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ARTICLE

The Human Post-fertile Lifespan in Comparative Evolutionary Context

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There persist two widely held but mutually inconsistent views on the evolution of post-fertile lifespan of human females. The first, prevalent within anthropology, sees post-fertile lifespan (PFLS) in the light of adaptive processes, focusing on the social and economic habits of humans that selected for a lengthy PFLS.[1–3] This view rests on the assumption that human PFLS is distinct from that of other species, and focuses on quantifying the selective causes and consequences of that difference. The second view, prevalent within gerontology and comparative biology, emphasizes that PFLS is a phylogenetically widespread trait[4–6] or that human PFLS is predictable based on life-history allometries.[7] In this view, human PFLS is part of a broad cross-species pattern and its genesis cannot, therefore, rely on human-specific traits. Those who advocate the second view have questioned the "special pleading" for human specific explanations of PFLS,[4] and have argued that human PFLS is quantitatively greater but not qualitatively different than PFLS in many other animals.[5,8] Papers asking whether human PFLS is explained by the importance of mothers more than grandmothers, whether paternal or maternal grandparents have more of an effect on child survival, or who is providing the excess calories are associated with the first view that assumes the need to explain the existence of human PFLS on the basis of a uniquely human socioecology. Anthropologists largely see human PFLS as derived, while comparative gerontologists point to evidence that it is one instance of a ubiquitous cross-species pattern. The two groups generally occupy non-overlapping research circles, in terms of conferences and journals, and therefore interact little enough to largely avoid the need to reconcile their views, allowing the persistence of misconceptions in each field. Our goal is to identify and address the most important of these misconceptions and thereby make clear that both of these seemingly incongruent views contain valid points. We argue that two distinct but related traits have been lumped together under the same concept of "post-reproductive lifespan," one (post-fertile viability) that is tremendously widespread and another (a post-fertile life stage) that is derived to hominins, and that the differences and connections between these two traits are necessary for understanding human life-history evolution.

The human post-fertile lifespan is so long that it seems obvious that it is something different, and hence a derived feature of the human life history. Anthropological approaches to explaining PFLS often assume, on the basis of length alone, that human PFLS is indeed a derived trait. However, to examine the *derivedness* of human PFLS, we must examine whether it is a product of special environments, whether it is the predictable result of cross-primate patterns, and whether it is a qualitatively different character state unique to humans. It cannot be derived if it arises because modern populations survive longer than their ancestors due to technological improvements. It cannot be derived if it results simply from primate scaling relationships, such that we would expect any primate of human brain and/or body mass to have similar post-fertile lifespan. Also, it cannot be considered derived if human PFLS is simply a longer version of the same trait found in related species. There are competing claims regarding whether human PFLS passes each of these tests. We address and attempt to clarify each of these disagreements, not only to

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arrive at a firmer understanding of the derivedness of human PFLS, but also to build a more holistic and precise understanding of the trait in general. Indeed, many of the competing claims we address have been previously identified (many by Williams[9]), but methodological shortcomings and miscommunications between and within fields have forestalled consensus.

In the following review we examine five disputed claims regarding human PFLS. We start with **Claim 1: Reproductive senescence is menopause**, as this conflation is in part responsible for the anthropological assumption that human PFLS is derived. The difficulty of interpreting the evidence against **Claim 2: PFLS should not exist unless it is selected for**, has lead to **Claim 3: Human PFLS is quantitatively but not qualitatively different,** which is true for one definition of PFLS but not for the one of primary interest in anthropology. We demonstrate this qualitative difference in the context of **Claim 4: Human PFLS is a function of protected environments,** and finish by showing that **Claim 5: Women stop reproducing early then live too long for primates of their size** requires re-examination with improved methods. While we ultimately conclude that human PFLS is derived, reaching this conclusion requires dispelling the usual arguments made in its support. We finish by describing how the particular way in which human PFLS is derived informs our understanding of how it arose and is maintained by evolution.

CLAIM 1: REPRODUCTIVE SENESCENCE IS MENOPAUSE

In studying the evolution of PFLS, some authors focus on survival after reproductive senescence,[5,10,11] others on menopause,[12,13] while still others seem not to distinguish between the two.[14] The two traits are undeniably related, but treating them as interchangeable causes confusion. Reproductive senescence is the decline over age in fertility (the production of live neonates), which may culminate in the total cessation of fertility, which in humans generally occurs some years before menopause,[15] the physiological cessation of menstrual cycling found across the population in individuals of similarly advanced age. Survival after the cessation of fertility, even where that survival predates menopause, is post-fertile lifespan. The conflation of these terms leads to demonstrably false statements such as "reproductive senescence [is] a life-history trait virtually unique to human females."[14]

In rejecting Claim 1, we should state that, like many others,[5,10,16,17] we focus on reproductive senescence, leaving menopause as a related but separate question. The premortem decline in fertility is more relevant to our question than is menopause because the evolutionary theory of senescence predicts mortality to evolve in relation to the fertility schedule, not the pattern of cycling. Hawkes and colleagues[10,16] emphasize the need to study fertility decline directly for similar reasons. Studying fertility decline avoids the tangentially related debates about exact definitions of menopause[18] and about whether captive primates, which clearly experience reproductive senescence, experience a true menopause.

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For our purposes, the key point in reproductive senescence is the age at which an individual becomes post-fertile. We define "post-fertile" to mean after the approximate end of the population's fertile ages. The end of the fertile period is specifically defined as age M, which is measured from a life table as the age at which 95% of cohort fertility is passed.[19] For natural fertility human populations, M approximates mean age at last birth. We will generally avoid the term "post-reproductive" in order to avoid the semantic disagreements associated with the term (for example, can one be post-reproductive if still raising offspring?),[20] but take past uses of "post-reproductive" to broadly mean post-fertile. See Box 1 for definitions of demographic terms and parameters.

CLAIM 2: POST-FERTILE LIFESPAN SHOULD NOT EXIST UNLESS IT IS SELECTED FOR

Many papers exploring the evolution of PFLS are motivated by the fact that "classical evolutionary theory suggests that natural selection should lead to an individual's reproductive capacity ending in unison with the capacity for somatic maintenance."[21] With increasing age, expected residual reproduction approaches zero and, as a result, the evolved mortality rate is predicted by the standard interpretation of classical theory to approach infinity.[22] This prediction is referred to as the "Wall of Death," (so named by Charlesworth and Partridge[23]) as no individual should surpass the barrier of reproductive cessation.

Because the evolutionary theory from which the Wall of Death prediction arises is so influential in current evolutionary demography and is the most commonly cited theoretical motivator for studies of post-fertile lifespan, we must examine why the Wall of Death has not been widely observed in real populations. Here, we first review the empirical evidence that contradicts the Wall of Death prediction, focusing on iteroparous species. Then we show that the Wall of Death prediction rests on what we call the Inversion Assumption. We briefly demonstrate how the Inversion Assumption relates to the Wall of Death and, in Box 2, present some of the reasons why it is false.

Box 1. Demographic Concepts and Life-Table Measures, B, M, Z, and PrR

We employ two different concepts of post-fertile lifespan (PFLS), post-fertile viability, and a post-fertile life-stage. These distinct phenomena require different measurements. Post-fertile Viability can be measured by asking the question, "how long can an individual or cohort live after fertility ceases?" As such, Post-fertile Viability is measured in units of time (for example, years), capturing the span of time between the end of fertility and death. For our examples, using population level data, we use age M as the end of fertility and age Z as the end of life, where M is the near endpoint of cohort fertility and Z is the near endpoint of cohort survival.

Specifically, age M, is calculated from the age specific fertility, m_x, of a population. M is the first time period of age at which 95% of lifetime fertility has been realized. That is:

$$\sum_{x=0}^{M} m_x \geq 0.95 \sum_{x=0}^{\infty} m_x$$

for M an integer.

Age Z is similar to age M, but rather than being calculated from cohort fertility, we calculate it from cohort survival, l_x.

$$\sum_{x=0}^{Z} l_x \geq 0.95 \sum_{x=0}^{\infty} l_x$$

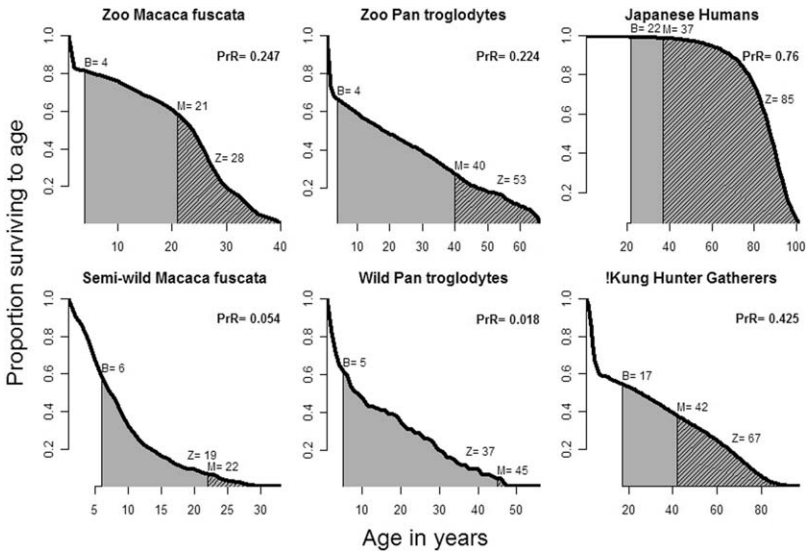
for Z an integer.

Z is the age at which 95% of cohort years lived have passed, and as such is a near endpoint of cohort survival. We use Z rather than the more common Maximum observed longevity (MaxO) for several reasons. MaxO is sensitive to sample size; it increases with the number of individuals observed for a population or species, particularly with small samples. This is potentially a serious limitation for comparing humans to other primates or mammals because the differences in sample size are very large (a few hundred observed lifespans versus several billion). MaxO is also sensitive to influence from a single false observation, one biologically extraordinary individual, or a heavy tailed distribution of ages at death. Finally, a species with greater variance in longevity (or observed in a greater variety of environments) will tend to have greater MaxO than an otherwise similar species. Z avoids these difficulties. Greater details on these parameters, and their calculated values can be found in Levitis and Bingaman Lackey.[19]

The difference, M subtracted from Z, is a population measure of Post-fertile Viability. Any population for which Z-M is positive has Post-fertile Viability.

Testing for Post-fertile Stage requires the use of a parameter which Levitis and Bingaman Lackey[19] labeled Postreproductive Representation (PrR). PrR is also calculated from a lifetable, as that proportion of adult-years lived which are post-fertile (meaning that they are after age M). In order to determine when adulthood begins, we say that age B is the beginning of adulthood. Age B is analogous to age M, but comes after 5%, rather than 95% of fertility is past. Thus B and M measure the approximate beginning and end of the reproductive lifespan, respectively. PrR is equal to the cohort-years lived after age M divided by the cohort-years lived after age B, or in demographic notation: $PrR=T_M/T_B$.

This notation is useful, because it allows us to employ the quantitative machinery demographers have developed for manipulating lifetable calculations. For example, $T_x=l_x*e_x$. That is, cohort-years lived after a particular age (T_x) are equal to the number of individuals alive at that age (l_x) times the mean life-



Box Figure 1. Postreproductive Representation as the ratio of areas. PrR is the ratio of post-fertile adult years lived (after age M, hatched area) to total adult years lived (after age B, shaded area). B and M estimate the beginning and end of the fertile period, excluding unusually early and late births. We plot survival over age from three primate species, each in a relatively safe and a relatively dangerous environment. Note that in wild chimpanzees and semi-wild macaques, Z comes before M and post-fertile survival, while fairly lengthy (from M to the end of each graph) has minimal area because few individuals are alive at post-fertile ages. Standard measures of post-fertile survival consider the length of the post-fertile period, but not its height (how many people are alive), considering only one dimension of the hatched area. The height of this hatched area is the primary driver of the difference in PrR between humans and non-humans.

expectancy of those remaining individuals at that age (e_x). This simple calculation makes clear that $PrR=T_M/T_B$ can also be written as $PrR=l_M/l_B * e_M/e_B$. This tells us that PrR is determined by the proportion of adults who survive to become post-fertile times the proportion of adult life-expectancy which remains when they become post-fertile. This demonstrates that the proportion surviving to age M is as important as the life-expectancy after age M in determining PrR.

Once PrR is calculated, its statistical significance may be tested by reference to a null distribution of simulated PrR values. This processes, described in detail by Levitis and Bingaman Lackey,[19] proceeds as follows. First, we pose the null hypothesis that age-specific survival (lx) and age-specific fecundity (mx) decline simultaneously and proportionately. Second, we stochastically simulate individuals to form a series of populations which are of the same size as the observed population. The probabilities of dying and reproducing for each individual at each age are determined by the observed life-table demography altered to match our null hypothesis. Specifically, the lx vector is altered to decline simultaneously and proportionately with the mx vector. Each individual is simulated independently, explicitly including demographic stochasticity in our estimates of what PrR would look like if the null hypothesis was true. Because of this stochasticity, PrR is always greater than zero (and by definition cannot be more than 1). We simulate 10,000 populations under this null hypothesis, and calculate PrR for each. PrR is significant if the observed value falls above the 97.5% quantile of the null distribution.

Because a protected environment can lead to significant Post-fertile Viability, we stipulate that a species should have significant PrR across the environments it occupies to conclude that it has a Post-fertile Stage.

Box Figure 1 displays B, M, Z, and PrR for six primate populations in three species, illustrating the importance of both species and environment.

Box 2. Why the Inversion Assumption Fails

All demographic traits are potentially subject to the action of natural selection, and this very clearly applies to age-specific survival. Nevertheless, the mortality pattern need not mirror the force of selection against mortality, because the interactions of selection with life-history tradeoffs and mechanistic constraints, rather than selection alone, determine evolutionary outcomes.

Hamilton[22] notes that the relationship between selection and mortality will depend on available variation. Charlesworth[32] points out, and Rose and colleagues[33] expand upon the idea, that if only some, but not all, genes have age-specific effects on survival, selection for increased survival early in life can maintain selectively neutral non-zero survival in late life. Cohen[5] argues that mortality risks at different ages strongly covary, such that selection shapes mortality trajectories broadly, rather than micromanaging mortality at each age. Patterns of gene expression are correlated with age but distinguishing between young and old individuals based on gene expression is difficult, even when comparing groups as different as 25-year-old and 75-year-old humans.[34] An excellent example of this is the breast-cancer gene, BRCA1, which increases mortality primarily in post-fertile women but also puts reproductive age women at risk because of variation in age of onset.[35] Indeed, there is little biological support for the widespread existence of genes whose effects are sufficiently exact in their age of action to allow the age-specific selection curve to produce a mirror image of itself in the pattern of mortality over age.[23]

Moorad and Promislow[36] review evidence from mutation-accumulation experiments and find that many de novo mutations influence survival in early life, but very few influence late-life mortality. This means that while selection is ineffective in removing mutations that influence only post-fertile survival, such mutations tend not to arise in the first place, limiting their accumulation.

Baudisch[37] and Vaupel and coworkers[38] have offered a more fundamental challenge to the logic underlying inversion. They stress the importance of life-history tradeoffs and the constraints they impose on the action of selection, finding that evolved mortality is dependent upon covariances between traits, perhaps more so than it is dependent on the declining force of selection.

These and several other examples make clear that we must be extremely careful in relating the age-specific force of selection and the age-specific mortality rate to each other. Mortality tends to increase over age because the force of selection against mortality declines but because tradeoffs and genetic covariances also determine the influence of selection on mortality, the shape of the mortality profile is not necessarily the mirror image of the force of selection.

Lastly, we explain how this allows for selectively neutral post-fertile survival.

The most damning empirical evidence against the Wall of Death is how widely post-fertile survival is observed. Williams[9] notes the broad occurrence of post-fertile survival in domesticated animals. Cohen[5] finds evidence of post-fertile individuals in most of the 42 mammal species he examines. However, the presence of post-fertile individuals is not limited to mammals, or species in which kin selection arguments would seem to apply. Common lab organisms that produce extremely post-fertile individuals without genetic or chemical manipulation include common yeast, *Saccharomyces cerevisiae*[24]; nematodes, *Caenorhabditis elegans*[25]; fruit flies, *Drosophila melanogaster*[26]; guppies, *Poecilia reticulata*[8]; Bdelloid rotifers, *Macrotrachela quadricornifera*[27]; and seed beetles, *Callosobruchus maculatus*.[28] Even some semelparous Pacific salmon (genus *Oncorhynchus*), the standard example for dying shortly after reproduction, will in some conditions live long after their single reproductive event.[29] Under Hamilton's assumptions, all of these post-fertile organisms in taxa where there is no apparent selective benefit to post-fertile survival should be stopped dead by the age-specific gene effects that compose the Wall of Death. Why is the Wall of Death not observed, and why was it expected?

The reasoning behind the Wall of Death prediction is fairly simple. Natural selection should not tend to increase age specific survival at ages when individuals are no longer able to contribute to their own lifetime reproductive success. Deleterious gene effects should therefore not be selected against if they are harmful only to post-fertile individuals. Such alleles will arise from time to time through mutation, and in the absence of opposing selection, alleles increasing post-fertile mortality should inexorably accumulate in lineages. This logic was formalized by Hamilton,[22] who calculated a rapidly declining force of selection against mortality over age, reaching zero at the age of reproductive cessation.

The evolutionary literature points to two routes for achieving post-fertile survival. The first route modifies Hamilton's calculations of the force of selection over age to include factors other than female fertility (kin care, resource transfers, male fertility) that also influence selection. The second route identifies mechanisms (e.g., life-history tradeoffs, population genetics, gene-expression patterns) that constrain the set of genotypic and phenotypic outcomes selective pressures can produce.

A common assumption amongst researchers specifically addressing selective forces is most clearly stated by Lee[67] in his influential paper on the evolution of the human mortality pattern: "Mortality [at each age] should be inversely proportional to force of selection [against mortality at that age]." This can be called the Inversion Assumption, and it is often used to estimate the unobserved total force of selection as the inverse of observed mortality. Hamilton did not state this assumption explicitly, but much of his reasoning (see Section 9 of that paper) strongly implies it. For example, if a population experiences high mortality at the beginning of life, both Hamilton and Lee suggest that the force of selection against mortality at the beginning of life in that population is weak (mechanistic counters to this argument are reviewed in Levitis 2011).

While the Inversion Assumption is rarely stated directly, it is implied in much of the literature on the evolution of post-fertile survival. For instance, Reznick and coworkers[8] state that "evolutionary theory predicts that an extended post-reproductive lifespan should evolve only when post-reproductive females can contribute significantly to the fitness of their offspring or relatives." More exactly, evolutionary theory predicts that selection for post-fertile survival should only occur when post-fertile individuals can continue to improve their fitness outcomes. The two statements are not equivalent, because one describes when PFLS can evolve, and the other describes when it is selectively advantageous. If we make the Inversion Assumption, then only selectively advantageous PFLS can evolve. Without this assumption, factors other than selectively beneficial PFLS can lead to the evolution of extended post-fertile viability, as Reznick and coworkers'[8] guppies demonstrate.

Indeed, there are numerous mechanistic reasons why a population's mortality pattern may not look like the inverse of the force of selection against mortality (summarized in Box 2). In short, natural selection can only select from among the sets of traits present in the population, and those arising by chance, and cannot manipulate each trait independently. Indeed, the widespread occurrence of Post-fertile Viability is a clear case where failure to consider biological mechanisms can lead to false expectations regarding how demographic traits should evolve.[20]

The Inversion Assumption, while false, has been important in providing a theoretical motivation for studying the evolution of post-fertile survival. Further, when the mortality pattern is not at least roughly inversely proportional to a proposed pattern of selective pressures, inversion is useful as a null expectation[30] and may suggest that some selective mechanisms have not been considered. The Wall of Death prediction fails spectacularly for iteroparous species, is not useful for understanding semelparous species[20,29,31] and therefore should not guide research on PFLS or aging patterns in general.

Once the shortcomings of the Wall of Death and the Inversion Assumption are realized, it is no longer surprising to find post-fertile individuals in populations without the need for specific selective forces. The fact that we do find such individuals in so many species leads to Claim 3.

CLAIM 3: HUMAN PFLS IS QUANTITATIVELY BUT NOT QUALITATIVELY DIFFERENT

How can we judge if human PFLS is quantitatively different? A simple approach would be to ask if humans have PFLS longer than other primates. In addition to the non-primates described above, the capacity for PFLS is widespread in primates. For the 66 primate species most commonly kept in zoos,[39] we calculated the maximum observed female

longevity and maximum observed age at last parturition. We found that each of these primate species has produced individuals that lived 25% to 95% longer than last observed parturition (mean=51% longer, SD=18%), a result so extreme and consistent that it likely cannot be accounted for by measurement error or demographic stochasticity. Last observed parturition in these captive populations closely estimates the same parameter in sympatric wild populations, implying that these individuals truly are surviving to meaningfully post-fertile ages, and other primates, like humans, have long PFLS.

However, such evidence in favor of widespread PFLS can be interpreted as showing that human PFLS is simply the same phylogenetically conserved trait, but scaled-up to the relatively large human body mass. This is true given the common definition of PFLS that we describe above, as the length of time between reproductive cessation and death. When PFLS is measured as this span of time then one can conclude that there is nothing qualitatively (or quantitatively) special about post-fertile survival in human females. However, this measure of PFLS demonstrates only a non-zero life expectancy of populations at reproductive cessation, but does not measure the proportion of the population that reaches reproductive cessation. Therefore, this "span of time" approach to measuring PFLS does not describe what portion of the lifespan individuals generally spend in a post-fertile state. Survival to reproductive cessation is at least as important as the life expectancy thereafter in determining the ecological, social and fitness effects of PFLS. Because measures incorporating both types of information have only recently been introduced, PFLS has almost always been measured in terms of life expectancies at cessation (but see Hawkes[1,40] and Levitis and Bingaman Lackey[19]). However, questions regarding post-fertile survival as an additional stage in the adult lifespan of a species require that PFLS be measured in a different way. We are interested in the overall volume of post-fertile life, but have instead been measuring only its length. This is analogous to comparing the meat consumption of two populations by only asking how much meat they eat when they eat it, but ignoring the question of how often it is consumed. Semi-wild *Macaca fuscata* are an excellent example of the length of PFLS being uninformative as to the importance of PFLS.[41] Their life expectancy of 3.1 years at age M=22 years is half as long as the pre-fertile period, and as such seems like an important life stage, until one considers that only 0.06 of individuals born survive to age M, such that even in this protected population fewer than 5% of adult females alive at any time are likely to be post-fertile.

Postreproductive Representation (PrR) measures the proportion of adult years lived which are post-fertile[19] and is more appropriate for measuring survival beyond fertility as an evolved life history characteristic. The span of time approach to measuring PFLS is useful for showing how widespread the ability to outlive fertility is, shows that the Wall of Death prediction is wrong, and shows that somatic and reproductive senescence are often decoupled, but does not help for questions about a derived life stage. In a population at equilibrium, PrR will equal that proportion of adult females who are beyond age M (roughly the age at which most females have their last parturition). PrR is also the mean proportion of the adult lifespan each female can expect to be post-fertile. PrR was developed as a tool for evaluating the significance of PFLS and comparing PFLS between demographically dissimilar populations, and is similar to a measure employed by Hawkes to compare humans and chimpanzees.[1] See Levitis and Bingaman Lackey[19] and Box 1 for details on how PrR is calculated and tested for statistical significance. In short, the significance of an observed value of PrR is tested by comparing it to a null distribution generated through stochastic simulations based on the null hypothesis that actuarial and reproductive senescence occur simultaneously and proportionately. If the observed value falls in the right tail of the simulated distribution, more post-fertile survival is observed than would be expected based on demographic stochasticity alone. Because survival to reproductive cessation is a primary determinant of PrR, populations with identical ages at reproductive cessation and identical life expectancies thereafter can have drastically different PrR.[19]

Survival to reproductive cessation is at least as important as the life expectancy thereafter in determining the ecological, social and fitness effects of PFLS.

PrR can be used to test for quantitative differences among populations, but as with any demographic measure the environments of the population must also be considered. Most of the data described under Claim 2 were gathered in environments designed to reduce extrinsic mortality, like zoos and laboratories. Zoo populations (provisioned, doctored, and well buffered from predation) differ from their wild counterparts, especially where mortality is concerned. A population that has many post-fertile individuals in a zoo clearly has the physiological capacity for a PFLS but need not have any sort of evolved post-fertile life stage. This distinction, between post-fertile viability and an evolved post-fertile life-history stage, first pointed out by Williams,[9] is key to understanding both the phylogenetic pattern of PFLS and how humans differ from other primates.,

These two distinct evolutionary phenomena have been conflated under the common label "post-reproductive lifespan," leading to much of the disagreement over how common PFLS is. Post-fertile Viability means

that individuals of a species have the capacity, given the right circumstances, to become post-fertile, regardless of what portion of the population does so. In principle, one could demonstrate Post-fertile Viability using data on only one long lived (and post-fertile) individual.[42]

Post-fertile Stage, on the other hand, is a significant stage in the evolved life-history of the species. How can we tell if a species has a significant post-fertile stage? Such a species should be expected not just to produce the occasional post-fertile individual, but to have a significant representation of post-fertile individuals across the range of environments the species inhabits. These simple criteria translate directly into a quantitative test for Post-fertile Stage using tests of significance of PrR. A finding of significant PrR for all populations of a species across a range of environments suggests that the PFLS is an evolved stage of the organism's life-history, rather than an artifact of a few exceptional individuals or a particular setting.

Although Post-fertile Viability and Post-fertile Stage are often treated as identical, they are sufficiently distinct to require consideration as separate traits. Post-fertile Viability, but not Post-fertile Stage, is expected in any species in which the environment can prolong survival without equally prolonging reproduction. The presence of either constitutes a violation of the Wall of Death prediction, such that gerontologists focusing on that hypothesis can safely treat them both as interchangeable counter evidence. However, the degree of population-wide post-fertile survival in a Post-fertile Stage species will greatly exceed that needed to show Post-fertile Viability in a species. As a result, the demographic experience implied by Post-fertile Stage is very different from that implied by Post-fertile Viability. Post-fertile Stage females live in a population with many post-fertile individuals, are likely to have post-fertile relatives, and a large fraction of those reaching adulthood can expect to be post-fertile themselves, even in high mortality environments. Most importantly, the evolutionary implications of Post-fertile Stage are quite distinct from those of Post-fertile Viability. PFLS can be thought of as having three very distinct character states: no PFLS (because no post-fertile individuals can be produced, as in the invertebrate *Hydra vulgaris,* which lacks reproductive senescence and therefore post-fertile individuals[43]), Post-fertile Viability (such individuals can be produced, as in yeast and non-human primates in zoos[24]), and Post-fertile Stage (significant PrR across environments).

We have arrived at a test for qualitative difference. If humans experience significant PrR in all environments, and therefore have Post-fertile Stage, while other primates do not, and therefore have Post-fertile Viability, human PFLS is qualitatively different. Under Claim 4, we will argue that this is so.

Even the worst surviving human population has Postreproductive Representation higher than the highest recorded value for nonhuman primates in protected environments. Further, and more importantly, humans experience significant PrR in high-mortality environments, while nonhuman primates do not.

CLAIM 4: HUMAN PFLS IS A FUNCTION OF PROTECTED ENVIRONMENTS

Lengthy human PFLS is often thought to be a byproduct of recent gains in longevity that have occurred only during the industrial age. If this were so, then certainly PFLS would not be an evolved stage of the human life history. Claim 4 is generally based on paleodemographic examinations of human skeletal collections that estimate very low survival to post-fertile ages.[44,45] However, detailed examinations of these methods and results have shown systematic bias in age representation such that many individuals in such populations may have indeed been post-fertile.[46,47] While there are those who still argue for the "benign environment" explanation of human PFLS based on paleodemographic data,[48] examination of modern hunter-gatherers has shown significant PFLS. For example, women of the Hadza population of hunter-gatherers[46] can expect to spend almost half (PrR=0.48) of their adult lives in a post-fertile state. Looking to an unnaturally harsh environment, we still find significant PrR. For the plantation slaves of Trinidad,[49] a remarkably mistreated historical population with unsustainably high mortality, PrR is still 0.315 ($p<0.001$). Mortality could not have been higher than that experienced by this slave population for sustained periods of time in the Paleolithic, or at any time, as this would lead to rapid extinction. Hence this is very strong evidence that essentially all human populations have significant PrR. Given the data available (Table 1) there is no demographic support for the claim that the human PFLS is a product of special environments. Women possess Post-fertile Stage, as they have highly significant PrR (beyond what one could expect if somatic and reproductive senescence were simultaneous and proportional) across the range of environments humans inhabit, from the technologically benign to the unnaturally abysmal (Fig. 1). Note that while all of the nonhuman primates depicted in Figure 1 also have significant PrR under benign conditions, two important considerations distinguish humans. Even the worst surviving human population has Postreproductive Representation higher than the highest recorded value for nonhuman primates in protected environments. Further, and more importantly, humans experience significant PrR in high-mortality

TABLE 1. Demographic Parameters for Selected Populations[a]

Species	Population	B (years)	M (years)	Z (years)	PrR	Source
Homo sapiens	Trinidad plantation slaves	19	45	60	0.315*	49
Homo sapiens	!Kung	17	42	67	0.425*	75
Homo sapiens	Ache	18	44	68	0.439*	76
Homo sapiens	Haiti 2002	19	43	72	0.460*	77
Homo sapiens	Sweden 1751	21	43	68	0.477*	78
Homo sapiens	Hadza	17	42	69	0.481*	46
Homo sapiens	Afghanistan 2002	18	43	71	0.486*	77
Homo sapiens	Papua New Guinea 2002	18	43	72	0.489*	77
Homo sapiens	Niger 2002	17	42	71	0.490*	77
Homo sapiens	Somalia 2002	17	42	72	0.497*	77
Homo sapiens	UN Less Developed Regions 2002	18	40	77	0.607*	77
Homo sapiens	UN Less Developed Countries 2002	19	39	78	0.643*	77
Homo sapiens	USA 2002	17	38	83	0.668*	77
Homo sapiens	Sweden 2002	21	39	83	0.707*	77
Homo sapiens	Japan 2002	22	37	85	0.760*	77
Cercopithecus mitis	Wild	6	25	23	0.041	57,79
Macaca fuscata	semi free ranging	6	22	19	0.054*	41,80
Macaca fuscata	Zoo	4	21	28	0.247*	39
Macaca mulatta	Wild	5	21	17	0.007	
Macaca mulatta	Zoo	4	24	30	0.178*	39
Pan troglodytes	synthetic wild	12	45	37	0.018	81,82
Pan troglodytes	Zoo	9	40	53	0.224*	39
Papio hamadryas	Wild	6	24	17	0.005	83
Papio hamadryas	Zoo	4	32	34	0.084*	39

[a]PrR marked with * indicates significance at the 0.05 level.
[b]No captive data are available for *Cercopithecus mitis.*
[c]Parameters B (cohort onset of fertility), M (cohort cessation of fertility), Z (cohort longevity) and PrR (proportion of adult years lived which are post-fertile years lived) are defined in greater detail in Box 1.

environments, while nonhuman primates do not. This is a qualitative difference between human PFLS and that of all other primates. We will argue that this difference is not predictable based on quantitative scaling patterns, but only after rejecting the reasons usually given for that conclusion.

CLAIM 5: WOMEN STOP REPRODUCING EARLY, THEN LIVE TOO LONG FOR PRIMATES THEIR SIZE

The uniqueness of human PFLS is often asserted based on the observation that women's time between reproductive cessation and death is longer than that of other female primates.[50] However, the counterargument has also been made that the longer human PFLS is predictable based on primate scaling patterns.[7] Longevity and age at reproductive cessation are both demographic traits that are expected to be greater in primates with higher body and brain mass; PFLS could seem longer in humans simply because we are large primates. Traits that vary as functions of mass are generally described as allometric relationships (see Box 3 for a synopsis of allometry). If human PFLS is simply that expected for a scaled-up primate, then it is not a derived trait. Likewise, if human PFLS is predictable allometrically, then focus would shift from socioecological variables in human evolution to cross-species relationships between body mass, brain mass, and demographic traits. The widely known allometric relationships between brain or body mass and demographic traits prompted Judge and Carey[7] to study the allometries of maximum observed longevity (MaxO) in primates and humans. They present seven regression analyses of primate MaxOs over either only body mass or body mass and encephalization as predictive variables including different primate clades in their sample. They then use these regressions to predict what MaxO should be for a primate of human mass. Their predicted values of human MaxO range from 47 years (based on body mass only and excluding prosimians) to 92 years (based on body mass and encephalization, excluding prosimians and New World monkeys). Their study draws attention to the point that brain mass and body mass are correlated and that brain mass is often the stronger predictor of demographic traits.[51–54] They conclude that the tendency for human females to outlive their reproduction was predictable based on the allometric primate pattern, but some authors have taken their study as evidence that human longevity is longer than predicted,[55] which is the opposite of their conclusion.

Because human age at reproductive cessation, longevity, and PFLS are all suspected to follow allometric constraints observed across primates, we calculated how strongly each is predicted by both body mass and

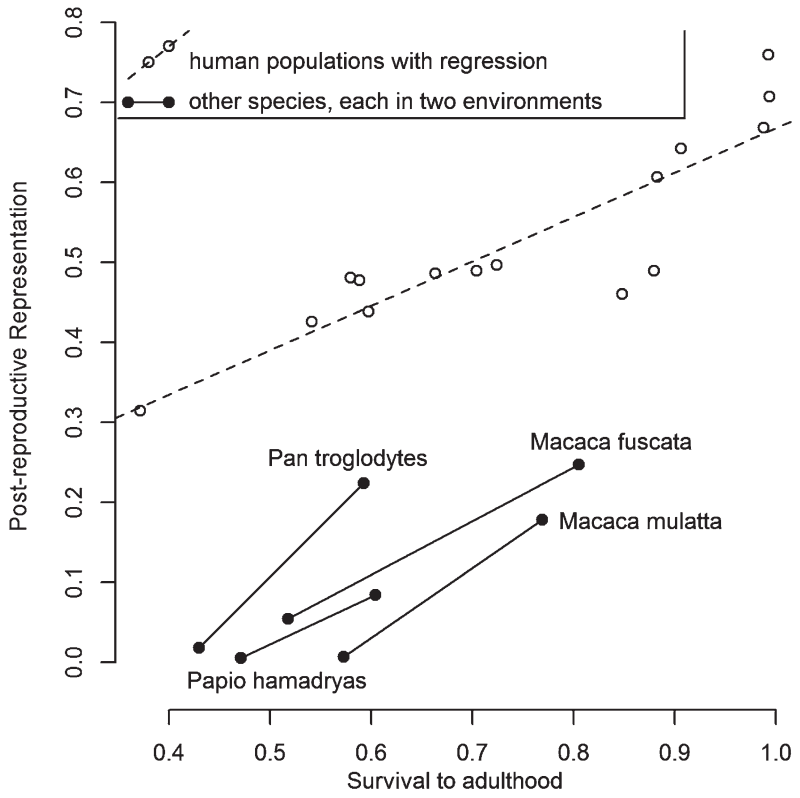


Figure 1. Postreproductive lifespan increases in lower mortality environments. Postreproductive representation (PrR), our quantitative measure of Post-fertile Stage, is plotted over proportion surviving to adulthood for 15 human (open circles) and eight non-human populations (closed circle). Each of four non-human species is represented by two populations, joined by a solid line. In each case, the upper right population is in zoos, while the lower left population is wild or semi-wild. Although pre-adult mortality does not influence the calculation of PrR, survival to adulthood is a strong predictor of PrR within human populations. Non-human primates experience similar increases in PrR with improving environment. The leftmost data point represents the plantation slaves of Trinidad, a very high mortality population which nonetheless has higher PrR (0.315) than any non-human primate population in our dataset. PrR is statistically significant in all human populations, all four captive primate populations shown, and (marginally, $p=0.041$) in the semifree ranging population of *Macaca fuscata* (PrR=0.055). Wild populations of *Macaca mulatta* (PrR=0.007), *Pan troglodytes* (0.018), and *Papio hamadryas* (PrR=0.005) do not have significant PrR (that is, not more than expected if somatic and reproductive senescence are simultaneous).

brain mass using recent improvements in phylogenetically controlled regression. Across all regressions, brain mass is the a stronger predictor variable than body mass (using Akaike information criterion, AIC,[56] See Box 3 and Table 2). Longevity is measured as age Z and the age of reproductive cessation is measured as age M (see Box 1).

M and Z are both well predicted by regression on brain mass in captive primates (Fig. 2). Z is predicted especially well, indicating that women are not unexpectedly long lived for their brain mass, corroborating Judge and Carey's[7] conclusion. Judging by women's M and Z, humans are indeed scaled-up primates, or at least scaled-up captive primates; women neither stop reproducing earlier[16] nor reach the near extinction of the cohort later than expected for primates with such large brains. Brain mass is the stronger predictor and we focus on the results of the brain mass allometries, but it is not completely clear if the reasons for this are biologically or evolutionarily relevant or purely statistical in nature (see Box 3). The predictability of Z and M implies that this difference between them is also predictable based on primate patterns and therefore best explained based on those patterns, rather than on-human socioecology. The difference Z-M is a measure of PFLS in time, roughly equivalent to life expectancy at reproductive cessation, the measure of PFLS most commonly used to argue that human PFLS is a derived trait. As such, we must reject Claim 5, and any argument claiming that this timespan between last birth and death (however measured) is unique to humans. Note that these results also suggest that human longevity is not greater than expected for a primate of our brain size, while keeping in mind that the result is based on captive primate populations.

The review and analysis presented so far points to the importance of the distinction between Post-fertile Stage and Post-fertile Viability. As discussed under Claim 4, humans are unique in having Post-fertile Stage, but there is nothing unique about human Post-fertile Viability, and this is what is captured by the allometries of Z and M.

It is likely the case that data on wild primates would make a different prediction regarding Z, our longevity measure. We have used data from protected environments where mortality is much lower and Z is much higher than in the wild. A comparable dataset on wild primate survival does not exist, but we can roughly estimate the relationships for wild primates using data recently published by Bronikowski and coworkers.[57] Recalculating lifetables for each of the seven species they report, we estimate a brain-Z allometry of $\ln(Z)=0.158*\ln(\text{brain mass})+2.6343$. From this equation and a human brain mass of 1,250g, we estimate human Z to be 43 years, lower than for even a human population in an unnaturally poor environment such as the plantation slaves of Trinidad ($Z=60$) and no higher than M for many human populations.[19] While this result is provisional, based on few species, and not phylogenetically controlled, it suggests that the analysis above is strongly influenced by the protected environment from which our data are gathered.

Box 3. Allometric Methods

Allometry is the biological study of how traits vary as a function of size. Size can be total body mass or sizes of other parts of the organism, such as the mass of the brain. Allometric relationships take the form $Y = a*X^Z$, where Y is the trait of interest (Y can be just about anything, but here it is M, Z, or PrR), X is size in units of mass or length (we consider brain mass and body mass separately), and a and z are coefficients fit to the data; a captures the "height" of the function and z captures the shape of the variation between Y and X, and is the slope of the regression line when the relationship is expressed logarithmically (that is, $\log(Y)=\log(a)+z\log(X)$). Nearly all life-history traits vary with body mass and longevity scales with mass across mammals to a power z of about 1/4.[58] This means that lifespan increases with body mass across species but it does so relatively slowly. Studies of allometry have revealed that life-history traits are often highly constrained and, from a certain vantage point, very different organisms can seem like rescaled versions of one another because, if one simply changes mass, many of its demographic and ecological traits change predictably according to these established allometric relationships. This explains the need to control for allometric effects when assessing if traits that scale allometrically with size might also be derived, and all things related to PFLS scale with size in both mammals and primates (and birds), hence one cannot answer the question: "is human PFLS a derived trait" without controlling for the effects of mass (and phylogeny).[59]

We considered both body mass and brain mass because brain mass has important scaling relationships in birds and mammals.[51,53,54] When we evaluated how M and Z vary as allometric functions of both body mass and brain mass, we found that in all cases regressing on brain mass was a better choice, on statistical grounds, for predicting M and Z than was body mass, as determined by Akaike information criteria (AIC). While our brain based allometries correctly predict human Z, our body mass based allometries do not, with observed human Z values falling outside the very wide prediction intervals. These prediction intervals were calculated in two ways, analytically and using simulation (which generate narrower intervals). These intervals give the range of values for a human-sized primate determined to be consistent with the primate allometry (Box Table 1).

We see two possible explanations for the finding that brain mass was a stronger predictor than body mass. The first is statistical in that brain mass may be both measured with less error than body mass and also may be less variable across individuals.[60] The second possibility is that investments in brain growth actually capture the key features of the slow primate life history better than body mass alone does. Primates have larger brains for their body mass, compared to other mammals, along with longer lifespans, later ages of maturity, and longer intervals between births.[59] If brain mass and a slow life-history go together in primates and brain mass captures the underlying tradeoffs better than body mass does, then there are also theoretical grounds to consider brain mass as the main predictor variable. However, the statistical explanation is, at this point, just as valid and both should be considered.

Lastly, we also adjust for the phylogenetic relationships of the species in our dataset because species may have similar trait values simply because of common descent, but the influence of phylogenetic relatedness can be accounted for.[61,62] Numerous methods have been developed for this and these techniques have improved greatly in recent years. We used the procedures recently synthesized in Revell.[63] In short, this involves using a maximum likelihood procedure that simultaneously estimates both the regression coefficients and Pagel's lambda.[63] Pagel's lambda[64] adjusts for the degree of covariation between the model residuals and the amount of shared ancestry. In this way, a phylogenetically controlled allometry can consider how two traits scale in relation to each other without that scaling pattern being distorted by patterns of common ancestry. Revell's method is more robust than the widely used method of independent contrasts, which can generate problematic coefficient estimates because they do not account for how much the phylogeny influences the residuals (Pagel's lambda accounts for this).

BOX TABLE 1. Values of M (Cohort Cessation of Fertility) and Z (Cohort Longevity) Predicted by Allometry for a Primate of Human Size[a]

		Analytically determined			Simulated		
Dependent	Independent	Lower	Upper	Range	Lower	Upper	Range
M	Body mass	3.03	3.86	0.84	3.18	3.88	0.70
		20.61	47.60	26.99	24.16	48.56	24.40
M	Brain mass	3.33	4.27	0.93	3.53	4.10	0.58
		28.07	71.20	43.13	33.99	60.41	26.42
Z	Body mass	3.23	4.07	0.84	3.22	4.11	0.90
		25.36	58.59	33.22	24.90	61.00	36.10
Z	Brain mass	3.64	4.57	0.93	3.79	4.40	0.61
		38.16	96.79	58.63	44.24	81.09	36.86

[a]For each regression, the upper row contains the logged values and the lower row is in years of age.

TABLE 2. Evaluation of Brain Versus Body Mass as an Independent Variable for Allometry Using AIC[a-c]

Dependent	Independent	Model type	Intercept	Slope	AIC	Lambda
M	Body mass	GLS	1.505	0.189	−16.183	0.561
M	Brain mass	GLS	2.172	0.238	−24.077	0.458
M	Body mass	OLS	1.465	0.196	−22.437	NA
M	Brain mass	OLS	2.212	0.221	−32.154	NA
Z	Body mass	GLS	1.616	0.198	−7.834	0.769
Z	Brain mass	GLS	2.228	0.273	−20.704	0.668
Z	Body mass	OLS	1.429	0.217	−9.739	NA
Z	Brain mass	OLS	2.278	0.240	−19.944	NA

[a]M is cohort cessation of fertility and Z is cohort longevity.
[b]GLS models are phylogenetically controlled generalized least squares adjusted with Pagel's lambda.
[c]OLS models are uncorrected ordinary least squares, for comparison.

The above allometries measure PFLS in units of time (Z-M), and indicate that the endpoints of human Post-fertile Viability are predictable from primate brain allometries. Because they do not measure PFLS in terms of PrR, they do not tell us anything about whether the human PFLS is an evolved stage, or whether human values of PrR are allometrically predictable. Again, PrR is necessary for addressing questions regarding Post-fertile Stage, and depends upon the proportion of a population surviving to M, not just the timespan between M and Z. However, the human values of PrR predicted by primate patterns can also be estimated from the zoo data used above. Allometric regression of PrR on brain mass yields a not quite significant slope (GLM, PrR=0.935+0.0176*ln(brain mass), lambda=0.31, df=45, p=0.08) and a human PrR of 0.219. This value is much lower than any human population in our dataset and is even lower than the observed value for chimpanzees (PrR=0.224), a species that lacks significant PrR in the wild (0.018). That PrR in one of the poorest surviving human populations known, Trinidad Slaves, is greater than the PrR predicted for large-brained primates in protected environments where mortality is greatly reduced, is extremely compelling evidence that something is qualitatively different about post-fertile life in *Homo sapiens*. As such, we reject Claim 5 while maintaining the finding that Post-fertile Stage is a derived trait.

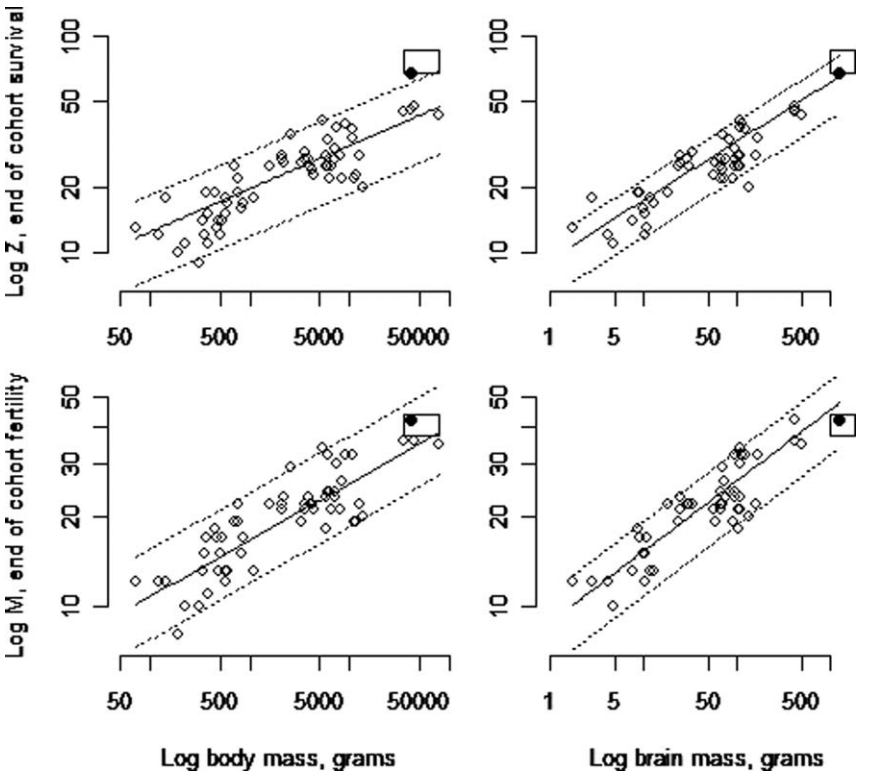


Figure 2. Allometric predictions of human demography from primate patterns. The results of allometric regressions of M and Z on body and brain mass. The open circles are non-human primates in captivity, and the black circle is the raw data for the !Kung. The box represents a rough estimate for the full range of human variation from hunter-gatherers to wealthy industrial nations. The dashed lines are the prediction intervals around the regression. Regression coefficients are in Table 1.

DISCUSSION: A POST-FERTILE STAGE AS A DERIVED TRAIT OF HUMANS

A precise understanding of PFLS has been hampered by a lack of consensus on what is being measured and how to measure it. Across many taxa, individuals outlive their reproductive lives, and there is no sound theoretical reason to expect otherwise; thus Post-fertile Viability clearly is not a derived trait of humans. Claims of phylogenetically widespread PFLS are supportable only where PFLS is defined as a non-zero life expectancy of post-fertile individuals, rather than as a distinct stage of the adult lifespan requiring particular evolutionary and ecological explanation. In short, our results demonstrate that Post-fertile Stage is a derived trait of humans even though Post-fertile Viability is not. Some set of selective forces acted to expand the post-fertile representation of human females by an order of magnitude compared to wild primates (PrR ranging from 0.3 to 0.8 vs. 0.01 to 0.05), bringing about Post-fertile Stage. Paleolithic humans

must have survived on average better than the plantation slaves of Trinidad, who experienced rapid population shrinkage due to high mortality. At least one third of women would have been post-fertile through out the bulk of human prehistory, a state of affairs unprecedented in primate evolution.[3,65]

As a thought experiment, consider a hypothetical organism in which somatic and reproductive senescence are coupled. Post-fertile viability does not exist and hence a post-fertile stage cannot evolve, even if post-fertile females would otherwise have positive effects on fitness. Even a more favorable environment could not produce post-fertile survival in such a species because anything that extended longevity would also extend reproduction. In other words, Post-fertile Stage cannot evolve unless individuals already have the ability to outlive their own reproductive period, requiring that the physiological basis of reproductive senescence is sufficiently decoupled from somatic senescence that environment and selection can manipulate each of the two forms of senescence separately.[66] Post-fertile Viability is a necessary preadaptation for a Post-fertile Stage. That portion of the literature on PFLS that focuses on menopause has obscured this by giving the impression that the physiological decoupling of reproductive and somatic senescence was a uniquely human trait. Guppies[8] produce new oocytes throughout life and stop reproducing at quite variable ages prior to death, and yeast[24] (single cellular fungi reproducing through budding) do not possess oocytes but eventually lose the ability to bud. These species clearly lack menopause, and yet clearly have a decoupling of reproductive and somatic senescence and therefore possess Post-fertile Viability.

This helps resolve the chicken-and-egg question of which came first, surviving grandmothers or the usefulness thereof, by making clearer how significant PrR (and therefore a stock of post-fertile matriarchs) could have nonadaptively arisen in humans. With a drop in mortality (due to intrinsic factors, extrinsic factors, or simple stochasticity) a population of early hominins, already possessing Post-fertile Viability, could, within a single generation, suddenly have a large group of post-fertile individuals. Such a single generation change can be observed when taking wild chimpanzees into captivity. Lacking any reproduction of their own, these matriarchs would likely invest in fitness-relevant activities, the most direct of which would be some form of allocare. If post-fertile individuals served to decrease mortality risk to the kin group (for example, by decreasing the dependency ratio),[67] this would tend to make the low mortality risk and higher PrR sustain and reinforce each other, first demographically, then evolutionarily. This scenario is supported by observation of Japanese macaques,[41] in which post-fertile individuals can contribute to the reproduction and survival of their descendents but are too rare to have a significantly impact on the population.

A comparative perspective is necessary for understanding how Post-fertile Stage could arise, but is also useful in understanding the selective forces at work. While the human step from Post-fertile Viability to Post-fertile Stage is unique among primates, it is *not unique* to humans. Some matrilineal group-living whales[11,68–70] have significant PrR in the wild. Generalizing from humans, it was first thought that whale grandmothers may aid in the survival of the their grandchildren, but no such effect has been detected.[70,71] Instead, Foster and colleagues[72] argue that post-fertile orca (*Orcinus orca)* females aid in the survival of adult sons. It is not yet clear what form this aid may take, or why their analysis indicates a stronger dependence on mothers of older rather than younger sons, but it does suggest that the benefits of Post-fertile Stage to whales may be distinct from its benefits to humans in important and informative ways.

Remarkably, the best evidence to date for adaptive PFLS[73] comes from gall aphids, *Quadrartus yoshinomiyai* (Nipponaphidini). Midway through their adulthoods, wingless females cease reproducing, freeing abdominal space for the production of defensive chemicals, with which they incapacitate predators threatening the largely clonal colony, and killing themselves in the process. The high representation of these living post-fertile glue bombs in the wild suggests they have Post-fertile Stage. Taken together, the available data on humans, whales, and aphids suggest convergent evolution of Post-fertile Stage in cases where older females can contribute to the fitness of their younger kin. This begs the question: Given that females of many species have post-fertile viability, and have contact with younger kin, what distinguishes those where post-fertile females provide a novel selective advantage from those where they do not? An answer to this question that could distinguish not only humans from other primates, but also orcas and short-finned pilot whales *Globicephala macrorhynchus* (in which Post-fertile Stage is observed) from long-finned pilot whales *Globicephala melaena* (which have only Post-fertile Viability)[11] would go a long way towards resolving the debate regarding human PFLS. Foote[11] argues that while factors such as juvenile dependency, kin structure, and resource sharing patterns, often used to explain derived human Post-fertile Stage, are important, they cannot differentiate the toothed whale species with Post-fertile Stage from those with only Post-fertile Viability. Comparative analysis, with detailed socioecological data on many species, will be needed to begin to draw conclusions. Knowing that humans, Japanese gall aphids, orcas, and short-finned pilot whales have Post-fertile Stage, while other primates, long-finned pilot whales, and African elephants only have Post-fertile Viability[19,74] represent only the beginning of a comparative dataset.

CONCLUSIONS

Many fields are strongly interested in the evolution of demographic traits, and the evolution of post-fertile survival is among the questions that have drawn the most attention across these fields. Despite many of the

necessary insights regarding the evolution of PFLS being available, methodological shortcomings and the sheer volume of the related literature have allowed false assumptions regarding PFLS to persist. These include the specific phenomenon it refers to, how it should be measured, among which taxa it should be observed, and how to identify differences in it between humans and other primates. Many anthropologists study PFLS (meaning a post-fertile life stage, but usually measured as post-fertile viability) as a human specific trait, while comparative biologists study PFLS (meaning and measured as post-fertile viability but then applied to post-fertile stage) as a ubiquitous trait found across species but quantitatively larger in humans. Neither group has entirely discarded the Wall of Death prediction, which, if correct, would necessitate selective explanations for Post-fertile Viability. The failure to distinguish Post-fertile Viability from a Post-fertile Stage leads to the misimpression that human PFLS is not a qualitatively different trait. At the same time, the intuitive sense that humans really are very different from other primates with respect to post-fertile survival, along with insufficient quantitative methods, lead to the misimpression that our ages at reproductive cessation and death are not to be expected of a primate of a woman's size. The fact that these ages actually are well predicted by captive primate brain allometry, and the failure to distinguish the maximum length of PFLS from the quantitative representation of post-fertile individuals in the population leads to the claim that the human post-fertile life stage can be expected from primate scaling patterns. All of these misconceptions can be addressed in a framework that distinguishes Post-fertile Viability from Post-fertile Stage, measuring the first in units of time, and the second in PrR. Such a framework reveals that rather than the length of the women's PFLS, it is its height (the number of women surviving to become post-fertile, see Box Figure 1), its area (the proportion of adult-years lived which are post-fertile) and its prevalence (across environments) that are extraordinary. Finally, an understanding of human demographic evolution requires insights from the range of fields interested in the evolution of demographic traits.

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