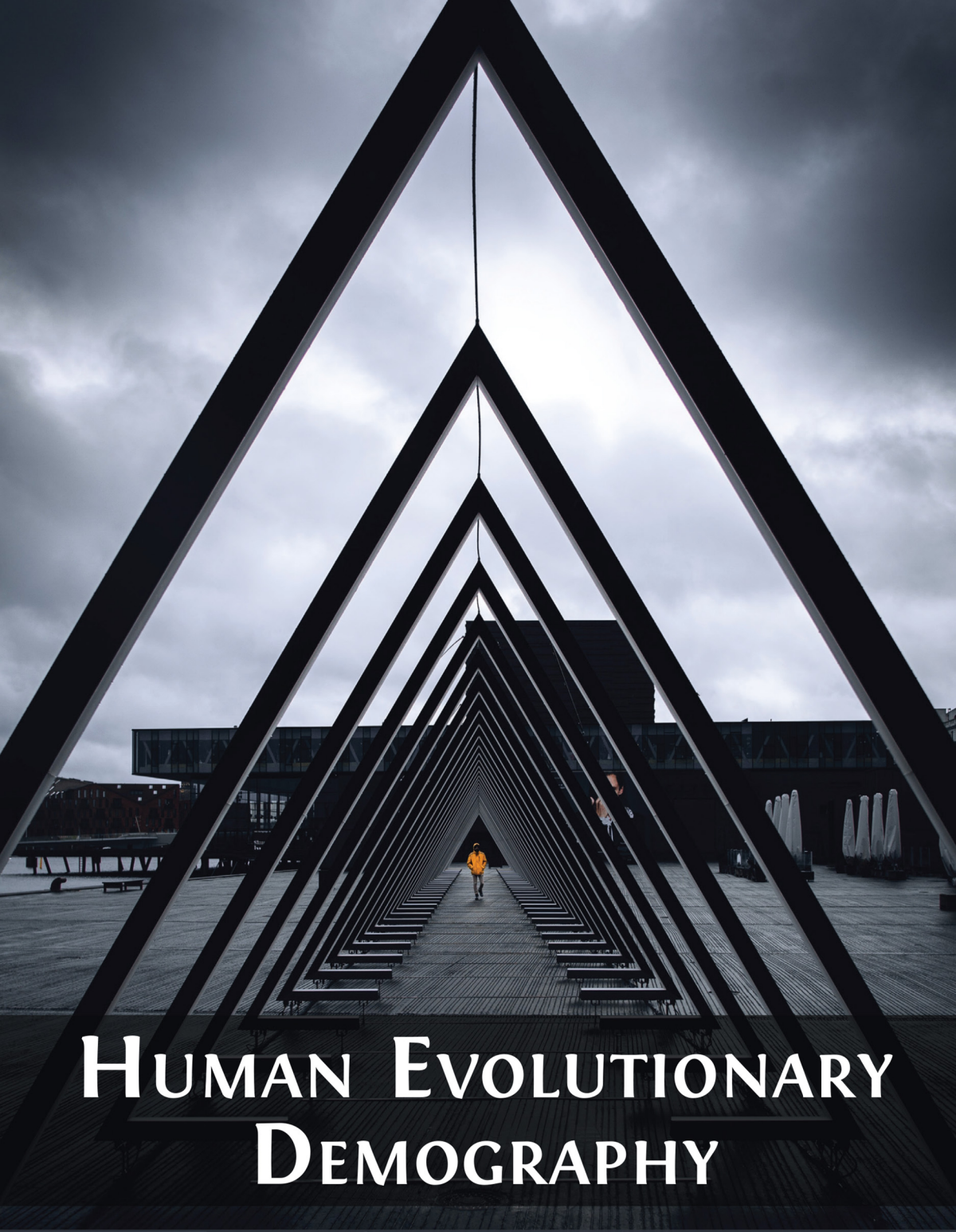

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21. The Challenges of Evolutionary Biodemography and the Example of Menopause

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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