of life history evolution. We then introduce a simple preliminary model of the optimization of the dynamics of non-senescent, tumorous and senescent cells built around the *senescent cells theory of ageing* and discuss implications for evolutionary outcomes related to cancer mortality. This model aims to be a "proof of principle" for two main concepts: first, trade-offs between mortality components may induce an increase of mortality by age that nevertheless reflects an optimal strategy emerging from balancing trade-offs between two different risks of death and; second, the *senescent cells theory of ageing* may be a mechanism that underpins such a trade-off. We conclude by discussing how this physiological framing of a trade-off between mortality components might resolve paradoxes relating to patterns of cancer prevalence among species, and patterns of cancer incidence across age within species. We place this discussion within the context of existing theory on aging, and point to methodological approaches that could open the way to further investigation of trade-offs between mortality components.

Potential for Trade-offs Between Mortality Components into Life History Theory

To survive, an organism must invest considerable energy into maintenance, which encompasses many different physiological functions; from higher physiological functions (breathing, digesting, maintaining homeostasis, cardiac and neural activities, maintaining the immune system and performing immune responses, etc.) to molecular and cellular functions (controlling and repairing DNA, maintaining proteostasis and cellular metabolism, etc.). Although these functions are tightly interconnected physiologically, they nevertheless depend on specific genetic architectures that do not completely overlap. For instance, genes involved in immunity differ from those controlling cell replication and tumour suppression. Categorizing genes according to their functions (or phenotypes) is one of the most intense current research focuses of molecular biologists. Construction of vast databases reporting action of genes at a higher integrative level (e.g., **Poly**morphism **Phen**otyping v**2**) is underway. A crucial focus of this effort is to link these emergent functions or phenotypes to the vast epidemiological genetic literature on diseases resulting from polymorphisms at underlying genes. This effort has yielded increasing evidence for so called "antagonistic pleiotropic" effects of gene(s), meaning effects which are positive for one fitness component, but negative for another. In particular, evidence for genetic trade-offs between mortality components has emerged. The most famous example is polymorphisms in *APOE*-ε4, a gene involved in many neurodegenerative syndromes in adults but that allows higher levels of vitamin D absorption, and for instance protects children against diarrhoea (reviewed in (Oriá, et al. 2007)). There are therefore multiple lines of evidence supporting the existence of genetic level trade-offs between mortality components.

But do these potential trade-offs, detectable in the impacts of a single gene, translate into unavoidable physiological trade-offs, reflecting differential allocation of resources between physiological functions linked to survival, thus of importance for life history evolution? To our knowledge, this has never been formally evaluated, and the mapping between evidence for antagonistic pleiotropy and physiological trade-offs is likely to be complex. Nevertheless, striking gradients across species in investment in survival-related functions suggest that physiological, resource allocation-based trade-offs are likely. For example, investment in immune system function, a key line of defence against pathogens, and thus important for individual survival, is highly variable across species (reviewed in (Schmid-Hempel 2003)). Digestive organs consume a significant fraction of metabolic energy (20–25% in vertebrates, reviewed in (Karasov and Douglas 2013)), but this varies considerably among species according to the biochemistry of food intake. Intriguingly, the surface of the intestine is also the major contact zone between the immune system and food-borne and microbial antigens, making interactions and trade-offs between digestive and immune systems crucial in ecophysiology (Meitern, et al. 2016). Similarly, the brain is an extremely energy consuming organ which demands high levels of maintenance. Yet brain size varies substantially across species and correlates positively with basal metabolic rate in mammals (Isler and van Schaik 2006). Given these striking life history gradients across species, one might therefore wonder why trade-offs between underlying mortality components (and associated physiological functions) have been little incorporated into evolutionary demography and life-history theory. Two theoretical predictions may have been responsible for curtailing research in this direction.

First, in George Christopher Williams' seminal article on the evolution of senescence, it is predicted that: "senescence should always be a generalized deterioration, and never due largely to changes in a single system" (Williams 1957). The idea, reframed by John Maynard-Smith (Smith 1962), is that all physiologically independent processes should senesce at the same rate as "natural selection will always be in greatest opposition to the decline of the most senescence-prone system" (Williams 1957). We illustrate this principle in Figure 1 showing three Gompertz-shaped distributions of deaths by age and by cause. The observed density of deaths from c_1 (red polygon) is much larger than densities of deaths from c_2 and c_3 (green and blue polygons) because few individuals survive until ages where c_2 and c_3 are most likely. This shows that selection pressure on susceptibility alleles to a specific cause of death is affected by the age-specific risk of other causes of death to which the population is exposed. For example, removing c_1 from the population will drastically increase the strength of negative selection on c_2. More generally, gradients of selection occur not only across age, but also emerge as a result of other causes of death. For example, assume that the spectrum of susceptibility alleles to c_2 is at a mutation-selection balance, and selection is just above the threshold at which it can overcome genetic drift. As a result, negative selection is weak but will eventually purge deleterious mutations associated with c_2. All else being equal, susceptibility alleles to c_1 will be more intensely negatively selected. Purifying selection will thus decrease the number and the frequency of c_1 alleles, eventually decreasing the frequency of deaths from c_1. By contrast, susceptibility mutations to c_3 are neutral and mutations will accumulate, eventually increasing the frequency of death from c_3. Overtime, therefore, natural selection will tend to homogenise the rate at which cause-specific mortality increases with age.

The validly of this hypothesis has been extensively discussed, and empirical evidence for challenges to it reviewed (Gaillard and Lemaître 2017). Several studies show that demographic, phenotypic and functional traits do not senesce synchronously (e.g., in Soay sheep in (Hayward, et al. 2015) or in reptiles in (Massot, et al. 2011)). However, this lack of synchronicity in rates of senescence among different functions is, as yet, largely unexplained. As shown in figure 2.B (solid line) trade-offs between mortality components may provide part of the answer.

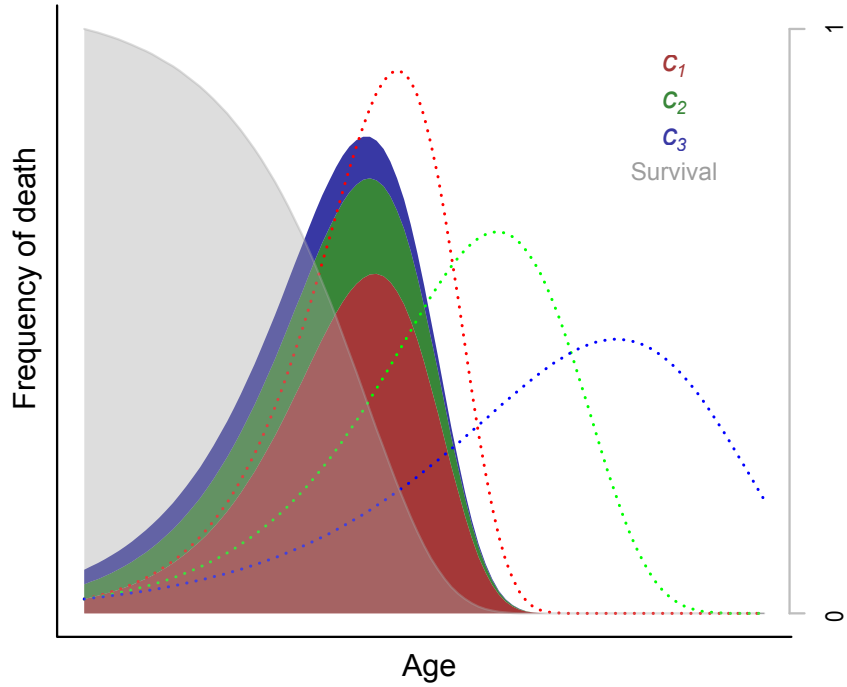

Fig. 1 Stacked distributions of deaths $f_c(t) = S_1(t)S_2(t)S_3(t)h_c(t)$ across age t for three causes of death c_1, c_2 and c_3 (red, green and blue polygons, respectively); the corresponding overall survival over age $S(t) = S_1(t)S_2(t)S_3(t)$ (grey polygon and right axis); as well as the distribution of death from each cause $f_c(t) = S_c(t)h_c(t)$ in the case where individuals die from only this cause (red, green, and blue dotted lines). In this example, cause-specific mortality hazards are Gompertz-shaped (such that $h(t) = ae^{b_c t}$, , with $a = 0.001$, $b_1 = 0.1$, $b_2 = 0.07$ and $b_3 = 0.05$).

The second theoretical feature that may have reduced research into the question of trade-offs between causes of death is the lack of independence among causes of death: multiplicative effects are likely to be ubiquitous. For example, inflammation underlies multiple causes of death, from heart disease to cancer (Coussens and Werb 2002, Willerson and Ridker 2004). This major concept in epidemiology also raises inferential difficulties in characterizing causes of death. Many causes likely contribute to each death, particularly in older individuals, and disentangling their contribution is consequentially statistically challenging, especially as each mortality event occurs only once. To disentangle these complex causal pathways, epidemiologists distinguish between proximal and distal factors leading to death. This has led researchers to envision senescence as the accelerated accumulation of a health deficit whose ultimate outcome, death, whatever its cause, cannot be seen as the result of the deterioration of a sole physiological function (Kulminski, et al. 2007, Yashin, et al. 2007). As a consequence,

selection on allocation strategies between mortality components at an evolutionary scale might be obscured by covariation between causes of death at the individual scale.

However, to our knowledge, this has not been formally framed, and we explore this in Figure 2. Extending the model presented in Fig. 1, we assume this time that the third cause of death is the result of an interaction between mortality components respectively responsible for causes of death 1 and 2. This model captures the fact that, over the course of an individual's life, factors that increase the risk of cause of death c_1 may also have the effect of increasing c_2 and *vice versa*. Figure 2A show the distribution by age and causes in the scenario where 25% of deaths result from these interactions. Assuming that cause-specific senescence rates result from an allocation strategy in the maintenance of the respective physiological components, we illustrate in figure 2B that an allocation strategy optimizing life-expectancy can still be identified in the case where causes of death (c_1 and c_2) covary, such that the cause of death is indistinguishable in 25% of cases. This scenario flattens the optimal allocation balance between the two biological functions relative to the case where both are fully independent (Figure 2B, solid line), but an intermediate optimum can still be identified (Figure 2B, dashed line).

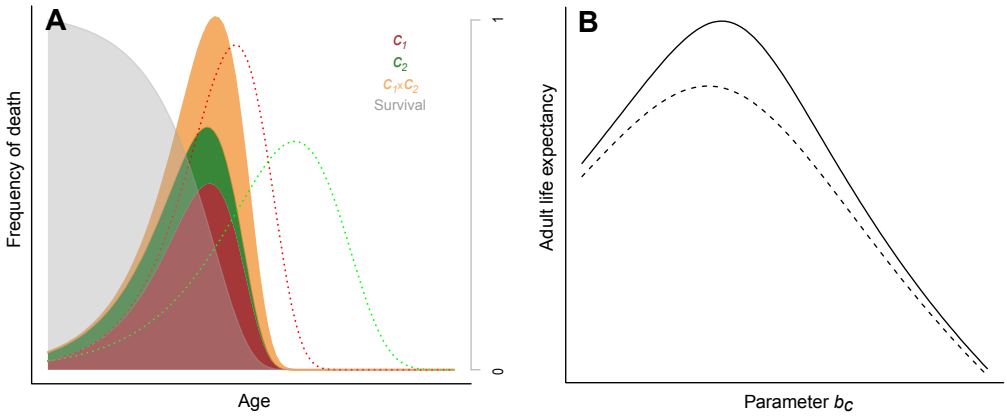

Fig. 2 As for figure 1, but in the case where causes of death are not independent. Cause c_3 is now the product of the interaction $c_1 \times c_2$ between c_1 and c_2 such that $h_{1\times 2}(t) = za^2e^{(b1+b2)t}$ (where z is the coefficient of this interaction, b_1 is a parameter shaping the hazard associated with cause c_1, and likewise for b_2). Panel (**A**) shows the distribution of deaths in this case (taking $z = 35$ such that $c_1 \times c_2$ accounts for more than 25% of observed deaths). Panel (**B**) further assume a stationary population of a species whose fertility rates are constant over age, such that remaining life expectancy e_α at age α is an adequate measure of adult fitness. We assume a linear negative relationship between b_1 and b_2 that captures a trade-off between causes of death. The remaining life expectancy e_α is then depicted for a range of parameters (b_1, b_2) in the case where $z = 0$ (no interaction between causes of death, solid line) and in the case where z = 35, dashed line).

Cancers and Physiological Ageing

Ageing is multifactorial. In their seminal review (Lopez-Otin, et al. 2013), Carlos López-Otín and colleagues described nine mechanisms leading to functional deterioration with age. Core aspects of deterioration have been usefully categorized as primary causes of intracellular damage (genomic instability, telomere attrition, epigenetic alteration and loss of proteostasis),

cellular dysfunction (deregulated nutrient sensing, mitochondrial dysfunction, and cellular senescence) and altered tissue dynamics (stem cells exhaustion, altered intercellular communication). To incorporate these proximal functional deteriorations into an evolutionary demographic framework for understanding ageing requires understanding to what extent each is linked to mortality in general, and to specific causes of death in particular.

Cancers may be the pathology for which this link is the most straightforward. All cancers appear to be genetic diseases at the cellular level. The current dominant theory of a cell carcinogenesis (but see below) is a multistage process requiring the accumulation of (epi) mutations in a mitotic cell lineage (often called "hits"), ranging up to large chromosomal abnormalities and aneuploidy. The outcome is liberation of neoplastic cells from homeostatic mechanisms of cell division, potentially resulting in development of cancer. This multistage theory of carcinogenesis was first proposed in the fifties by Peter Armitage and Richard Doll (Armitage and Doll 1954) and mathematically formalized in its most frequently used expression by Richard Peto (Peto 1977). However, the underlying multistage genetic processes required to generate the series of 6 to 8 "hallmarks" of cellular physiology that transform a normal cell to a neoplastic cell have only recently been characterized (reviewed in Hanahan and Weinberg 2011). The number of "hits" required for carcinogenesis varies from two (Knudson 1971) to eight (Vogelstein, et al. 2013) as a complex — and yet unknown — interactions between species, tissues and cancer types (Nunney and Muir 2015). Quantifying the rate of accumulation of somatic mutations with age (due to increased mutation rate and decreased repair efficiency) and amongst species; for example using transgenic LacZ animals (reviewed in Moskalev, et al. 2013) is therefore fundamental to characterizing the links between proximal somatic mutation accumulation to distal cancer morbidity.

Importantly, despite the detailed mechanisms proposed to explain carcinogenesis, the functional relationship between mutation accumulation in stem cells lineages and cancer risk remains poorly described. It has been shown recently that lifetime risk of cancer correlates with total number of cell divisions in tissues (Tomasetti, et al. 2017, Tomasetti and Vogelstein 2015), but the kinetics of damage accumulation with age, and how this shapes cancer incidence is still little known and intensively debated. As recently pointed out, about 50% of stem cell somatic mutations occur during ontogenesis; and this mutation accumulation does not translate into increases in cancer at that life stage. By contrast, cell divisions slow down during adult life, yet this does translate into an exponential increase in cancer incidence (Rozhok and DeGregori 2016). The Doll-Armitage multistage model is therefore not sufficient to explain cancer incidence by age and alternative evolutionary-based hypotheses have been proposed (DeGregori 2017).

Telomere inhibited-attrition is also linked to risk of cancer. In normal cells, telomerase inhibition and telomere shortening limits the number of cell divisions, a phenomenon denoted as "the Hayflick limit" (Hayflick and Moorhead 1961), corresponding for example to under 50–70 divisions in fibroblasts. Once this limit is reached, cells enter a so-called "senescent" state and stop replicating. In most human cancers, telomerase activation past this limit impedes telomere attrition and confers cell immortality (Donate and Blasco 2011, Shay and Bacchetti 1997). The role of epi-mutations in carcinogenesis is also increasingly studied. Recently, a strong correlation between chronological and epigenetic age has been identified, accounting for tissues and cells type (Horvath 2013). Epi-mutations — mutations leading to abnormal

repression or activation of genes — have been proved to be a frequent proximate mutational event leading to carcinogenesis (Banno, et al. 2012).

Risk of cancers is also linked to cellular dynamics. Carcinogenesis can indeed be framed as the result of a dynamical interplay between predation by the immune system, and competition between cancer and normal cell lineages in a changing fitness landscape (Rozhok and DeGregori 2016), with a pinch of stochastic drift (Crespi and Summers 2005, Pepper, et al. 2009, Shpak and Lu 2016). This "ecological theater of carcinogenesis" (Crespi and Summers 2005) also changes through age, in ways expected to make cancer occurrence more likely. For example, hematopoietic stem cell exhaustion resulting from other ageing processes (such as increased genetic instability due to accumulation of oxidative damage (Ito, et al. 2004)) leads to a decline of the production of adaptive immune cells with age (a process called immunoscenescence) and therefore reduces the organisms ability to keep cancerous cell lineages in check (reviewed in Henry, et al. 2011).

Overall, and although alternative theories are emerging (DeGregori 2017), (epi)genetic instability leading to mutation accumulation in stem cell lineages is still considered the primary factor required for cancer development at the cellular level; and prevention of telomere attrition the most common way for cancer cells to escape replication homeostasis. Accumulation of mutations with age should therefore correlate with increased cancer incidence. Importantly, these drivers are the very same as those invoked for senescence at the individual level. However, this link is paradoxical at two levels: between and within species.

Prevalence of Cancers Across Species and Peto's Paradox

Cancers are ubiquitous in multicellular organisms and occur each time that a cheating cell escapes the bounds defining cooperation among cells by escaping inhibition of proliferation and cellular death (Aktipis, et al. 2015). Data on the prevalence of cancers across the tree of life are scarce and the ecological, physiological and phylogenetic determinants of a species cancer prevalence are mostly unknown (see below). However, preliminary results (based on limited data) tend to confirm one of the most puzzling paradoxes of evolutionary biology (Abegglen, et al. 2015): the fact that cancer prevalence is not a function of organism size or longevity.

The fact that this is paradoxical was first pointed out by Sir Richard Peto (Peto 1977): in a multistage carcinogenesis process, accumulation of damage within cells' lineages should be a function of the number of cell divisions, itself a function of the number of cells and lifespan. However, while mice are 1000 times smaller and about 30 times shorter lived than humans, cancer incidence is about the same (Rangarajan and Weinberg 2003). This led Peto to ask whether our stem cells are "a billion or a trillion times more "cancer-proof" than murine stem cells?" and "Why don't we all die of multiple carcinomas at an early age?" (Peto 1977, pp. 1413–14). Nunney (1999) first denoted this "Peto's paradox"; and explored the issue via a population genetic model where cancer incidence depends on the number of "hits" required for a given cell to turn into cancer. This model demonstrated that highly proliferating tissues require additional controls of carcinogenesis as organism size increases to prevent cancer prevalence from wiping out the entire species (Nunney and Muir 2015).

The degree to which Peto's paradox holds across taxa remains however largely unresolved. To date the most extensive comparative study of cancer incidence with body mass and lifespan included only 31 mammal species; and cancer incidence estimates for a subset of these species were based on only 10 necropsies (Abegglen, et al. 2015). Both richer data, but also more complete

statistical analyses are required to answer this question. The latter is necessary because, first, while multistage carcinogenesis theory predicts that longevity should be positively correlated with cancer prevalence; cancer morbidity is obviously negatively correlated to longevity and comparative studies should account for this. Second, longevity emerges from both a species' magnitude of intrinsic mortality per unit of time (e.g. the *a* parameter of a Gompertz-shaped mortality, or the intercept of the log mortality function) but also the rate at which mortality rises with age (e.g., the *b* parameter of a Gompertz-shaped mortality, or the slope of the log mortality function). Each, both or neither might be associated with cancer incidence and thus inform the generality of Peto's paradox. Overall, age-specific data are urgently needed in comparative oncology to assess the role played by cancer in ageing.

Furthermore, cancer prevalence does vary between species beyond the effect of size. A recent review of cancer prevalence and etiology in wild and captive animal populations (Madsen, et al. 2017) revealed that prevalence in wild vertebrates ranges from 0.2% to more than 50%. Two striking conclusions emerged from this review. First, in some species, cancers are one of the most prevalent current causes of death in nature, making them an important fitness components, and therefore, of crucial ecological and evolutionary significance (McAloose and Newton 2009, Vittecoq, et al. 2013) (although one should note that the extent to which the documented cancer risks emerge from recent environmental conditions is still unclear (Hochberg and Noble 2017)). Second, cancer prevalence depends less on species' size and life expectancy and more on phylogeny (e.g., reptiles seem more sensitive to cancer than mammals) or ecology (e.g., small carnivores exhibit larger average prevalence of cancer than large herbivores). Further, despite some such broad patterns, most of the time, species specific cancer prevalence defies prediction (e.g. one of the larger prevalences of cancer, about 50%, is observed in a large herbivorous mammal, the Cape mountain zebra, but this results from a particular case of equine skin cancer (Marais and Page 2011)). In domestic dogs, large dogs have larger rates of cancers than small dogs (Fleming, et al. 2011). This is likely due to artificial selection on size, which has also resulted in the fact that life expectancy of large dogs is lower than that of small dogs (Kraus, et al. 2013) and which makes dogs an outlier relative to the usual pattern linking size to lifespan across species, and therefore preventing generalization. Other species, such as the naked mole rat, exhibit as yet unexplained low cancer incidence (Buffenstein 2008, Taylor, et al. 2017). More generally, rodents are promising model species for research on cancer suppression mechanism and its links to ageing (Gorbunova, et al. 2014).

Despite these heterogeneities, arguably, the broad sweep of available evidence continues to align with Peto's paradox. Many explanations have been proposed to resolve Peto's paradox (reviewed in (Caulin and Maley 2011)). Among others, a lower mutation rate could be efficient in limiting carcinogenesis — for example, a threefold decrease in the mutation rate in humans compared to mice would be sufficient to lead to similar cancer incidence (Caulin, et al. 2015). However, mutation rates have not yet been proved to differ between mice and humans. Alternatively, differences in the efficiency of "gatekeeper" tumour suppressor genes — genes enforcing checkpoints to suppress neoplastic transformation — could make the number of "hits" required for carcinogenesis differ among species. It has been mathematically shown several times that increasing the number of "hits" required for neoplastic transformation is a particularly efficient approach to preventing cancers in large species (Caulin, et al. 2015, Nunney and Muir 2015), but, to date, there is no empirical evidence to support this. For example, the number of replicates of tumour suppression genes of the *TP* family might be expected to

correlate with species' size, as this would make tumour suppression more efficient. However, this does not seem to be the case overall, although, suggestively, a large number copies of *TP53* is found in elephant species (Caulin, et al. 2015). Cells' dynamics (mainly influenced by cells' anatomical compartmentalization, and cells' effective size, both of which affect the stochastic disappearance of cancer cell lineages), immune and apoptotic efficiencies, regulation of the number of potential divisions through telomere length are all good hypotheses that should be investigated with respect to the link between size, cancers, and phylogeny. The metabolic hypothesis is also receiving increasing support (Caulin and Maley 2011, Dang 2015): because the by-products of metabolism (such as reactive oxygen species) correlate with metabolic rate, and because basal metabolic rate scales with mass, cells may be less exposed to metabolic damages as an animal's size increases. Finally, an aspect that may have received too little attention to date is the role of cell division rate. Caulin et al. (2015) demonstrate that a decrease from 1 division every four days to 1 division every 8–13 days would be enough to account for similar cancer incidence between species differing in size by a factor 1000.

Overall, these various lines of evidence suggest that there is striking gap in life history theory around understanding the role of cancers, particularly in the context of Peto's (as yet unresolved) paradox, and associated elucidation of the drivers of cross-species patterns. As physiological trade-offs are the clay from which life-history is moulded, a key step will be to identify which other life-history components are affected by physiology investments that reduce cancer incidence.

The Paradox of Deceleration and Decline of Cancer Incidence with Age

Close examination of existing research on the topic of cancer incidence reveals another striking paradox that has so far been neglected in studies of life history evolution. As summarized above, cancer is the cause the death that is the most easily tied to the most proximal functional mechanisms of senescence, in particular, the accumulation of damage in somatic cells lineages with age due to genomic instability. Cellular dysfunctions also increase with age and are linked to increased cancer incidence. Together, these patterns suggest that increases in cancer incidence should significantly contribute to, and closely match in shape, the increase of mortality with age. Surprisingly and paradoxically, this is not the case. The proportion of deaths due to cancer decreases after age 50–60 years old, mostly to the gain of diseases of the circulatory and respiratory systems (see figure 3A and 3B). As a result, cancers become the cause of death the least involved in senescence past these ages. A decline in cancer mortality rates has even been demonstrated for very old ages (Smith 1996). The general pattern for most cancers in humans is that incidence first increases with age, then decelerates and even declines at old ages (see figure 3C). This has been known since the sixties (e.g. in (Cook, et al. 1969)) and has since been demonstrated for a large diversity of cancers and populations, for example in 2005 France (Bélot, et al. 2008) or in 2012 Korea (see Fig. 3A in (Jung, et al. 2015)). Deceleration is even visible for a given population of a given age, at a given site, but for different histopathologic subtypes of breast cancers (Anderson, et al. 2006). This proves true even when prevalence of cancers differs due to environmental conditions, or between the sexes. For example, prevalence of oesophageal cancer is much larger in rural than in urban China, and in males than in females (Chen, et al. 2014). However, a deceleration of incidence of cancer past 70 years old, or its decline past age 80 is always observable. It must be stressed that deceleration of cancer

incidence alone is paradoxical; but the decline in the incidence in the oldest old makes it even more puzzling. Yet, recent reviews of the literature unambiguously confirm this decline in the oldest old (Harding, et al. 2008, Nolen, et al. 2017, Pavlidis, et al. 2012); making cancer one of the least prevalent causes of death in centenarians. For example, cancers account for 24.5% of the deaths at 80–84 years old in 2001–2010 England and drop down to 4.4% in people surviving 100 years old. Finally, humans are not exceptional in this respect. Similar decelerations and declines of cancer incidence with age has also been demonstrated in rats (e.g., (Pompei, et al. 2001) and reviewed in (Anisimov, et al. 2005)) and in domestic dogs (Fleming, et al. 2011).

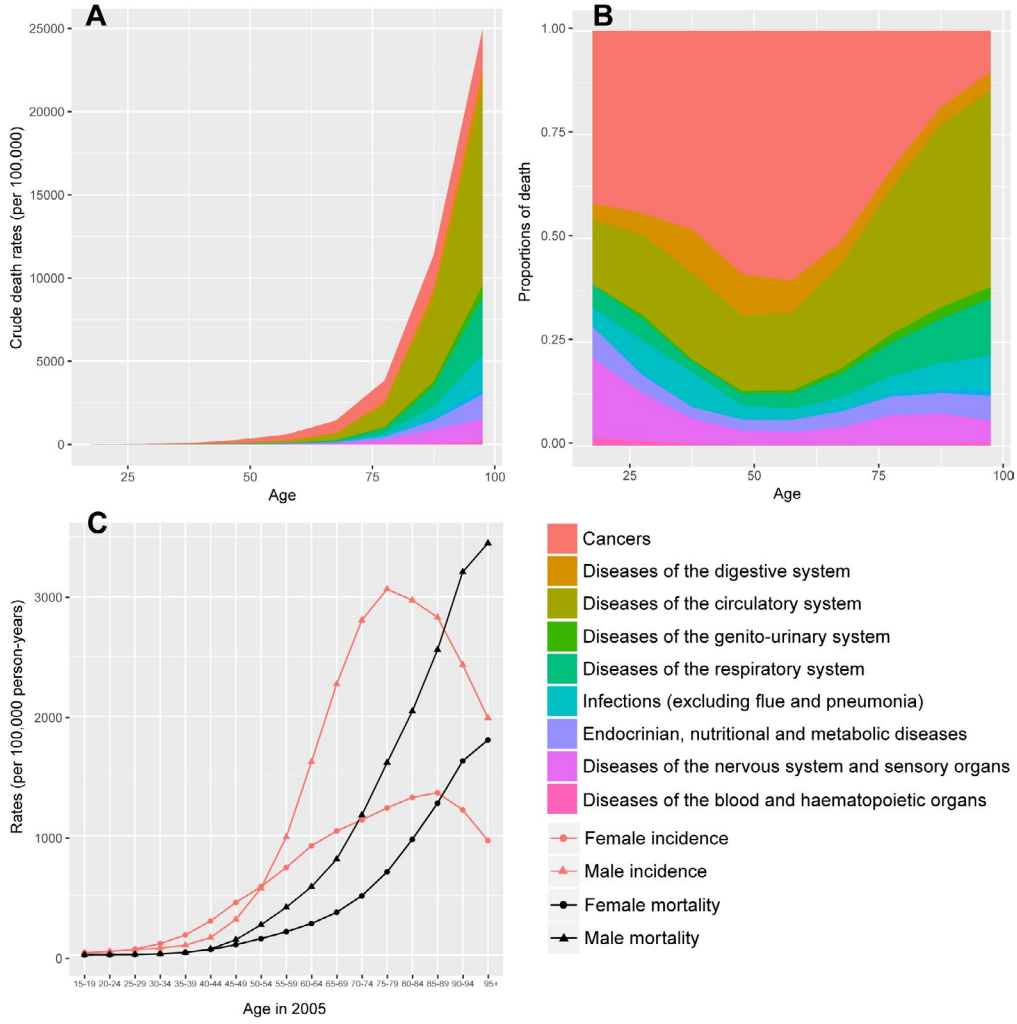

Fig. 3 Stacked crude death rates (**A**) and proportions of death (**B**) per causes in France in 2005. Data were gathered by the Centre d'épidémiologie sur les causes médicales de décès de l'Institut national de la santé et de la recherche médical (Inserm-CépiDc). Data, data documentation, and methods for death rates calculation can be found here http://www.cepidc.inserm.fr/. Panel (**C**) shows sex-specific incidence and mortality by cancers by age in 2005 France. Figure is reproduced from Bélot et al. (2008); where exceptionally detailed data on cancer incidence per site can also be found.

Several explanations for deceleration and decline in cancer incidence with age are discussed in (Anisimov, et al. 2005, Arbeev, et al. 2005, Hanson, et al. 2015). Detection bias (i.e. resulting from the fact that diagnosing cancers is more difficult in the oldest, frailest individuals, because many procedures are too invasive) or cohort-period bias (i.e. resulting from the fact that oldest cohort may have been less exposed to a cancer-prone environment) can only partially explain the results in humans, and are unlikely in other species. Two major hypotheses remain. Both were first proposed in studies carried out by A.I. Yashin. The first hypothesis is an application to cancer mortality of "the impact of heterogeneity in individual frailty on the dynamics of mortality" developed by (Vaupel, et al. 1979). The selective disappearance with age (also called differential selection) of individuals genetically or environmentally more susceptible to cancer will mean that the proportion of individuals less prone to cancer will increase with age. Change in population structure in oldest age-classes may lead to an apparent decline of the aggregated incidence rates (Vaupel and Yashin 1986). However, evidence from laboratory animals that display this decline despite low levels of genetic and environmental heterogeneity (Anisimov, et al. 2005) suggest that this heterogeneity is unlikely to be the sole cause of this pattern. Even in humans, aspects of the age profile of mortality suggest that heterogeneity in cancer susceptibility is unlikely to be the only factor underlying this pattern. In a frailty model, the inflection of the hazard should be maximal when selective disappearance is the highest. In most cases of cancer, this inflection occurs at the oldest ages (after age 75–85) while cancer incidence starts rising quickly much sooner, at ages around 40–50 (e.g., (Jung, et al. 2015)), suggesting that inflection in incidence should occur sooner than observed in most cases (although further investigation is necessary). But this may also be because magnitude and typology (individual, familial or social; (epi)genetic or environmental; inherited or acquired) of heterogeneity in cancer susceptibility is unknown for most cases of cancers. At the end of the day, the role played by heterogeneity (among other causes) in shaping cancer incidence has been largely understudied (Hanson, et al. 2015).

A good example of this is breast cancer, perhaps the type of cancer for which genetic determinants are the best known. In this case, it is estimated that "only" 5–10% of women with breast cancer have a familial history, making selective disappearance of women at higher risk of cancer due to genetic susceptibility or shared familial cancerogenous environment likely insufficient to explain decline of incidence at old age (Balmain, et al. 2003, Melchor and Benítez 2013). For example, in 2000–2005 USA, breast cancer incidence starts declining after age 75 years old. This decline is unlikely to be due to the selective disappearance of individuals carrying mutations on the two major susceptibility gene to breast and ovarian cancers, *BRCA1* and *BRCA2*, because recent evidence show that they account only for about 20–25% of the familial risk and because 39–65% and 11–39% of carriers would have already developed either a breast or ovarian cancer by this age. However the age-specific incidence of numerous susceptibility genes of low, moderate and high penetrance, and accounting for about 25% of the familial risk, have yet to be investigated (Melchor and Benítez 2013). About 50% of familial risk is as yet unknown, and is likely to result from polygenic risk factors, gene-environment interactions or shared familial cancerogenous environment. Finally, both the age at which the deceleration starts and the age at which the decline in incidence occurs varies widely between populations (e.g., between Japan and US (Tsuchida, et al. 2015) or between Asian populations (Youlden, et al. 2014)). The range of differences is far too large to result from differences in frequencies

of genetic susceptibility between populations, emphasizing the role played by other types of heterogeneity (beyond the familial or genetic to social or environmental) — in shaping breast cancer incidence by age.

A second set of hypotheses relates to the fact that somatic ageing may slow down incidence of cancer with age (Ukraintseva and Yashin 2001, Ukraintseva and Yashin 2003). As introduced above, cancer incidence may be linked to metabolic rates, and cell proliferation rates. Although humans have been recently proved to have a larger basal (or resting) metabolic rate (BMR) than great apes (Pontzer, et al. 2014), this latter declines with age in adults ((Mitchell 1962); reviewed in (Manini 2010)), and this might thus shape declines in cancer incidence. However, the existence of such a decline across species is debated (reviewed, and argued for, in (O'Connor, et al. 2002)) preventing generalizations as to this cause of declines in cancer incidence with age across species. Moreover, it is expected that such a decline in metabolic and cell proliferation rates would reduce genomic instability and therefore should decelerate both cancer incidence and senescence; failing therefore to solve the paradox of the deceleration/decline of cancer incidence with age. A related set of hypotheses specifically involve trade-offs between cancers and other causes of death (reviewed in (Ukraintseva, et al. 2010)). A novel one (not discussed by Yashin and colleagues, and so far rarely considered in the literature) is rooted in the role of the accumulation of cells in senescent-state in tissues with age. We discuss this in detail in the next section.

Overall, whether decline in cancer incidence is related to selective disappearance at the population level, or molecular or physiological mechanisms occurring at the individual level, is a question of fundamental importance for life-history theory. Increases in mortality with age have been shown to be erratic in many species (Jones, et al. 2014). Whether this pattern occurs because of population levels bias in the estimation of vital rates aggregated at the population level (for example resulting in mortality plateaus), or the product of physiological mechanisms occurring at the individual level is the subject of intense debate. Resolving this debate is likely to require leveraging existing knowledge of physiological mechanisms underpinning causes of mortality. As a result, focusing on cancers may provide crucial progress towards solving this question.

Avoiding Death by Senescence: The Senescent-cells Theory of Ageing as a Unifying Theory?

In renewing tissues, mitotic cells (i.e. pluripotent stem cells or unipotent progenitor cells) divide to produce a specialized cell that assures tissue function, and a mitotic cell that maintains the tissue's rejuvenating capabilities. The mitotic cells' division is paramount to maintaining tissues' integrity as they age. Tissue stem cells avoid mutations, and excessively rapid telomere shortening, by dividing very infrequently (once every 40 weeks for hematopoietic stem cells according to (Catlin, et al. 2011)). Still, dividing cells are more at risk of accumulating DNA mutations (Moskalev, et al. 2013), and are thus at risk of turning neoplastic. This is why the number of cell divisions is limited, a fact first discovered by (Hayflick and Moorhead 1961) who showed that in vitro cultivated fibroblasts eventually stop replication even when space and nutrients are abundantly provided. Known as the Hayflick limit, this is due to the fact that cells enter a life-cycle state known as cellular senescence: "a stable arrest of the cell coupled to stereotyped phenotypic changes" (Lopez-Otin, et al. 2013). These phenotypic changes

(reviewed in (Campisi and d'Adda di Fagagna 2007, Kuilman, et al. 2010)) include a permanent growth arrest, mainly in G1 phase, linked to altered gene expression, and the secretion of pro-inflammatory molecules. Importantly, they also include resistance to apoptosis, meaning that senescent cells ultimately die by necrosis and are eliminated by phagocytosis.

Causes of cellular senescence are reviewed in (Campisi and d'Adda di Fagagna 2007, Collado, et al. 2007, Kuilman, et al. 2010, Lopez-Otin, et al. 2013). Most of them are associated with mechanisms preventing the replication of cells that have accumulated intracellular damage. As such, these mechanisms obviously play a crucial role in preventing cancers. First, telomeres shorten at each division. When telomeres become critically short, the *p53* tumour suppressor protein pathway activates either cellular senescence or apoptosis (although the drivers directing a given cell towards one or the other fate are, as yet, unknown). This limits the number of normal cell divisions, and contributes to replicative senescence (Kuilman, et al. 2010). It may also lead to premature senescence of cells that have initiated neoplasic transformations: as discussed above, in most cases of cancers, cells have mutated to higher activation of telomerase, and associated telomere length maintenance. Severe DNA damage (unrepaired DNA damage and chromosomal damage) can also activate p53 pathways towards senescence or apoptosis. Molecular biologists have also identified many pathways (e.g., Ras/Raf/MEK) that detect problems in the expression of specific genes which may induce cancers (called oncogenes). These pathways then activate the production of two major proteins $p16^{\text{INKa}}$ and $p19^{\text{AFR}}$ (in mice, or $p14^{\text{AFR}}$ in humans) which induce cellular senescence.

While cellular senescence plays a key role in preventing development of cancers, importantly, it may also have deleterious effects: accumulation of senescent cells in tissues may alter their renewal and function. There is ample evidence that cells in a senescent state do accumulate in tissues as individuals age (e.g., (Dimri, et al. 1995) in humans or (Herbig, et al. 2006) in baboons, reviewed in (Jeyapalan and Sedivy 2008)); although the kinetics of this accumulation over age remains unknown. Moreover, evidence that this accumulation is a cause rather than a consequence of ageing has long been largely circumstantial. Recently, the development of a technique allowing selective killing of senescent cells in tissues in transgenic mice (Baker, et al. 2011) allowed the issue to be resolved. Removal of senescent cells delayed onset of age-related disorders (Baker, et al. 2011, Ogrodnik, et al. 2017) and even increased lifespan (Baker, et al. 2016, Ogrodnik, et al. 2017). Unexpectedly, this procedure also delayed carcinogenesis, the opposite of what would have been expected under the "senescent cells theory of aging". Possible explanations for this discrepancy include delayed carcinogenesis as a consequence of the complicated experimental procedure on transgenic mice lineages used in these studies; or potentially non-linear patterns of damage accumulation in stem cells lineages as organisms age, under differential replication rates and relative importance of apoptosis and senescent-state transitions, making the age at which treatment is applied drive potentially different outcomes. A final explanation might link this outcome to induction of hyperplasia (often an initial stage of cancer) associated with the pro-inflammatory phenotype of senescent cells demonstrated in some cases of experimentally induced cellular senescence (Campisi 2013), as for instance in the case of genotoxic chemotherapies (Demaria, et al. 2017).

Overall, recent studies tend to confirm a hypothesis expressed by many molecular and cellular biologists: molecular pathways that suppress carcinogenesis (such as the p53–ARF pathway) are also involved in apoptosis, and cell entry into a senescent-state (Alderton 2007). As such,

cellular senescence or telomere shortening are "strategies that protect us from cancer" but also "might hasten our rate of ageing" (Finkel, et al. 2007). Accumulation of senescent cells in tissues with age could be an adaptive mechanism to prevent cancers, at the cost of a decline of tissue function and rejuvenation capacity with age. This maps onto the definition of a physiological trade-off in life-history theory, but, strikingly, it is framed as a trade-off between two mortality components: mortality by cancer, and mortality by other causes of death underpinning actuarial senescence. Following this logic through using an evolutionary biology perspective suggests that senescence could be optimal because it allows reduced cancer mortality at early ages, at the cost of increased mortality associated with deterioration of physiological function at older ages. It has curiously not yet (to our knowledge) been confronted with evolutionary theory of trade-offs and ageing; and even less been mathematically modelled.

A Preliminary Model of Cellular Dynamics with Age

Applying a mathematical model to investigate the implications of this trade-off is a key step: an integrated theory encompassing different forms of mortality may have the power to solve both Peto's paradox and the paradox of the deceleration/decline of cancer incidence with age. First, if accumulation of senescent cells with age is optimal, it may lead to non-continuous increases of mortality by cancer and senescence with age (investigated below), thus resolving the incidence by age paradox. Second, since an accumulation of senescent cells with age is, at least partially, a function of the number of replicating cells in a given organism, this sets it in line with all hypotheses proposed to solve Peto's paradox. If senescent cells optimally accumulate at different paces within tissues between species of different sizes and longevities, large and long-lived species may exhibit increased aging rates that would consequently decrease prevalence of cancers.

To investigate whether the accumulation of senescent cells with age could be optimal, and might lead to deceleration and decline of incidence of cancer with age, we develop in Box 1 a simple preliminary model of the dynamics of senescent and tumorous cells with age, and their effects on cause-specific mortality (see Box 1 for the definition of tumorous cells used here).This model incorporates three core attributes describing cellular dynamics, including, first, an increased risk that a cell becomes oncogenic as the organism's age increases (captured by the expression α_{x_i} which increases linearly from 0 to 1 over Φ time-steps, implying that age is "biological", corresponding to the unspecified amount of chronological time required for a proportion α_x of cells to turn neoplastic); second, a constant probability that tumorous cells enter a senescent state (reflected by the parameter σ); and third, a probability that a tumorous cell turns neoplastic as a function of the proportion of senescent cells in a focal tissue (modulated by a parameter r_{CS}). Two parameters are further used to translate the cellular makeup of an individual into morbidity: r_{OS} and r_{OT} respectively modulate the organism's morbidity as a function of the proportion of senescent and tumorous cells (both sources of morbidity are assumed to be increasing concave up functions of their respective fractions).

Box 1 — A demographic model for senescent and tumorous cells dynamics and their consequent effects on organism morbidity.

We consider a model organism dying only from two causes: cancer or mortality resulting from the accumulation of senescent cells in the organism. We denote S_x the proportion of senescent cells at exact age x , and T_x the proportion of tumorous cells of exact age x amongst non-senescent cells. Tumorous cells are defined as cells which have accumulated damage, and are at risk of turning neoplastic, and thus leading to cancer, but have not done so yet. Senescent cells do not further replicate, do not participate in tissue functioning and eventually die from necrosis (not formally modelled here). Tumorous cells are cells that have accumulated enough unrepaired deleterious mutations (or "hits"), yet have not been eliminated by apoptosis, putting them at risk of carcinogenesis if they do not enter into a senescent-stage. In one time-step, the proportion of new tumorous cells δ_x is defined by:

$$\delta_x = (1 - S_x - T_x)[\alpha_x + (1 - \alpha_x)(S_x)^{rCS}], \tag{0.1}$$

where α_x is the increasing proportion of cells becoming tumorous with age in a tissue (modelling a potentially increased genomic instability with age x) where there are no senescent cells. This might reflect either an increase of genomic instability with age, a decrease in apoptosis efficiency with age, or their interaction — i.e. apoptosis rates increase with age and the resulting gaps might allow replication of damaged neighbouring cells, allowing them to become oncogenic. The term $(1 - \alpha_x)s(x)^{rCS}$ captures the fact that tumorous cells might be more likely to be generated in tissues whose cell functioning is compromised by the accumulation of senescent cells; r_{CS} captures cells' ability to withstand the impact of the proportion of senescent cells. The proportions S_x and T_x are respectively then given by:

$$S_{x+1} = S_x + (T_x + \delta_x)\sigma, \tag{0.2}$$

where σ is the proportion of tumorous cells entering into senescence, and:

$$T_{x+1} = (T_x + \delta_x)(1 - \sigma). \tag{0.3}$$

In each time-step, the probability of surviving mortality via senescence and mortality via cancer are defined from and such that:

$$P^s_x = 1 - (S_x)^{rOS}, \tag{0.4}$$

where r_{OS} is the organism resistance to accumulation of senescent cells for its survival, and:

$$P^c_x = 1 - ((T_x + \delta_x)(1 - \sigma))^{rOT}, \tag{0.5}$$

where r_{OT} is the organism resistance to tumorous cells.

With this framework in hand, we numerically identified the optimal value σ^* that maximizes the organism's life expectancy for a set of parameters (Φ, r_{CS}, r_{OS}, r_{OT}) . It must be stressed that optimality is used here in its loose sense as a proof of principle that, somehow, natural selection may have an influence on the evolution of the trait (Orzack and Sober 1994) — here entrance in a senescent-state — which may in turn have shaped incidence of cancer by age and senescence.

Optimal σ^* is found for a large range of parameter as soon as $r_{OS} < r_{OT}$. Parameter r_{CS} has little effect on the optimum but controls the pace of the decline in cancer incidence at old age. One example that maps closely onto the empirical patterns of mortality we describe above is illustrated in Figure 4. The optimal strategy is to accumulate senescent cells at a low rate with $\sigma^* \in [0, 0.1]$. As a result, cancer incidence increases rapidly with age then decelerates and declines; thus leading to one potential resolution to the paradox of incidence of cancer with age.

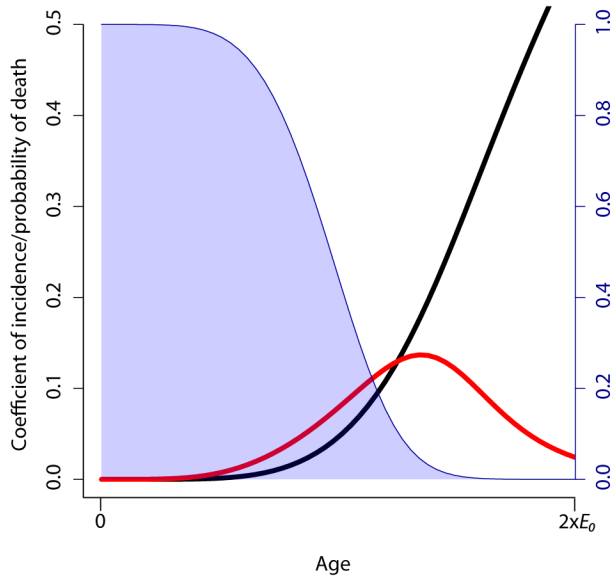

Fig. 4 Incidence of cancer (red) and probability of death resulting from the accumulation of senescent cells (black) for an optimal accumulation of senescent cells by time-step of $\sigma^* = 0.05$ for parameters $\Phi = 500$, $r_{CS} = 2.5$, $r_{OS} = 2$, $r_{OT} = 2.6$. In this case $E_0(\sigma^*) = 27$.

In the example illustrated here (Figure 4), the optimal strategy consists of diverting 5% of damaged cells per time step towards the senescent-state. This optimal strategy allows the accumulation of damaged cells early in life, leading to an early and rapid increase in cancer incidence (red line). In parallel, senescent cells accumulate at a slower pace, such that mortality by other causes rises more slowly than cancers (black line). Eventually, however, when ~40% of stem cells are in the senescent-state, cell proliferation becomes sufficiently reduced as to considerably decrease the number of new damaged cells per time-step; leading to a decline in cancer incidence, thereby preventing cancers from wiping out the entire cohort in a few time steps. Beyond this point in time, the increase of new senescent cells per time-step obviously also slows down. And yet, as the addition of even a small proportion of senescent cells considerably compromises tissue functioning, mortality by other causes of death continues to rise (black line). The optimal σ^* results therefore from a balance between mortality due to cancer early in life and mortality by other causes later in life.

Discussion

We have taken the first step towards characterizing the implications for life history evolution of *the senescent-cell theory of ageing*, proposed by molecular and cellular biologists. Senescent-cells (i.e. stem cells that have ceased to replicate) accumulate in tissues with age, and, according to the theory, this has two implications for individual mortality. First, diverting damaged stem cells towards a senescent-state prevents their replication, and thus prevents accumulation of more mutations, and the risk of that these cells become neoplastic, ultimately resulting in a reduction

in the risk of cancer. However, the accumulation of senescent cells in tissues compromises their rejuvenation capacity and functioning, and this leads to organismal senescence (Alderton 2007). Following this logic through, we suggest that senescence could be (at least in part) selected for because accumulation of senescent cells allows reduced cancer mortality at early ages, at the cost of increased mortality-associated deterioration of physiological function at older ages. Our preliminary model optimizes organism life expectancy as a function of the dynamics of non-senescent, tumorous and senescent cells within tissues over age, and their respective relationships with mortality from cancer and other causes. We show that accumulation of senescent cells with age can be under the influence of natural selection leading to a peculiar pattern of cancer age-incidence: an increase in early life followed by a deceleration and decline at old ages.

This novel result might resolve what we refer to as the "the incidence by age paradox" (see above). The paradox is that despite the fact that genomic instability is thought to be the proximal cause of both cancer incidence and senescence, and accumulation of mutations with age leads to an exponential rise of mortality by many causes, cancers oddly show reduced incidence at old ages (at least in humans and rats). Our model shows that this pattern may emerge as a result of the senescent cell theory of ageing. We also argue (although we did not formally model this) that accumulation of senescent cells with age might also resolve Peto's paradox, e.g., the fact that cancer prevalence is not a function of species' size and longevity. If senescent cells accumulate at different paces within tissues between species of different sizes and longevities, large and long-lived species may exhibit increased aging rates and consequently postponed incidence of cancers.

This preliminary model provides a "proof of principle" that trade-offs between mortality components can interestingly shape mortality patterns over age, and provides a first step in investigating these phenomena. But there are many key directions for further investigation, including (i) explicitly incorporating models of apoptosis and carcinogenesis (as in (Nunney 1993, Nunney and Muir 2015)); (ii) exploring the effect of size and metabolism across the tree of life for developing predictions about the potential of the senescent cells theory to resolve Peto's paradox (as in (Dang 2015)); (iii) incorporating more life-history parameters, including fertility and extrinsic mortality into the demographic model framed here. Moreover, the kinetics of senescent cells accumulation with age in tissues, and how it relates to cancer incidence by age, is not known. We hope that further modelling of tissues dynamics will help generate testable predictions on these functional relationships.

Is the senescent cells theory really a new theory of aging? In this field of work, there is a tendency to elevate a new finding related to senescence to the rank of an evolutionary theory. To us, rather than a new theory of ageing, and although this should be further discussed, this may be a core mechanism that fits within the broader umbrella of the Disposable Soma Theory (DST) (Kirkwood 1977, Kirkwood and Holliday 1979). The DST explains senescence by the accumulation of damage at different physiological levels due to the fact that some resources have to be invested in other functions, mainly to reproduction, then repair and maintenance. For the DST, senescence is the outcome of an evolved optimal allocation strategy under the constraints of physiological trade-offs. We see the senescent cells theory as a special case of DST where the allocation strategy concerns trade-offs between two mortality components, and determines the level at which organisms invest in tissue rejuvenation and slower actuarial senescence, at the

cost of an increased risk of cancer (of course, controlling for an organism's size, metabolism, phylogeny, mutation rates, efficiency in DNA repair and alternative immunological and anatomical mechanisms preventing cancer). Many authors have tried to reconcile DST with the Antagonistic Pleiotropy theory of aging (Williams 1957) *via* the existence of genes determining the allocation strategies between investing in early versus late fitness components (Kirkwood and Rose 1991, Partridge, et al. 1991). The senescent cells theory may very well be one of the rare examples for which evidence could be obtained: genes controlling the entrance of a cell into a senescent-state control the amount of cell division and will therefore control the amount of energy invested in rejuvenating tissue, thus defining the core processes underpinning cell dynamics over the course of an organism's life span. Selection on such genes will ultimately result in a balancing of the trajectories of the two types of mortality.

Considering empirical evidence for this and other theories on trade-offs between mortality components, we note the challenges in using aggregated population level data: in our model both cancer mortality and mortality by other causes will increase over most of the organism's life, making trade-offs more difficult to observe than, for instance, if the decline of one cause were correlated to the increase of the other. The only solution to addressing this challenge is to derive strong theoretical expectations regarding the age-specific shape of cause-specific mortality in a model where trade-offs of the kind we describe here are formally implemented. Furthermore, because individuals die only once, trade-offs are also not observable at the individual level if one has only information on cause of death. To investigate such trade-offs using individual data will require (i) grouping individuals according to factors likely to shape cause-specific mortality outcomes (for instance according to their genotypes or the experimental setting), or (ii) measuring biomarkers known to be good predictors of individuals' future cause-specific mortality, or (iii) empirically manipulating, over the course of an individual's life, the functions underpinning trade-offs between mortality components (for example, via the technique that allowed selective killing of senescent cells (Baker, et al. 2011)). Finally, comparison between species requires simultaneously comparing differential investment in physiological functions and relating these to differential pattern of cause-specific mortality.

In conclusion, trade-offs between mortality components are likely to be an important driver of life history evolution and yet have been strangely neglected by the field to date; possibly for both theoretical but also logistical reasons that we outline above. In particular, given the challenges to empirical investigation, we feel that such trade-offs cannot be investigated by evolutionary demographers acting alone, but detailed epidemiological and demographic data on causes of death must be allied with a nuanced understanding of the molecular, cellular, physiologic drivers at the individual and species level, likely requiring a profoundly interdisciplinary approach.

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32. Human Evolutionary Demography: Closing Thoughts

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A complete understanding of demographic patterns and behaviours is not possible without including the role of evolutionary processes. Many challenges in the social sciences, and in demography in particular, can be more readily met if they include the rich collection of perspectives, models, tools, and theories that evolutionary sciences can provide. Perhaps unexpectedly, the benefits of this inclusion can be indirect, as many benefits of an evolutionary perspective may take the form of a new way of approaching an old problem that leads to insights independent of any goal related to isolating the role of natural selection or adaptation. In other cases, the role of adaptation may have been under-appreciated and can lead to a different understanding of the mechanisms involved. To help human evolutionary demography improve going forward, we offer two general recommendations. One is improving the integration of contemporary developments in evolutionary thought about the role of culture and environment, such as dual-inheritance theory, epigenetics, and the role of social learning and cultural transmission. Many of these developments reflect an increasingly sophisticated understanding of cultural processes in the evolutionary social sciences, but also include conceptual improvements in the definition and understanding of core concepts like fitness and heritability. The role of culture may be a productive point of contact between the social sciences and evolutionary social sciences given shared interests in this area. Second is a call to re-invigorate evolutionary demography with some of the classical ideas that come from life history theory and population ecology, such as the use of energy and resource budgets to structure trade-offs, a focus on the role of ecological factors like density and resources, and the use of formal mathematical models.

One over-arching point in these closing thoughts is simply that a complete understanding of demographic patterns and behaviours is not possible without including the role of evolutionary processes. Evolutionary processes have shaped the histories of all human characteristics and contribute to their contemporary variation. The majority of demographic topics could therefore benefit from consideration of how evolutionary processes affect the topic under study. We are not suggesting that evolution should be the focus of every research article or project. For instance, even in the seemingly evolutionary-focused field of cell biology, one might find that the content of many papers is about describing chemical reactions across membranes with extensive focus on description of a chemical reaction and little to no direct reference to adaptation or evolution. Social science can be analogous to this; each individual paper need not focus on adaptation or evolution, as many will focus on pattern description, detailed understanding of processes, or

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evaluations of proximate mechanisms. Nevertheless, most social science topics would still be able to connect with processes and explanations grounded in evolutionary approaches. In fact, much of the gain of applying evolutionary explanation to demography has to do with framing demographic variation differently or recognizing the multitude of processes that can contribute to observed patterns. Note, this means we also consider that demographic topics should involve consideration of other disciplinary approaches, such as those from anthropology, public health, or sociology, involving an appreciation of the diversity of demographic processes, and the importance of how context affects them. As such, we are also arguing for even greater multi-disciplinary underpinnings to demography and for greater appreciation of demography across the social and biological sciences.

This volume presents a wonderful collection of chapters that shed light on the potential of evolutionary perspectives to inform research on a wide range of demographic topics. It leaves us with a thorough overview of the tools of the trade, the development of central ideas, and their application to overarching and applied subjects. A classically trained demographer presents the similarities that exist in the study of population, describes the natural complementarity of the approaches, and articulates the importance of top-down approaches in demography (Kreager chapter). We also have a succinct presentation of 10 major topics in evolutionary demography, written from the perspective of someone deeply rooted in evolutionary human sciences who also has an extensive background in demographic methods (Hill chapter). The potential for depth and nuance in a full-spectrum evolutionary approach is illustrated by the Tinbergen chapters (Jones, Vitzthum, Sheppard, Mace); hopefully in a way that alleviates the occasional tendency to (mis-) perceive tension between evolutionary and mainstream demography: evolutionary explanations are typically not alternatives to social science explanations, but complementary to them. We see in some chapters how anthropological and human-ecological frameworks enhance our understanding of how culture and ecology can shape demographic decisions (Shenk, Uggla, Blurton-Jones, Borgerhoff Mulder).

Several more chapters in this volume show that conventional or mainstream demographic topics like households, family structures, and culture can be fruitfully examined through a lens of evolution without forced invocations of adaptationism (Colleran, Borgerhoff Mulder, Rotkirch, Jennings, Wilfuehr, Lee, Tuljakpurkar). Others demonstrate that a diverse range of applied topics can be better-understood when framed with more deeply rooted evolutionary context, including our understanding of health, cancer, inequality and global development issues (Lawson et al., DeLong, Gurven et al., Pavard and Metcalf, Wells). Taxonomic depth to human variation is given by comparison across the great apes and beyond (Emery Thompson and Jones et al). We also see how evolutionary demography furthers not just our understanding of demographic processes but of evolutionary processes too (van Daalen and Caswell, Lee, Pavard). Fitness is a computationally intensive metric that can often only be measured by proxy, but we nonetheless have a state-of-the-art understanding for its measurement and interpretation (Moorad, Wachter, vanDaalen and Caswell, Orzack and Levitis, Lee).

With the vigour behind the approach well-established by the preceding chapters, we would like to close with a few thoughts about where some of these efforts might head, emphasizing key ideas that we think could use more attention. We also hope to guide interested readers to useful papers and concepts based on admittedly brief treatments of a number of complex ideas. With evolutionary approaches in hand, mainstream demographic topics can be moved

forward without making the research less applied or strictly about adaptation. The emphasis on multiple levels of explanation, multiple processes, and interdisciplinarity are all strengths that can contribute to fundamental demographic questions.

In an article about population growth for *Science*, one of us (RL) noted some shortcomings in common approaches to population forecasts. First, while resource constraints must ultimately limit the underlying positive and negative feedbacks that affect population growth, "population forecasts largely ignore economic and resource constraints." Second, in the absence of "grand dynamic theories" forecasters tend to use a descriptive framework called the demographic transition (Lee 2011). The strengths of evolutionary demography, as proposed in the chapters of this volume, could be leveraged to help improve both of these shortcomings: (i) the under-appreciation of constraints and feedbacks from the environment, and (ii) the reliance on description rather than on dynamical theory. Evolutionary approaches and tools of population and evolutionary ecology can provide dynamic theory and can also provide guidance for how to include ecological factors like population density or differential access to resources, whether the topic is population growth or some other demographic phenomenon.

For "how" human evolutionary demography can provide help like this more effectively, we make two overarching recommendations: embrace the new and appreciate the classic.

Embrace the New

The first is to *embrace the new* (selectively,[1] of course), meaning increased appreciation of recent theoretical and conceptual developments across the evolutionary sciences that are particularly valuable. Specifically, a modern understanding of heritability includes a number of inter-related developments for studying the non-genetic but nonetheless heritable (cross-generational) implications of key processes like cultural learning, social transmission, or epigenetics (Uchiyama, Spicer, and Muthukrishna 2020; Jablonka and Lamb 2014). Genetic evolution interacts with cultural evolution and understanding this interaction needs to be a major priority of evolutionary demography. The investigation and modelling of cultural evolutionary processes is simply called "cultural evolution" but this branch of theory focusing on how genes and culture co-evolve is "dual-inheritance theory" (Henrich and McElreath 2007). Dual-inheritance theory and studies of cultural transmission overlap with what demography knows as ideational approaches to the demographic transition (Cleland and Wilson 1987; Bongaarts and Watkins 1996). Culture and epigenetics both underlie what demography and public health know as household and community effects.

We call appreciating the significance of these processes and theories "new", in part because they are new relative to the development of natural selection, but there have been some fairly recent syntheses pointing out the importance of these pathways of inheritance for evolutionary theory in general (Laland et al. 2015; Jablonka and Lamb 2014; Uchiyama, Spicer, and Muthukrishna 2020). However, "new" does not imply under-developed. Evolutionary researchers who specialize in culture have a rich architecture of formal models and explicit theory that can help serve as bridges to mainstream demography and social science. The

1 We say "selectively" only to emphasize that these topics aren't being recommended simply because they are new, but rather because they are important, well-vetted and fundamentally important. They happen to be new relative to the core concepts in evolutionary theory.

theories and methods for each of these areas are established and mathematically formalized (Cavalli-Sforza and Feldman 1981; Creanza, Kolodny, and Feldman 2017; Boyd and Richerson 1988; Derex and Mesoudi 2020; Mesoudi 2011) and can provide points-of-entry for classically trained demographers to frame their research in terms of evolutionary processes.

Culture

For examples specifically relevant to population growth, culturally transmitted values regarding son preferences have been modelled using a dual-inheritance framework (Bhattacharjya et al. 2008) and there are several models that specifically target low fertility preference (reviewed in Colleran 2016). Relatively little work has been done including these processes in population forecasts, many (but not all) of which assume a universal preference for a completed fertility of around two births per woman and that in time most countries have an average fertility very close to this (e.g., United Nations World Population Prospects 2019[2]). Newcomers to these ideas about cultural evolution and inheritance could refer to: (Richerson and Boyd 2008; Mesoudi 2011; Colleran 2016).

By including the full complement of factors that affect inter-generational transmission, comes the further emphasis that there is a great deal more to an evolutionary approach than isolating genetic mutations or focusing strictly on adaptation. Much evolutionary research focuses on social transmission, the processes of learning and teaching, and ways in which genes and environments interact across generations with application to crucial applied topics like persistent inequality in health (Wells 2016; Wells 2010; Kuzawa 2005; Thayer and Kuzawa 2014; Kuzawa and Sweet 2009) (also Wells this volume, Lawson and Gibson this volume). However, evolutionary research also helps resolve a range of important questions from what causes aging to where cognitive biases come from. The recommendation to embrace some of these newer areas of evolutionary thought is not limited to newcomers to evolutionary analysis; those already well-practiced with human evolutionary demography might also find ways to shed new light on old problems by examining cultural or epigenetic processes in their areas of interest.

Research in cultural evolution brings some of the nuance that someone only familiar with the (often problematic or flawed) studies in human evolutionary research that grab the headlines might find lacking. As one of us has pointed out (RS), many of the headline-grabbing problematic studies are flawed because they are overly deterministic (e.g., make simplistic arguments of the form: "men do X, women do Y, because evolution") and lack acknowledgement of the obvious variation within- and between-populations that is typically the focus of much social science research (Sear 2020). Cultural evolutionary studies do not ignore the possibility of evolved species-typical traits; but they also acknowledge, and try to understand, how such traits interact with our social environment to produce behaviours and demographic outcomes. This greater incorporation of cultural evolutionary studies builds on the existing strengths in evolutionary demography and in human behavioural ecology, which considers how the environment shapes behaviours to produce within- and between- population variation in fitness relevant outcomes in our species (Cully and Shenk this volume, Blurton-Jones this volume, Hill this volume, Borgerhoff Mulder this volume). It is important to emphasise that cultural evolution and human behavioural ecology approaches to demography are able to help explain

2 https://population.un.org/wpp/Publications/Files/WPP2019_Highlights.pdf

contemporary variation in demographic patterns, since evolutionary approaches are sometimes misinterpreted to only encompass the study of human universals.

In a review of cultural evolutionary approaches to fertility decline, Colleran argued for more integration of cultural evolution into demography "as a means to develop multi-level models of fertility decline that emphasize the coevolution of economic and cultural change and not the a priori privileging of one over the other" (2016:2). Likewise, Creanza et al (2017:7786) put it rather matter-of-factly: "The literature on the interaction between cultural transmission and formal demography is quite sparse." Cultural institutions, such as economic systems or education, are often treated as the "non-cultural" alternative to a hypothesis that fertility norms or biased transmission is responsible for fertility decline when in fact the institutions themselves and the values that drive them are also products of human cultural evolution (Colleran 2016). Another important insight from work on cumulative cultural evolution that is perhaps under-appreciated in demography is that not only do cultural processes affect demographic behaviours but the structure of populations (size, age structure) affects the way cultural information accumulates and is transmitted (Derex and Mesoudi 2020). Further, cultural evolution is not just about inheritance, but includes the mechanisms and implications for social transmission among individuals both within and across generations (Bachrach 2013). It is worth noting that mainstream demography has also been criticised for inadequate incorporation of culture in its models (Petit and Charbit 2013), but also that some attempts to incorporate culture into so-far rather intractable demographic problems such as understanding variation in fertility have also considered evolutionary processes (the Theory of Conjunctural Action (Johnson-Hanks et al. 2011)), suggesting that both some mainstream and evolutionary demographers have come to similar conclusions about the importance of culture for demography.

Epigenetics

Another fascinating feature of this broader multi-faceted approach to the cross-generational transmission of information that affects human phenotypes is epigenetics. Epigenetic systems of inheritance involve molecular factors that affect how the genetic code is read or expressed.[3] The molecular factors themselves can be inherited or may be physiological responses to stressful environmental conditions. While the way that epigenetic systems of inheritance function must surely be governed by genetic systems of inheritance, they make it possible for environmental signals to affect future generations by essentially turning genes on and off. This opens a pathway for the environment to affect some aspects of how traits manifest across generations without directly changing the underlying genes. Because the conditions experienced by a mother during or before pregnancy may affect the biology and physiology of her offspring and grand-offspring, there is a legacy effect of certain environmental factors that affects many outcomes relevant to demography, public health, and other fields (Kuzawa and Thayer 2011; Furrow, Christiansen, and Feldman 2013; Benyshek 2013). If these processes are not adequately understood, they could be inadvertently attributed to genetic factors because the changes can follow family lines if the factors that produce the signal triggering the epigenetic response are experienced persistently

3 The most recognizable form of epigenetics is that it makes it possible for cells to differentiate into different types as seen in bone cells and muscle cells and the like, which is accomplished by turning parts of the full DNA sequence on and off, but a more subtle version of this 'turning on and off' happens within and between generations and affects a range of factors from longevity to mental health.

across generations (Kuzawa and Sweet 2009). Numerous studies have shown enough empirical support for epigenetic pathways that a "proof-of-concept" is well established (see the study of the Dutch Hunger Winter by Heijmans et al (2008) for a well-known example). Associations between parental age and offspring health, a topic of interest in demography (Goisis et al. 2018), might be mediated by epigenetic effects (Markunas et al. 2016). However, a great deal more work could be done on the influence of epigenetics on demographic behaviour, as it is increasingly clear that these maternal signals from certain forms of stress, household effects, or parental age can be carried across generations.

A more nuanced understanding of the mechanisms of inheritance would be one very productive step forward for evolutionary demography, as well as a means of helping alleviate concerns with evolutionary approaches that are based on extremely problematic misapplication of evolutionary theory. How traits are transmitted between generations is widely misunderstood, in both the social sciences and evolutionary social sciences. Arguments about inheritance of traits are also misused, as "hereditarian" arguments about supposedly "genetic" group differences in traits such as intelligence seem to be on the rise again (Sear 2021). Increasing familiarity with, rather than closing the door on, the full suite of evolutionary processes will provide more effective tools to refute such studies. The genetic underpinnings of traits with relevance to human cognition, behaviour and demography are extremely complex, and are affected by a multiplicity of factors, including epigenetics, social determinants of health, and other gene-environment interactions, such as cultural evolution (Uchiyama, Spicer, and Muthukrishna 2020). An evolutionary demography that is able to develop and promote an appropriately nuanced understanding of the complexity of inheritance mechanisms would both be beneficial scientifically and might help dispel myths used to promote scientific racism and classism.

Appreciate the Classics

Second, in addition to (selectively) embracing the new, we recommend *appreciating the classic* as a means of continuing to advance evolutionary demography. In particular, human evolutionary demography would benefit from maintaining the key formative ideas from life history theory (sensu Charnov 1991; Charnov 1993; Stearns 1992; Roff 1993; Kozlowski 1992),[4] population ecology (sensu Ginzburg and Colyvan 2004; Turchin 2003; MacArthur and Wilson 2001; May 1974; Roughgarden 1971), and where the two overlap (Abrams 1993; Reznick, Bryga, and Endler 1990; Fowler 1981). As Mace said in a 2014 article in *Demographic Research* "Biologists generally define evolutionary demography as the application of life history theory to population processes" (Mace 2014). Life history theory was initially practiced mostly by biologists but shares the same main endeavour as evolutionary demography: to apply evolutionary theory to demographic processes. However, contemporary human evolutionary demography has become at times detached from the main strengths of classical life history theory. Ideas that could use wider attention include using the principle of allocation to study trade-offs (Bolund 2020), the roles of population density and resources (Laskowski, Moiron, and Niemelä 2021),

4 This use of 'life history theory' exclusively refers to the original sense of the term originating in evolutionary biology and does not include the psychometric approaches used in some subfields of psychology, which have little to nothing to do with the theory developed in evolutionary biology (Sear 2020; Nettle and Frankenhuis 2019; Stearns and Rodrigues 2020).

more rigorous and theoretically grounded measures of fitness, and the use of formal analytical models (Jones and Bird 2014; Moorad and Nussey 2016). Life history theory and evolutionary ecology have used these things a lot, and still do, but we think there could be greater use of these classic techniques in human evolutionary demography.

Trade-offs and Energy Budgets

Formal models that incorporate energy budgets and trade-offs are central to classical life history theory. A highly influential example demonstrating how these concepts led to a deeper understanding of human life history evolution is Charnov's mammal life history model (1991; 1993). In it, Charnov takes an empirical finding as a given (the size of an offspring when it is energetically independent from its mother has a central tendency of about 1/3 of its mother's size) combined with the general shape of a growth curve that is typical of mammals and birds. He then derives a series of expressions that link demographic/life history traits together. Importantly, the model recreates known allometric patterns (traits like life span and age at first reproduction vary consistently with the adult size of an animal) and also articulates residuals from the average patterns across several demographic and physical characteristics; e.g., an animal with a larger than expected size at weaning will tend to have a later age at first birth, a longer lifespan, a slower growth rate, and a slower birth rate. Charnov's mammal model was tested a few years after its publication and held up extremely well in a cross-species study using high-quality life table data (Purvis and Harvey 1995) (the empirical evaluation in Purvis and Harvey (1995) also lends strong support for many of the central ideas in life history theory in general).

Having a theoretically explicit and mathematically formalized model articulating why biologists observed so much structure in demographic traits across species proved to be highly useful, and was extremely influential for several prominent evolutionary demographers and biological anthropologists (Jones 2011). Since its publication, Charnov's life history model (and related life history insights) has guided research on many topics, from why animals change size if they migrate from a continent to an island (Palkovacs 2003) to the relationships among a large "expensive" brain, slow growth, and long life in humans (Kuzawa et al. 2014). It was applied to primates and humans in what became the "life in the slow lane" approach to thinking about why primates are different from other mammals (Charnov and Berrigan 1993; Walker et al. 2006)(the "slow lane" refers to slower rates of growth and reproduction). In a thorough review of how life history theory explains the evolution of these canonical primate and human characteristics, Jones (2011:710) referred to the main question poised in Charnov and Berrigan (1993) ("Why do female primates live so long and have so few babies?") as "the central question for understanding primate life histories".

Indeed, the focus on trade-offs and cross-species analysis of primates and other animals recently led to a key observation regarding the human life history pattern. Human body growth is extremely slow on average from birth to maturity (humans have growth rates so low that they are closer to a boa constrictor than a mammal of similar size, Walker et al. (2006)) and has characteristic changes in velocity that occur during development. Human body growth slows down dramatically mid-childhood and stays low for a few years, a pattern that we do not see in other large primates. While many researchers had hypothesized that brain development might be responsible for the slowed body growth, the life history emphasis on trade-offs led

Kuzawa et al (2014) to study the energy metabolized during growth, using direct measures of glucose consumed by brain tissue, in humans, and compare that to changes in body growth. Indeed, they found that brains consume the most energy at about age 5 at a time when body growth is at its slowest and also found a clear inverse relationship between the rate of energy consumed by the brain and the rate of body growth across development. Showing that this trade-off, previously identified with cross-species comparisons, is so clearly evident in the physiological processing of glucose during development within one species, humans, was a major step forward and the culmination of years of research that was at least partly inspired by Charnov's mammal model. We still have a lot to learn about how these trade-offs affect human growth and development, and how evolutionary pressures moulded these responses across the evolution of mammals and primates.

Because Charnov's mammal model emphasizes the co-evolution of inter-related traits, anthropologists started thinking about ways that human patterns systematically differed from those of mammals and what this might tell us about how the evolution of some uniquely human characteristics.[5] For example, the "grandmother hypothesis" appears several times in this volume (Tuljapurkar chapter, Dillon et al chapter). While this hypothesis has a few sub-variations, the main themes link the slow growth rates and extended periods of dependency that we observe among human children to a sexual division of activity and long post-reproductive life spans that were seen among adults in the foraging populations where these anthropologists worked (e.g., the Ache of Paraguay or the Hiwi of Venezuela or the Hadza of Tanzania; see Blurton-Jones Chapter and Hill Chapter). Identifying these relationships as an interesting and important demographic problem was inspired in part by Williams and Hamilton's work on senescence (Hamilton 1966; Williams 1957), but also by the theoretical framework provided by Charnov's mammal model (Hawkes et al. 1998).

Furthermore, the influential embodied capital theory of human evolution explains the link between the high-skill niche characteristic of human substance patterns, the complexity of human social interactions, and low adult mortality rates to the notably late-ages at which human foragers have their "peaks" in terms of ability to proficiently forage in complex environments (Kaplan and Robson 2002; Kaplan et al. 2000). The linkage of these different characteristics presented an interesting research problem because evolution tends to favour early and fast reproduction and studies of human foragers show that the most successful foragers, in terms of calories produced per unit time, were older individuals well beyond the ages of peak health and agility. It takes a long time to learn the skill needed to be a human forager and this extended learning time is so instrumental to our package of life history traits that mortality rates have to be extremely low relative to our nearest ancestors for this extended period of learning to pay off (this observation is important to models of cultural evolution as well, because childhood is an extended time of energetic dependence where a lot of social learning occurs). Part of the embodied capital approach suggests that the slow growth and brain development by children before adulthood, when they are fairly "bad" at getting their own food, is made possible by the excess production of older individuals (Lee Chapter). While a lot more could be said about the history of research on this topic and its anthropological relevance, our point here is that some

5 Perhaps not coincidentally, many of the early influential anthropologists working in this area overlapped with Charnov as graduate students or faculty at the University of Utah in the 1980s.

highly influential work on the human life history, and hence the evolution of demographic traits, was inspired by an elegant model that used trade-offs and mathematical formulation.

Population Density and Resources

Evolutionary approaches often include the role of ecological variation as a predictor of, or constraint on, change. Hence, they also highlight how variations in vital rates are influenced by social and ecological factors like population density, competition, and resource availability, all of which affect human populations. One of the first influential works in demography, Malthus' 1798 "Essay On the Principle of Population…" focused on exactly these issues, but — for understandable reasons — they have fallen out of favour in contemporary approaches of population. We suggest they could use more attention, with appropriate caution, in areas like projecting population sizes of the future.

Despite a relative lack of attention to these issues, a few classic works in demography have shown that population densities influence vital rates. For instance, Lutz et al (2006) showed that population density was negatively associated with fertility outcomes and preferences in a time series analysis of 145 countries. They recommended that population density be included in research on fertility determinants but did not mention population projections directly.

In an article that in many ways anticipated key strengths of evolutionary demography, Lee (1987) investigated density-dependence and homeostasis in historical data sets for Europe. This analysis was in part motivated by noticing the prominence of population density in animal ecology compared to its near absence in human demography. For countries with long time depth, Lee found a strong negative relationship between population density and income (detrended). For example, across Europe as a whole, he observed a statistically "large" effect of density on real wages and a clear sign of density-dependence on the ability of people to gain a key resource for the period from 1260 to 1840. The relationship between density and fertility was also negative. In the "modern world" Lee found that the nature of population homeostasis has changed as the constraints on growth are drastically different. When fertility is low and land has less of a constraint on economic growth, because inputs become less tied to photosynthesis and land area, constraints on growth are less tied to population density, but increasingly more tied to the actual availability of energy (Wrigley 2013).

Other historical analyses have found correlations between wages and population growth and other lines of evidence indicating density-dependence, with effects that weaken with the transition to fossil fuel economies (Wrigley 2013; Wrigley 1990; Kander, Malanima, and Warde 2014). The work of historical demographer Tony Wrigley showed that the demographic transition was associated with a large increase in inputs from fossil fuels and these change the way that land constrains growth and production (Wrigley 2013; Wrigley 1990). Once the use of coal became a major part of the English economy, relationships decoupled between population growth rate and consumables and between population growth rate and real wages, meaning that the nature of density dependence changed dramatically. In contemporary societies, many demographic traits correlate with energy availability at the national level (Burger, DeLong, and Hamilton 2011; DeLong, Burger, and Hamilton 2010), and such relationships are rarely if ever included in formal projections of future population size (Lee 2011).

The realization that density effects are different for post-industrial humans than in earlier times has led many to assume that they must not matter at all. The common dismissal of density

and ecological factors in demography probably has less to do with theoretical perspective than with the widely publicized predictions of impending disaster for humans that have not come to be but ignoring socio-ecological factors is ultimately short-sighted. The dismissal of density and ecological factors in demography may also be related to their politicisation: Malthus is read as a call to maintain the political status quo, and twentieth- and twenty-first-century concerns about rapid population growth and environmental degradation have been used to promote population control, at the expense of individual human rights. This potential for political misuse must absolutely be borne in mind in future research, alongside acknowledgement that there is little to be gained from making strong assumptions about specific fixed and static carrying capacities, but there is a great deal to be gained from understanding the mechanisms or pathways that alleviate density-dependent pressures or that change the way they operate (again, such mechanisms could and should be included in projections of population growth).

More abstractly, forecasters and social scientists generally need to realize that human populations have been growing for many generations and are far from an equilibrium state. The theoretical implications of human non-equilibrium dynamics is not at all well understood and rarely formally included in discussions of population growth (DeLong and Burger 2015; DeLong, Burger, and Hamilton 2010). Population forecasts commonly take for granted that a future equilibrium state exists (often that preference and behaviour will fix near two births per couple, globally), but the factors that draw us toward it, or those that interfere with density-dependent feedbacks, have not been thoroughly studied and applied in the area of population growth. Something as socially complicated as desired family size likely has inputs from many factors and these need to be understood before assuming a stasis and universal preference for the same value (Burger and DeLong 2016).

One of the reasons ecologists expect density to affect population dynamics, is that as population density increases, it will usually pass some threshold after which individuals in the population experience reduced access to a key resource. This kind of thinking has been used to address several questions of interest in ecology, such as what group sizes are optimal for different species or populations. Typically, there are benefits and costs to adding group members, such as reduced risk from predators up to a point where resources become limiting. Part of the human demographic relevance of population density is its effect on resources, but this is not the only reason for human demographers to be interested in population density (especially given humans have repeatedly shown they can change the exact relationship between population density and resources with technological innovation). Population density also affects social interactions and cumulative cultural evolution. As population size increases within a given area, density necessarily increases as well and density may interact with other factors, like mortality risk by age, or competition over favoured nesting/housing sites, or the nature and complexity of social hierarchies, or the number of cultural models one observes during childhood, or psychological mechanisms affected by the number of people, accidents, and deaths one observes in an urban setting which can skew one's own perception of risk. One of the mechanisms of cumulative cultural evolution is socially observing a wide range of social models for the reproduction and change of technologies or norms, as observing a greater variety of these in turn seems to spur more rapid innovation rates.

A modern understanding of energy, and the extra-somatic resources made possible by human cooperation, conflict, colonisation, and technological developments, could be essential

for resolving apparent tensions between Boserupian (technology and innovation focused) and Malthusian (constraint focused) approaches to thinking about and modelling population growth. Likewise, urbanization is such a key aspect of globalization and the near- to mid-term future of many human populations. Seeking general theory and approaches for how the changes that accompany urbanization affect demographic and social behaviour will be an increasingly important endeavour.

Better/Appropriate Measures of Fitness

Evolutionary demography could also focus on, and more-widely adopt, explicit and theoretically valid measures of fitness (McGraw and Caswell 1996, vanDaalen and Caswell this volume, Moorad this volume). This will not only improve rigour in the field but will also help move forward our understanding of the demographic transition. A great deal of evolutionary research in the human sciences uses proxy measures for fitness. Sometimes this is a matter of necessity as the data requirements of a good fitness measure can be hard to meet, but for all the conceptual centrality of fitness to evolutionary theory, it is not appropriately quantified near often enough. For example, we know stunningly little, even at the descriptive level, about changes in actual fitness during the demographic transition and such knowledge would be useful for both accurate description and explanation of the transition.[6] We rarely have a detailed study of a valid measure of fitness applied to a long time series during the transition, nor do we have thorough understanding for how the components of fitness like child mortality and age at first birth change over time or how the strength of their correlation with fitness changes with time. This would help map out the timing and magnitude of fitness change or the correlations among fitness and its components (timing and number of births, survival, population growth rate).

The demographic transition is defined by reductions in fertility, that typically follow reductions in mortality, to very low levels resulting in populations that often have fertility levels below replacement. Many studies have looked at correlates of fitness but we know from evolutionary demography that such studies can be highly misleading, especially if changes in timing are one of the variables that can affect variation in fitness (McGraw and Caswell 1996). Variation in timing of the age at first birth is important for historical, evolutionary, and policy reasons, but the fitness implications of this cannot be understood using many common definitions of fitness that do not capture variation in timing. Likewise, the demographic transition is sometimes used to argue that human fertility behaviour is inherently sub-optimal or non-fitness maximizing. While this is certainly the outcome at the individual level in low-fertility contexts, for such an important topic there is a great deal to learn about whens, wheres, and hows of the fitness-aspects of the demographic transition and the individual- and

6 Two population-level quantities are especially likely to be used as fitness measures. One of them, r or the intrinsic rate of increase, tends to actually increase in the early part of the transition as mortality declines and only falls to pre-transition levels very late after fertility and mortality are both at very low levels. Another, NRR or the net reproductive rate, stays relatively flat (unchanging) through a large temporal swath of the middle of the transition. Neither of these capture within population variation in mean fitness, which is likely more important for explaining the impact of individual level decisions and behavioral responses to changing circumstances. The demographic transition is often referred to as something that is just 'bad' for fitness, and while we need to know a lot more than we do, we know there is a lot more to it than that.

group-level decisions and dynamics that co-occur with it. How various mechanisms of change are rate- or scale- dependent also needs more attention, as does the role of non-equilibrium dynamics. For example, what kinds of biological and cultural processes are especially relevant or irrelevant when change is so rapid and appears to be driven by individual adjustments to rapidly changing external circumstances?

For the demographic transition, ages of marriage and first reproduction change a lot and, importantly, neither age at first birth nor total fertility has a simple linear relationship with actual fitness. Individuals with the same reproductive success (total offspring produced) will have different fitness based on the timing of the births. The rate at which the population is growing and the fertility levels of the rest of the population also affect fitness. Linking back to earlier sections, there has been a resurgence of interest in the importance of population density on life history dynamics in evolutionary biology. Key to applying these insights to human evolutionary demography is an improved understanding of fitness (Dańko, Burger, and Kozlowski 2017; Dańko et al. 2018; Vries, Galipaud, and Kokko 2022). We need to know more about these relationships to simply have an adequate description of the demographic transition that would guide theory on how social/economic institutions interact to lead to the opportunities and perceptions behind the clearly non-fitness maximizing fertility decisions we see late in the transition. A dual-inheritance framework would help, by including cultural and genetic factors that affect fertility and mortality related decisions, but appropriate measures of fitness are needed as well. The case of more robust fitness measures and examples of their application can be found in these papers: McGraw and Caswell (1996); Korpelainen (2003); Jones and Bird (2014); Moorad (2013).

Many Benefits of an Evolutionary Approach

Some of the benefits of human evolutionary demography are not about adaptation. Likewise, evolutionary approaches apply to cases where non-adaptive behaviours seem common (like below replacement fertility). The focus on multiple levels of explanation and sources of variation can result in looking at a problem in a different way or may help guide questions that link mechanism and process to observed patterns. For example, an interest in how natural selection affects change in any given trait or behaviour requires an understanding of not just the mean or most typically observed, but of the variation as well. One reason for emphasizing the variance is that natural selection acts on heritable variation and if there is no heritable variation, there is a narrower range of phenotypes that can be modified across generations by differential fertility and mortality (Crow 1989). A second reason is that fitness is a relative measure. As such, understanding how evolutionary processes are affecting a given observed behaviour or physical characteristic depends on how it compares to the values (and associated strategies) of the rest of the population (e.g., a completed fertility of 3 is low if the population average is 6 but quite high if the population average is 1.5).

The emphasis on both the mean and variance present in many metrics designed to quantify evolutionary processes led two evolutionary demographers, including one of us (OB), to notice that studies of the fertility transition are often focused on changes in mean fertility while the variance is rarely discussed in detail (Hruschka and Burger 2016). They analysed variance in completed fertility across 72 low- and middle- income countries, emphasizing how variance changed as the mean declined, an important and overlooked topic, which was motivated by

an evolutionary approach. Yet, the findings of their analysis did not involve arguments about evolutionarily optimal fertility levels nor about changing allele frequencies, but were rather pragmatic and applied in nature. A lot of the variance in fertility among individuals is likely due to stochastic processes rather than to individual-level variables like wealth or education (91% of the samples they analysed were consistent with a Poisson process). This key finding suggests that there appear to be hard limits to how much of the variation in fertility can be explained by individual differences. Many studies of fertility at least implicitly assume that most of the variation in fertility behaviour among individuals is explainable with variables of the sort typically used in regression analysis (education, family size, wealth, media exposure, etc), when a lot of this variance may be inaccessible to such measures (if the underlying process is indeed consistent with a Poisson counting process). Moreover, the relative importance of individual-level variables was likely greater at low levels of fertility than at medium or high levels, which is again probably not the kind of finding that social scientists would typically associate with "an evolutionary approach", but is indicative of the strength of the multi-disciplinary and multi-level approach of human evolutionary demography. Indeed, most of the results of this analysis were "applied" in nature and of high practical relevance to public health and demography with seemingly little to do, at the surface level, with explanations of how fertility patterns evolved. That is, the benefits came from the framing of the question rather than a quest to find an adaptive explanation for an observed trend.

Melting Dichotomies with Evolutionary Demography

In these Closing Thoughts, we are emphasizing some strengths of evolutionary demography and suggesting some paths forward to help achieve further integration between social and evolutionary sciences. The combinations of the emphasis of evolutionary demography on multiple levels of explanation and interdisciplinary nature combined with realizations that multiple processes influence phenotypic change, that the role of culture can be explicitly modelled and analysed, and that non-adaptive perspectives are both possible and common, lead to the melting of several long-held structural dichotomies that have served to hold research back.

Many dichotomies end up converging on an answer that takes some form of "a bit of both". By incorporating contemporary understanding of heritability one such dichotomy that dissolves into "a bit of both" is nature vs. nurture. Research in cultural transmission and epigenetics has demonstrated that traits that are strictly one or the other are exceedingly rare. Inclusion of understandings of cultural transmission and epigenetics could further dissolve many of the problematic issues that come from placing nature and nurture in opposition to each other while also identifying the mechanisms for how the genetic and biological interact with and are part of the social and the learned. Another dichotomy that disappears is that between structural and ideational explanations of the demographic transition. With a contemporary understanding of evolutionary processes, it is difficult to claim that the demographic transition could be all large-scale macro-economic drivers. However, it is perhaps even less tenable to claim that it is totally driven by norms spread by television and/or imitation of behaviours one observes in their neighbourhood. The economic circumstances, the actual and perceived trade-offs for investing in capital, and the processes of innovation and imitation that underlie cultural transmission of fertility norms are inter-connected. A modern science of cultural evolution

that includes attention to trade-offs recognizes both of these factors. A third dichotomy that the recommendations here can help dissolve is that between Boserupian and Malthusian processes of population growth. Do population processes drive innovations that lead to technological developments that in turn relieve population pressures? Yes, of course. Are population growth rates and sizes ultimately governed by finite resource structures? Yes, of course. Both are true. The challenge is not letting an interest in one of the processes, often driven by the scale of a question or the dataset to which it is applied, lead to the denial of the other.[7]

Traditional and evolutionary demography need one another. In our view, one of the negative perceptions of any field with "evolutionary" in the name derives from the worst instances of attempted evolutionary analysis that often seem to get the most press (to be clear, this describes a minority of evolutionary research). Such studies are based on rather hackneyed post-hoc adaptive explanations, poorly designed sampling strategies, occasionally even obviously political motivation, or all three. Most evolutionary research is not like that. It is increasingly clear to us that an eyeroll or snarky tweet is not sufficient response to this minority of evolutionary research, which continues to do so much damage to the attempts of many researchers to cross the evolutionary-social science boundary. So, two additional recommendations we have for evolutionary demographers, and other evolutionary social sciences, are (i) to take much more active steps to improve rigour in the evolutionary field, and (ii) to continue to break through disciplinary walls by finding points of overlap among evolutionary and social science perspectives that strengthen both science and policy. The Chapters in this volume provide excellent examples for both of these points.

Across these dissolved dichotomies are many opportunities to advance research and understanding of a variety of topics. Certainly, the modelling of population growth and the components that contribute to it are strong contenders for areas that could be improved with more input from multiple-levels of explanation and dynamic models informed by theory. But many other topics, from persistent inequality to wealth disparity to family planning, that are reasonably considered as fundamental demographic topics could be advanced with the nuanced perspectives presented by the chapters of this volume. Indeed, in today's world, approaches that have the potential to dissolve dichotomies or reduce polarization are much needed across sectors of science, policy, and elsewhere.

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7 From the great demographer Wrigley: "There is an important sense in which the problem identified by the classical economists remains with us today since, as long as fossil fuels provide the bulk of the energy consumed in advanced economies, difficulties are postponed rather than indefinitely set aside."

8 Note this chapter has been posted on the Open Science Framework website since 26/02/2022, after it was accepted for publication, so the references will reflect when the chapter was written and not the OBP publication date.

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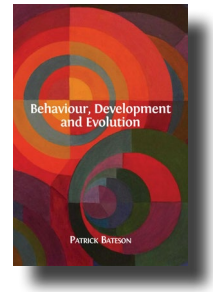

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Daniel Nettle

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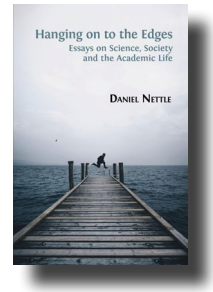
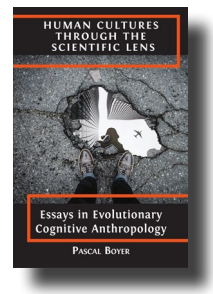


Human Cultures through the Scientific Lens
Essays in Evolutionary Cognitive Anthropology
Pascal Boyer

https://doi.org/10.11647/obp.0257



Human Evolutionary Demography

Edited By Oskar Burger, Ronald Lee and Rebecca Sear

Human evolutionary demography is an emerging field blending natural science with social science. This edited volume provides a much-needed, interdisciplinary introduction to the field and highlights cutting-edge research for interested readers and researchers in demography, the evolutionary behavioural sciences, biology, and related disciplines.

By bridging the boundaries between social and biological sciences, the volume stresses the importance of a unified understanding of both in order to grasp past and current demographic patterns. Demographic traits, and traits related to demographic outcomes, including fertility and mortality rates, marriage, parental care, menopause, and cooperative behavior are subject to evolutionary processes. Bringing an understanding of evolution into demography therefore incorporates valuable insights into this field; just as knowledge of demography is key to understanding evolutionary processes. By asking questions about old patterns from a new perspective, the volume—composed of contributions from established and early-career academics—demonstrates that a combination of social science research and evolutionary theory offers holistic understandings and approaches that benefit both fields.

Human Evolutionary Demography introduces an emerging field in an accessible style. It is suitable for graduate courses in demography, as well as upper-level undergraduates. Its range of research is sure to be of interest to academics working on demographic topics (anthropologists, sociologists, demographers), natural scientists working on evolutionary processes, and disciplines which cross-cut natural and social science, such as evolutionary psychology, human behavioral ecology, cultural evolution, and evolutionary medicine. As an accessible introduction, it should interest readers whether or not they are currently familiar with human evolutionary demography.

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