ARTICLES

Menopause: Adaptation or Epiphenomenon?

JOCELYN SCOTT PECCEI

Menopause is a nonfacultative and irreversible cessation of fertility that occurs in all female conspecifics well before the senescence of other somatic systems and the end of the average adult life span (Fig. 1).[1–3] So defined, menopause occurs only in humans and one species of toothed whales.[1–4] According to evolutionary theories of senescence, there should be no selection for postreproductive individuals.[5] Thus, evolutionary biologists and anthropologists have long been interested in why human females have menopause. Many have suggested that menopause is a hominine adaptation, the result of selection for a postreproductive life span that permitted increased maternal investment in existing offspring.[3,6–9] Others are persuaded that premature reproductive senescence is an epiphenomenon, either the result of a physiological trade-off favoring efficient reproduction early in the fertile part of life or simply the by-product of increases in life span or life expectancies.[10–17] Menopause poses two separate questions: why it originated and what is maintaining it?

Evolutionary biologists consider all complex design features of organisms to be ultimately the result of natural selection. As such, menopause can always be considered an adaptation. At the same time, it is also recognized that an adaptation is always morphologically, physiologically, and developmentally constrained by an organism's phylogenetic heritage.[18] The question of origin is whether menopause is primarily an adaptation, in the sense that selection directly favored a postreproductive life span in human females, or whether it is an epiphenomenon of selection for efficient early reproduction or physiological constraints preventing prolongation of fertility in the presence of increases in human longevity. The distinction between adaptations, fitness trade-offs, and true phylogenetic constraints is the level of explanation. Appeals to the role of constraints without explanation of why these constraints themselves are not subject to evolutionary change constitute proximate explanations. Although it is useful for heuristic purposes to think in terms of three distinct alternatives—adaptation, trade-off, or constraint—the explanations overlap and interact. For instance, a trade-off is part adaptation and part constraint. In addition, the question of menopause cannot be isolated from the question of human longevity. In all three cases, the bottom line is that selection favors early reproduction relative to life span. In this sense, reproductive senescence is always premature. In this paper I critically review the evidence for the adaptation, physiological trade-off and by-product of increased longevity explanations for the origin of menopause.

Jocelyn Scott Peccei is a Research Associate at the Department of Anthropology, University of California Los Angeles, Los Angeles, CA 90095-1553, USA. E-mail: jpeccei@ucla.edu

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THE ADAPTATION EXPLANATION

In the strictly adaptationist view of menopause, maternal investment is the key factor. Human females have menopause because their hominid ancestors who ceased reproducing before the end of their lives gained a fitness advantage over their still-fertile sisters in that they they could direct their remaining reproductive effort more profitably toward enhancing the reproductive success of existing progeny.[3,5–9,52–54] The fitness trade-off between greater investment in offspring already born versus producing more offspring, amounting to a quality versus quantity trade-off, very likely culminated during a time of rapid encephalization in the hominine line, which brought with it increases in infant altriciality and the prolongation of juvenile dependence.[8,9,52–54] With prolonged helplessness and nutritional dependence of offspring, each successive offspring imposed a greater cost on the mother,[6] in terms of the depletion of her physical reserves and the resources available for her to invest in existing offspring. With her own survival in question and the future of her existing offspring at stake, producing late babies with low survival probability was likely to lead to reproductive failure. In this scenario, menopause results from selection for reproductive cessation and is about stopping early.

There are two not necessarily mutually exclusive, adaptation hypotheses. The grandmother hypothesis is about inclusive fitness; the benefit comes from increasing the fertility of adult daughters and nieces, and the survival of their offspring. The principal proponents of this hypothesis are Hawkes, Blurton Jones, and O'Connell,[7,22,55] but many other researchers have examined it.[1,3,56–60] The mother hypothesis is about increasing the survival and fertility of one's own offspring.[5,6,8,9,61,62] The grandmother hypothesis of Hawkes and colleagues[7,22] and has recently

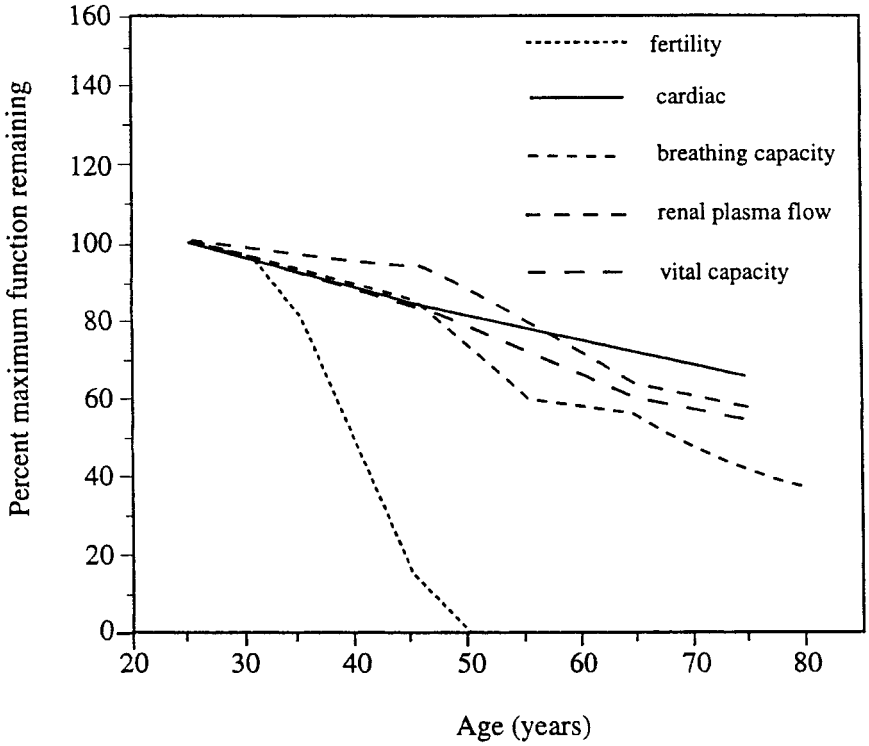

Figure 1. Comparative senescence of physiological functions in human females (redrawn from Hill and Hurtado[3]).

evolved from an explanation for menopause into an explanation for the exceptionally long postreproductive life span of human females.[22,55] In the old grandmother hypothesis, menopause is an adaptation facilitating grandmothering. In the new grandmother hypothesis, grandmothering is the adaptation facilitating increases in longevity; menopause is a by-product. Although the grandmother hypotheses have received more attention than the mother hypothesis, I have argued that both versions of the former are more problematic than the mother hypothesis.[62]

If menopause originated as an adaptation for postreproductive maternal investment, whether mothering or grandmothering, conditions had to exist that favored this adaptation. Box 2 lists the important predictions for which the evidence supports menopause as an adaptation, followed by the adaptation predictions that are not supported.

Reasons for Increased Maternal Investment

If menopause is an adaptation, we ought to find reasons in the course of human evolution for intensification in the quantity-quality trade-off between prolonged fertility and increased maternal investment. Two reasons can be advanced for substantial increases in maternal investment in the environment of evolutionary adaptedness: new ecological pressures due to changes in climate and diet and secondary altriciality of hominid infants due to cranial expansion and pelvic constraints.

Ecological pressures

Cooling and drying trends during the Plio-Pleistocene resulted in patchiness and seasonality in vegetation, necessitating dietary changes for survival.[21,63–65] For the hominine line, this dietary shift is believed to have led to expansion of home ranges, exploitation of a variety of habitats, and increasing foraging flexibility, including greater reliance on meat.[20,64–70] These changes required increased investment in offspring.

Secondary altriciality

In addition, there were periods of encephalization in the hominine line.[71–73] Whereas initially the limitation on increasing adult brain size was imposed by maternal metabolic output during gestation, there came a point when changes in pelvic dimensions facilitating more efficient bipedal locomotion put a stop to further rapid prenatal brain growth.[71] The resulting compromise was that the further rapid brain growth needed to attain greater adult brain size had to continue postpartum.[52,71,72] With much organizational development in addition to growth still to take place postnatally, *Homo* infants were born in a state of secondary altriciality, requiring intensified investment in infants and prolonged care of juveniles. Exactly when these life-history changes occurred is still debated. Many researchers believe major anatomical and behavioral changes began with *H. erectus* around 1.6 mya[72,75–78] (but see Ruff, Trinkhaus, and Holliday[79]). Recent studies agree that maximum life span has exceeded 50 years since early *H. erectus*,[74,80] which could indicate that menopause is 1.8 mya. However, early *H. erectus* (*H. ergaster*) is not generally associated with delayed maturity.[74,81,82] (but see Clegg and Aiello[77]). The first appearance of delayed maturity is estimated at 1.5 mya.[82] It seems reasonable that changes in rates of development kept pace with, but followed, increases in both adult brain size and secondary altriciality during human evolution.[76] It also would be logical to assume that the transformation from australopithecine to human-like life histories was completed by the time anatomically modern *H. sapiens* appeared on the scene. Thus, menopause could have arisen any time between 1.6 and 0.15 mya.

Menopause Unique to Human Females

If a protracted period of postreproductive maternal investment is the response to a unique set of socioecological, anatomical, and physiological pressures, and is also of critical importance to the way we evolved from our hominoid ancestors, we would expect menopause to be unique to humans or at least rare in other species.

Nonhuman primates

Some decline in age-specific fertility in later life is not uncommon among

Box 1. Background on Menopause

We know that menopause has been around for at least 3,000 years from a Biblical reference to Sarah in the book of Genesis (18:11),[19] which says, "it ceased to be with Sarah after the manner of women," from which we can infer not only that Sarah herself had stopped menstruating, but that the authors were quite familiar with the phenomenon. Although many researchers have speculated that menopause is a very old trait,[20–22] we do not know whether menopause has been around since the hominoid-hominid split, *Homo erectus,* anatomically modern *H. sapiens,* or simply since maximum life spans exceeded 50 years. The life span predicted from body and brain size in early *Homo* suggests that a female postreproductive life span predates *H. sapiens.*[23]

Although there is a strong central tendency in the age of menopause in developed countries, with medians clustering around 50 years, there is considerable variation in the age of menopause both within and between populations.[16] In a sample of American women, the age of menopause ranged from 40 to 59 years.[24] Any age within this range is considered normal. Across populations, medians range from 43 years in Central Africa to 51.4 years among Caucasian Americans.[16] With heritability estimates of 40% to 60% heritability in the age of menopause,[25–27] there is plenty of genetic variation to increase or decrease the mean age of menopause if such changes brought increased fitness. Yet research suggests that there has been no secular trend of any kind in the age of menopause over the last 150 years.[28–31] More importantly, Greek and Roman writings suggest that there probably has been little or no secular movement for the last 2,500 years. Aristotle (fourth century B.C.) and Pliny (first century A.D.) give 50 years as the maximum age of menopause.[32] Texts from the Middle Ages give 50 years as the average age, with a range of 35 to 60 years.[33,34]

Physiologically, menopause is the cessation of menses due to the depletion of oocytes.[35] Human females produce all the oocytes they will ever have by the fifth month of gestation. Semelgametogenesis, the character of producing all of one's gametes at one time, is a trait common to female birds and mammals.[11] In humans the maximum of approximately 7 million oocytes is reduced to 2 million by birth, 400,000 at puberty, and 1,000 at menopause.[36] Oocytes, which are surrounded by follicles in the ovaries, are lost mostly through a programmed process of cell death induced by hormone withdrawal, known as atresia.[37] Atresia is the sole cause of follicular death before puberty and remains the predominant cause thereafter, because the number of follicles lost to ovulation is relatively small.[38] About 400 oocytes are ovulated over the menstruating lifespan.[11]

Oocytes remain inactive in their follicles in an arrested phase of meiosis from the fifth month of gestation until they either succumb to atresia or become part of an ovulatory cohort.[39] Normally, only one oocyte in a cohort is singled out to complete meiosis; the rest provide hormonal support for the development of the primary follicle or oocyte, after which they too become atretic.[39] As the follicle pool shrinks, it becomes more and more difficult to recruit a large enough cohort of follicles to produce ovulation.[39] In addition, as human females age, the "chosen" oocytes become increasingly susceptible to malfunction during completion of the meiotic process, producing chromosomally abnormal ova.[39]

Physiological sources of variation in the age of menopause include the original number of oocytes and the rates of atresia.[35] At present, histological investigation of ovaries removed from females of all ages, including embryos, suggests that human females experience at least three different rates of atresia, from birth to puberty, from puberty to about age 40, and from age 40 to menopause.[37,40] The change in rate of atresia of greatest interest is the acceleration that occurs around age 40, because it is believed to be functionally related to menopause.[36,41] Without this apparent acceleration, which is thought to begin when some threshold number of oocytes remain (for example, 25,000), women would have enough oocytes to last 70 years.[42] It is unclear why the rate of atresia increases. However, it is likely that individual variation in the age at which the acceleration occurs is a major determinant of variation in the age of menopause.[38] Not surprisingly, given the high heritability in the age at which menopause occurs, women with a family history of clinically premature ($<$ 40 years) ovarian failure have earlier menopause.[43–45]

Several studies have examined the contribution of various environmental and life-history factors to variation in the age of menopause.[24,29,46–51] As with menarche, nutritional status has been a prime environmental suspect. However, the large-scale multivariate studies that have attempted to control for confounding variables such as socioeconomic status, ethnicity, marital status and parity have failed to show a nutritional effect.[16] Other suspected risk factors are body weight, weight loss, alcohol consumption, and stress. The only well-established environmental risk factor is long-term cigarette smoking, which lowers the median age of menopause by approximately 1.5 years.[29]

nonhuman primates.[83] Indeed, all iteroparous organisms can be expected to exhibit declining fertility as a function of general senescence.[84] However, in contrast to human females, nonhuman primates and even longer-lived species such as elephants, whales, and tortoises retain their capacity to reproduce until very old age. East African female elephants with a maximum life span of about 60 years still retain 50% of their reproductive capacity at age 55 years, an age which only 5% of the population ever reaches.[85–87] Female tortoises remain fertile far longer than 60 years,[53] and baleen whales are still reproductive in their nineties.[88] The exception to this general animal pattern are short-finned pilot whales (*Globicephala macrorynchus*). In this species, females experience menopause between the ages of 30 and 40 years and have a mean survival of $\geq$ 14 years after menopause.[4]

Box 2. Adaptation Predictions

Predictions Supported

1. Reasons for reproductive senescence in the environment of evolutionary adaptedness: altriciality of hominid infants and ecological pressures.
2. Protracted postreproductive life span unique to human females.
3. Fitness costs to prolonged fertility: reduced offspring survival.

Predictions Not Supported

4. Fitness costs to prolonged fertility: premature maternal death and sibling competition.
5. Fitness benefits of reproductive cessation.
6. Trade-off between fertility and longevity.
7. Nutritional contribution of females greater than males.
8. No negative health consequences of menopause.

In both field and captive studies, researchers have reported menopause-like physiological phenomena in monkeys and apes.[2,83,89–92] However, on close scrutiny of these reports, it is clear that the reproductive changes observed in nonhuman primates represent something different from human menopause, at least from a comparative life-history perspective.[1,2] The reproductive changes reported are idiosyncratic and generally far from species-wide, while age at reproductive cessation is extremely variable and postreproductive life spans are relatively short.[1,2] In field studies, the majority of the oldest individuals in all species investigated show no signs of ovarian failure.[1] In studies of captive primate species in breeding institutions, where extrinsic sources of mortality are minimal, on average about 67% of old females continued to reproduce all their lives, and in all species postreproductive life spans were still relatively short compared to those of humans.[83]

Perhaps the best way to illustrate how different the human female fertility pattern is from that of other primates is to compare the maximum age of reproduction and mean life expectancy at maturity of women and our closest relatives, *Pan troglodytes*. Hill and Hurtado's[87] comparative life-history diagram for Ache women, former foragers from Eastern Paraguay, and common chimpanzees shows that the maximum reproductive life span ends at age 42 years for Ache women and age 34 for chimpanzee females (Fig. 2). But whereas the average Ache woman who reaches maturity lives to age 60, the average reproductive-age chimpanzee female does not live past 27 years. In other words, half of all chimpanzee mothers never outlive their reproductive capacity; half of all reproductive-age Ache women live at least 18 years after reproductive cessation.

There is evidence that life spans exceeding reproductive capacity are part of our catarrhine legacy,[23] and it is clear that, given the right environment, female nonhuman primates will outlive their reproductive capacity. This means that in many species there is plenty of variation and raw material for selection to work on if a postreproductive life span were to become advantageous. It also implies that variation in the length of reproductive life span is an ancestral condition, indicating that selection for a postreproductive life span in hominid females would have been possible. Interestingly, in Caro and coworkers'[83] study, chimpanzees had the highest percentage of females terminating reproduction before death, with 60%.

Human males

Although human males usually exhibit a decline in fertility, in most cases this is a function of advanced overall senescence, ill health, or sociocultural and economic factors.[1,93] This is not premature reproductive senescence. Most human males are physiologically capable of siring offspring until very old age, and it is in their interest to do so. For males and females, the estimated hazard rates of reproductive cessation due to biological causes are very different. For males the hazard rate of reproductive cessation due impotence rises very

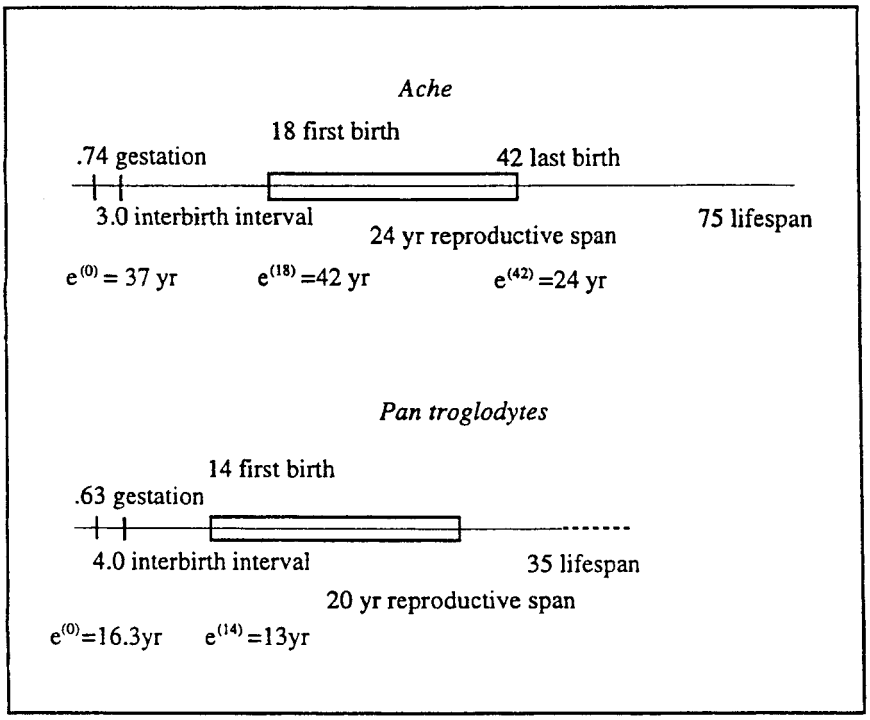

Figure 2. Comparative reproductive life histories of Ache women and female chimpanzees (redrawn from Hill and Hurtado[87]).

gradually after age 50 years, reaching only 0.3 at age 80. For females the hazard rate of reproductive cessation due to menopause rises sharply from less than 0.1 to almost 1.0 between the ages of 40 and 50.[16]

Fitness Costs of Prolonged Fertility

If menopause is an adaptation, we should find fitness costs of prolonged fertility in the form of reduced offspring survival and fertility. Here I will address three issues: reduced infant survival with late pregnancies in modern industrialized societies; the fitness cost of premature maternal death in one foraging population; and the fertility cost to offspring from sibling competition. Evidence supports only the first, although clearly more data from traditional societies are needed.

Elevated risk to baby associated with late pregnancies

If older mothers themselves are not at high risk of dying in childbirth, late pregnancy is still generally considered to carry high risk for fetuses and neonates, at least in Western societies for which we have data.[94–99] The risks include increased probability of fetal loss, stillbirth, and birth defects. Controlling for frequency of intercourse, older women experience increasing difficulty in becoming and staying pregnant.[16] Fetal wastage is the major contributor to fertility decline up to age 45 years.[16] The overall rate of fetal loss for women over 40 is 0.5. Maternal age is also predictive of an increasing incidence of most birth defects.[96] Indeed, 85% of the abortuses of women over age 40 have some detectable chromosomal abnormality, with Down syndrome representing the most dramatic example of this trend.[16] By age 43, the risk of carrying a baby with Down syndrome baby is one in ten.[100]

Fitness cost of premature maternal death

It used to be that the birth of each child jeopardized a mother's previous children because of her risk of dying in childbirth, a risk that increased with age.[16] In affluent Western societies of today, even though that risk is still seven times higher for women over the age of 40 than it is for 20-year-olds, the risk of dying in childbirth at any age is small.[16] Data from one traditional society—the Ache when they still lived exclusively from hunting and gathering—suggest that for hunter-gatherer women the risk of dying in childbirth is also small (about 1 in 150.)[87] Moreover, older women may not be at considerably greater risk.[87] If there was an increase in age-specific risk, it would have been minimal, since the annual mortality rate for 20-year-old and 50-year-old women was not much different (1% and 2%, respectively).[87] Thus, even though among the Ache a mother's death led to the certain death of her children under the age of one year and a five-fold increase in age-specific mortality for her children over that age, reduced offspring survival due to premature maternal death was not a major source of infant mortality. According to Hill and Hurtado,[87] "the assumption that older women should cease reproduction because they may die soon and their children will not survive maternal loss is notably incorrect" (p. 433).

Fertility costs to offspring from sibling competition

Regarding fitness costs to high and, by inference, prolonged maternal fertility in the form of reduced offspring fertility, data from the Ache suggest that fertility costs from sibling competition may be gender-dependent and variable. For example, during the forest period, there was a negative association between Ache daughters' fertility and number of siblings—that is, between daughters' fertility and mothers'.[87] The association between daughters' fertility and number of brothers was strongly negative. In contrast, there was a positive and much greater correlation between sons' fertility and number of siblings.

Fitness Benefits Associated with Reproductive Cessation

As a corollary to the fitness costs of prolonged reproduction, we expect to find fitness benefits associated with cessation of reproduction before death if menopause is an adaptation. To discover these benefits, researchers have traditionally relied on studies of the work performed by postreproductive women in extant foraging and horticultural populations. Evidence of these benefits is mixed.

In some traditional societies, including the Hadza,[7] the !Kung,[101] the Ye'kwana[57,102] (horticulturalists in Venezuela), and the residents of the Micronesian atoll, Ifaluk,[56] evidence supports the proposition that by providing material goods and services postreproductive women enable their daughters to raise more offspring, thereby contributing to their own inclusive fitness. Among the Hadza, the time women allocate to food acquisition continues to increase with age beyond menopause.[7,22] Older women spend significantly more time foraging than do females in any other age category. Variation in children's weight is correlated with grandmothers' foraging time. Although the hypothesis has not been formally tested, the implication is that relatively higher weight in grandchildren translates into a fitness advantage for the children and an inclusive fitness advantage for their hardworking grandmothers. However, hardworking grandmothers, like Hadza women aged 45 to 60, who bring in approximately 1,000 more calories on a daily basis than the average person consumes, appear to be exceptional.[60] Ache and Hiwi women never produce surplus calories.[60] In addition, even Hadza grandmothers provide less than 6% of the average daily protein intake.[60] Still, food sharing does not constitute the sole contribution of postmenopausal women to daughters' reproductive success in traditional societies. Grandmothers are frequent providers of child care and shelter and help with food processing and firewood collecting. Older females also acquire authority and are eligible for special status.[103] This newly acquired authority involves the ability to influence important decisions affecting younger kin, as well as the right to extract labor from younger family members. Accordingly, the work of older women tends to be administrative, involving the delegation and assignment of subsistence tasks to younger women. Nonetheless, Hill and Hurtado's[3,87] mathematical analysis, which was designed to test the hypothesis that reproductive cessation offers fitness-enhancing opportu-

nities, suggests that the fertility of Ache sons and daughters and the survival of grandchildren were not significantly increased by the presence of postmenopausal mothers or grandmothers. I appreciate the difficulties of detecting such effects. To begin with, both the costs and the benefits are probably difficult to establish in extant populations because menopause already exists. In addition, if women differ in access to resources, those with abundant resources may continue bearing healthy fertile daughters late in life, so that the phenotypic relationship we observe between mother's and daughters' fertility is positive, even if in less optimal conditions late fertility is associated with reduced fitness. [104]

Trade-Off Between Decreased Fertility and Increased Longevity

Here again, the evidence is mixed. According to life-history theory, increased longevity should come at the cost of reduced fertility.[105] Although there are many reasons why studies working with variation among individuals might fail to show such a trade-off,[18,104] when considered in historical perspective there does appear to be a trade-off between fertility and human longevity.[106] A recent study of an historical dataset containing life-history records gathered from the British aristocracy between the eighth and nineteenth centuries, reveals that for women who reached 60 years, longevity was negatively correlated with number of progeny and positively correlated with age at first childbirth.[106] In addition, for women, maternal and paternal heritability of longevity were about 40% and 20%, respectively. According to Westendorp and Kirkwood,[106] these findings are "compatible with the hypothesis that human life history has a heritable component that involves a trade-off between fertility and longevity" (p. 746).

In extant foraging societies, the extreme degree and duration of offspring dependence probably presents a fitness challenge for women even today. Yet a trade-off between maternal fertility and subsequent survival has not been identified. Data from the Ache do not suggest a trade-off, even controlling for access to resources.[87] This trade-off may be undetectable in the Ache because the relevant data come from a period of population growth and abundance when life-history trade-offs may have been relaxed, or because help from close kin with few or no dependents permitted relatives to enjoy high fertility without negative consequences.[87]

Female Investment in Offspring Greater than Male Investment

If the postreproductive life span of human females is an adaptation permitting increased maternal investment, we might expect female investment in the nutrition of their progeny to be more important than male investment, at least after menopause. This does not appear to be the case. In hunter-gatherer societies for which there are quantitative data, Kaplan and colleagues[60] show that juveniles and reproductive-age women do not meet their own energy requirements. Comparison of maternal effort among baboons and Ache foragers supports the notion that, despite the heavier burden of multiple dependents, human females work less hard during their reproductive years, whereas baboon mothers work harder.[107] However, it appears to be men, not postreproductive women, who provide the necessary nutritional supplement.[60] Even the famous hardworking Hadza grandmothers of Tanzania provide far fewer calories and protein through foraging than do Hadza male hunters of all ages. With new evidence of meat consumption during the Plio-Pleistocene[65,70] and new evidence of males' nutritional contribution in extant foraging societies, it is difficult to dismiss the importance of male investment in human reproduction. In general, women, young or old, do not appear to contribute more than males do to the nutrition of weaned offspring. Moreover, it is uncommon for postreproductive women to produce a nutritional surplus.[60] Male hunter-gatherers, in contrast, appear to subsidize the energetics of reproduction throughout their adult lives.[60] Thus, the evidence does not support menopause as an adaptation favoring postreproductive maternal investment, at least as far as nutrition is concerned.

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Negative Health Consequences of Menopause

Kenneth Hill[108] has suggested that "the female reproductive period is broadly protective of health" (p. 113) whereas, in affluent Western countries in particular, reproductive senescence is associated with increases in cardiovascular disorders, osteoporosis, and cognitive impairment.[46,107–117] The notion of menopause as an unhealthy state may not seem consistent with menopause as an adaptation. However, all adaptations have costs and benefits, and the evidence regarding negative health consequences of menopause is mixed. As Leidy[11] emphasizes, chronic conditions commonly associated with estrogen withdrawal need to be considered cross-culturally and historically. Menopause may not be the most important risk factor for heart disease or osteoporotic fractures.[114] Indeed, considering the incidence of osteoporosis in historical and cross-cultural perspectives indicates that diet and exercise levels are important variables determining the rate at which bone is lost.[118]

DISCUSSION

On balance, the evidence for selection for a postreproductive life span to increase maternal investment in existing offspring is inconclusive. I find little support for the notion that women, postreproductive or otherwise, are the major providers of the nutritional resources subsidizing human reproduction. However, the nutritional contribution of males versus females is probably the wrong comparison. First, food sharing is not the only contribution open to postreproductive females. Second, in sexually reproducing species males and females have an equal need for surviving offspring. What differs within and between species is how males and females allocate resources to somatic effort and reproductive effort, and how reproductive effort is divided between mating effort and parental effort.[119] When males contribute more than women do to the nutrition of their offspring, it is because women must allocate their energetic resources to other reproduction-related requirements. The diversity of human mating systems reflects the fact that male and female reproductive strategies differ within our species as a result of varying socioenvironmental conditions. Yet in all societies, historically and cross-culturally, women undergo menopause if they live long enough. If premature reproductive senescence is the result of certain conditions in the environment of evolutionary adaptedness, the universality of menopause implies that those conditions must still be present today. Menopause, or the postreproductive life span, must be the response to some unvarying constellation of pressures; my vote is for prolonged offspring dependence and overlapping child care.

Rapid encephalization required a change in female reproductive strategy. The challenge to females resulting from encephalization included the risk of the birth process itself,[120] the high metabolic costs of gestation and lactation,[16,52,121] offspring altriciality, and increased and prolonged dependence of juveniles.[6,64,122] It was not feasible to solve the problem of prolonged offspring dependence by increasing interbirth intervals. The period of dependence was too long: A mother faced a high probability of not surviving long enough to have a second child if she waited for the first to become fully independent before giving birth again. In the face of limited adult life expectancies,[123,124,125] the fitness-enhancing strategy was to shorten interbirth intervals and adapt to overlapping child care.

Besides optimizing birth spacing, overlapping child care also had some potential advantages. First, a mother with multiple dependents can reduce her total lifetime reproductive cost because certain tasks, such as protection against predators and conspecifics, provision of shelter, and food preparation, can benefit more than one dependent without the expenditure of much extra time and energy. Second, older siblings can help with provisioning and child care and, in the process, learn parenting skills. Still, even with male provisioning and some economies of scale associated with overlapping child care, early *Homo* females probably suffered chronic and increasing energy deficits during their reproductive years due to overlapping child care.[126–133]

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In a previous analysis of the adaptation hypotheses, I emphasize how difficult it is to show that premature reproductive senescence is the result of selection for increased maternal investment.[62] However, if menopause is an adaptation, I believe that mothering, not grandmothering, is more likely to be the reason. Although studies on grandmother investment are numerous, this approach is probably flawed, mainly because these women already have undergone menopause. They are not choosing between having another baby and helping daughters; their only option is helping. To me the mother hypothesis is more compelling, given Hamilton's[134] rule, low life expectancies in the environment of evolutionary adaptedness,[80,123,124] the likelihood of male philopatry,[133,135,136] as well as the expediency of overlapping child care and the logical inference that when fertility ceases a woman is still likely to have a last-born to care for.

THE EPIPHENOMENON EXPLANATIONS

I come to the two explanations that cast menopause as an epiphenomenon: the physiological trade-off favoring efficient early reproduction and the by-product of increased longevity hypotheses. There is less to say about these, for until recently most evolutionary biologists and anthropologists have dismissed them as proximate explanations that fail to justify the postreproductive life span in human females. The underlying assumption of both these explanations is that evolution is constrained by phylogenetic history, developmental limitations, and genetic correlations.[18] In both cases, response to selection is limited by the "technology" of an organism, which forces design trade-offs and negatively correlated, or antagonistic, responses. For example, a gardener might wish to breed peapods containing more and larger peas, only to dis-

cover that she cannot have both, either because oversized pods are too heavy to ripen on the vine (design trade-off) or because the size and number of peas are controlled by negatively correlated genes (antagonistic pleiotropy). Negative genetic correlations can result in intertemporal physiological trade-offs as well. Such is thought to be the case with reproductive senescence in human females: Pleiotropic genes are favored because they have positive effects at younger ages, even though they have negative effects later in life.[10–12,137] According to evolutionary theories of senescence, such time-delayed antagonistic pleiotropy can exist because selection against fitness-reducing traits weakens with age.[5]

THE PHYSIOLOGICAL TRADE-OFF

Given semelgametogenesis, the reason for a physiological trade-off favoring efficient early reproduction is that human female reproductive physiology should be designed to maximize reproductive output early in life before the dwindling supply of oocytes jeopardizes hormonal support for ovulation[12,38,137] and to terminate fertility before the negative consequences of "old eggs" predominate.[10] Selection might favor a longer reproductive life span in human females, but selection for efficient early fertility, which results in decreased fertility and eventual sterility in later life, provides greater lifetime reproductive success. Selection for efficient early fertility is not about starting reproduction early. In fact, the hypothesis is silent regarding when reproduction begins; what is at issue is the intensity of reproduction early in the fertile life span. In this scenario, as in the adaptation theory, reproductive senescence is about stopping early.

The physiological trade-off explanation and the adaptation explanation are not necessarily mutually exclusive. Besides the fact that selection for efficient early fertility can be considered an adaptation in its own right—for example, to accommodate overlapping child care—antagonistic pleiotropy or a design trade-off could be the proximate mechanism creating a postreproductive life span if that were favored. The difference between the adaptation and physiological trade-off hypotheses is that in the latter menopause per se is not what you are seeking to explain. What needs explaining is why age-specific fertility begins decreasing decades before menopause. Menopause is simply the last event in the process of declining fertility. Whether menopause is the result of selection for efficient early reproduction or selection for a postreproductive life span is exceedingly difficult to tease apart. Indeed, if overlapping child care is the truly unique reproductive character in human females, both the adaptation and physiological trade-off explanations are applicable. At any rate, these two explanations share some of the same predictions: long postreproductive life spans should be unique to human females; prolonged fertility should have fitness costs in the form of reduced offspring survival and fertility; and fitness benefits should be associated with reproductive cessation. Earlier, we saw that a long postreproductive life span is unique to human females, but that the costs of prolonged fertility and the benefits of reproductive cessation are more difficult to establish. There are also two predictions that are specific to the physiological trade-off favoring early fertility.

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Historical and Cross-Populational Evidence of Early Fertility

In support of the physiological trade-off explanation, we find historical and cross-population evidence of selection for early fertility in human populations. In extant and historical natural-fertility populations, total fertility rates vary enormously, as do nutritional status and mortality rates.[16,87,138,139] Despite these differences, the pattern of age-specific marital fertility remains remarkably consistent across time and geographical boundaries, peaking around age 25, then decreasing monotonically with the cessation of fertility, generally preceding menopause by several years (Fig. 3).[140] This supports the importance of a biological explanation for reproductive cessation before menopause and for menopause itself.

Additional indication that the causes of the premenopausal cessation of fertility are physiological comes from analysis of age-specific apparent fecundabilities, or monthly probabilities of a recognized conception. Based on data from Taiwanese and Hutterite women and a statistical model, Wood and Weinstein[141] showed that apparent fecundability begins to decline around age 25 years. Declining coital rates are responsible for most of the yearly decrease in apparent fecundability up to age 35.[140,141] After age 35, biological causes take on major importance, eventually totally predominating.[16] The model suggests that the biological portion of the decline stems from the increasing risk of early fetal loss.[140]

Further validation of the physiological trade-off explanation comes from Wood and colleagues.[12,142] According to these investigators, the menstruating life spans of women of all ages are characterized by periods of ovarian inactivity in which regular cycling and, presumably, ovulation, do not occur because the production of ovarian steroids is insufficient to maintain negative feedback on lutenizing hormone and follicle-stimulating hor-

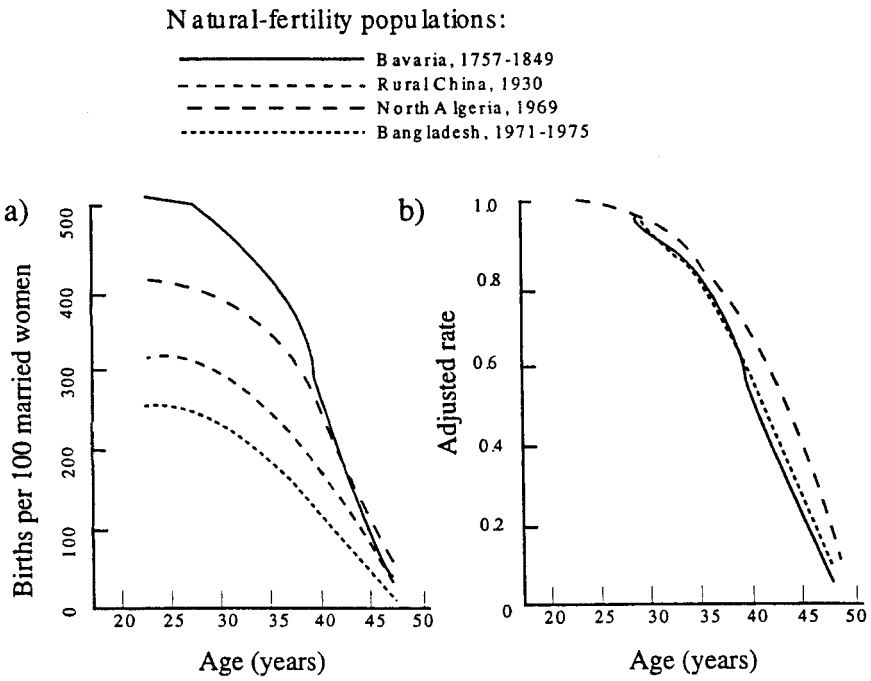

Figure 3. a) Comparison of age-specific marital fertility rates in natural fertility populations and b) age-specific marital fertility rates corrected for differences in total fertility rates. Raw rates at each age are divided by the rate at age 20 to 24 years. (Redrawn from Wood[16]).

mone, which is essential for ovulation. The pattern of these "inactive phases," which increase in frequency and duration as menopause is approached, is predictable from the age pattern of follicular depletion, suggesting that the characteristics of the follicular-depletion system determine the distribution of inactive phases and the probability of ovulatory cycles.[12,142] Wood, O'Connor, and Holman[12] hypothesize that menopause evolved by antagonistic pleiotropy because of selection acting on the follicular depletion system to maintain regular ovarian cycles at young adult ages.

Fertility in Other Mammals

The fact that biological factors involving a design trade-off and antagonistic pleiotropy (or both) appear to be responsible for the early reproductive peak in human females is still insufficient to explain menopause. One must also show that fertility functions of humans are different from those of other mammals. One must show that human females are unique both in terms of their early fertility peak and long postreproductive life span. Fertility patterns for nonprovisioned or semi-provisioned species such as Barbary macaques,[143] olive baboons,[144] lions,[144] and East African elephants[145] do, in fact, look very different from the human pattern.[146] Instead of resembling a left-skewed triangle with fertility starting to decrease when females are in their mid-twenties, the age-specific fertility functions of macaques[143] and elephants,[145] are box-like, with fertility remaining relatively constant over a relatively long period, then terminating abruptly only a few years before maximum age at death (Fig. 4). For Mahale chimpanzees,[71] there is no correlation between females' age at last birth and length of interbirth interval [$r = 0.09 + -8.66$, $N = 19$], suggesting that noncaptive chimpanzees also have a box-like fertility pattern.[91] Many similar examples can probably be adduced. In addition, though some female nonhuman primates living in captivity show age-specific fertility patterns similar to those of humans, only among chimpanzees do more than than 50% of females have a postreproductive life span.[83] The finding that chimpanzees have menopause and a post reproductive lifespan if they live long enough is consistent with the similarity between the follicular depletion patterns and mechanisms in humans and chimpanzees.[12]

Across mammals, females' supply of follicles at puberty scales allometrically with species body size and life span.[15] To allow for the fact that the number of follicles at puberty rises more steeply with body weight than with life span, and to insure a lifetime supply, ovulation rates vary inversely with size.[15] At maturity, human females have the number of follicles predicted by body weight, and there is no evidence for a relatively high rate of follicular attrition.[15] In fact, there is some evidence that longer-lived species use follicles more conservatively.[15] Still, in other species senescence and death usually precede depletion of oocytes, whereas the postreproductive life span of human females is long and universal.[147] The questions, then, are why does human fertility start to decrease when women are in their mid-twenties and terminate when they are around age 40, and why do human females outlive their supply of eggs? Leidy[148] hypothesizes that "the process of follicular atresia evolved as integral to the process of sexual reproduction and that the entire hominid somatic lifespan was more amenable to change than was the process of atresia" (p. 149). In my view, selection for efficient early reproduction is an adaptation, accommodating prolonged offspring dependence and the expedience of overlapping child care. Indeed, I consider the physiological trade-off favoring early fertility to be the most powerful explanation of all. In addition, although the proponents of selection for early fertility do not address the value of the postreproductive life span, a postreproductive period, albeit a modest one at first, would have afforded a fitness advantage if it increased offspring survival. That brings us back to the adaptation hypothesis.

ARTIFACT OF LONGER LIFE SPANS OR LIFE EXPECTANCIES

I come to the explanation of menopause as a by-product of long life span or increase in life expectancies. The elucidation here is that, given the phylogenetic, physiological, and developmental constraints of semelgametogenesis, menopause is simply the result of human life spans and life ex-

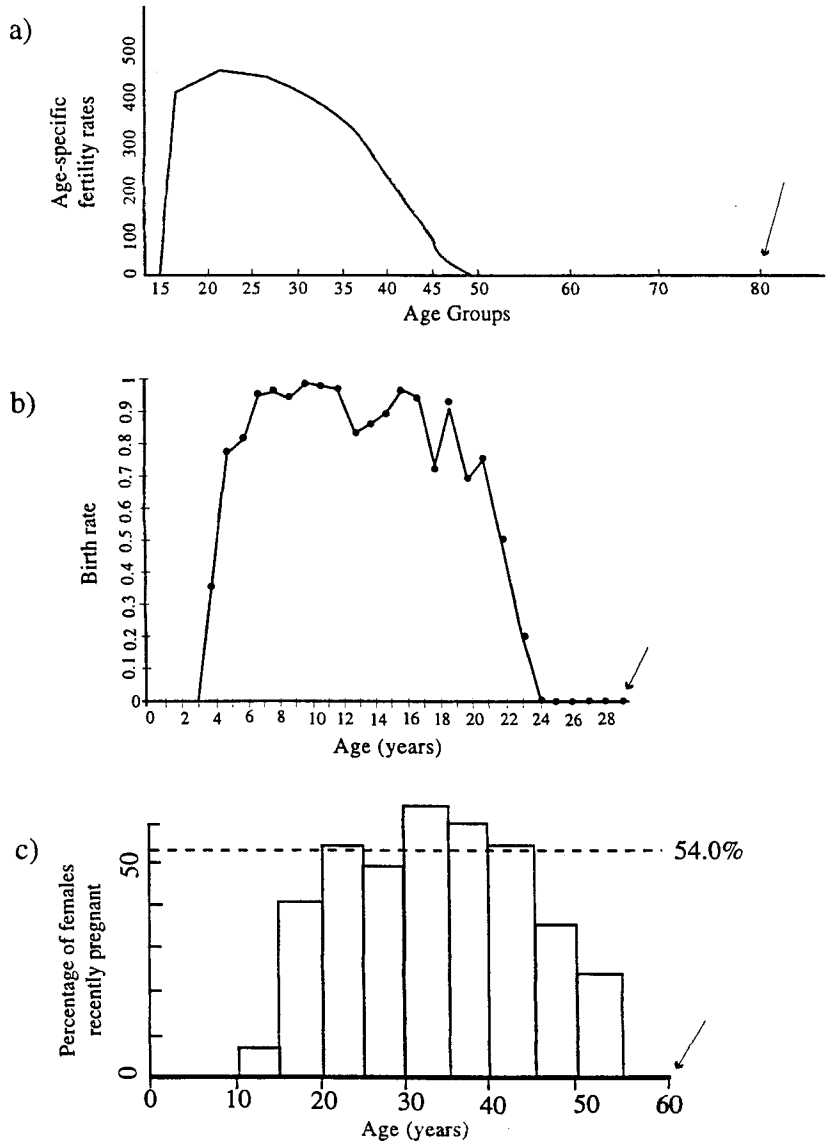

Figure 4. Age-specific fertility functions: a) Hutterite women, b) semi-free-ranging Barbary macaques, and c) East African elephants. Arrows indicate maximum age at death (redrawn from Robinson[146]; Paul, Keuster, and Podzuweit[143]; and Laws, Parker, and Johnstone[144]).

pectancies increasing beyond the female's supply of eggs or ability to sustain ovulatory cycles.[13–17]

The physiological trade-off and artifact explanations are not mutually exclusive for two reasons. First, design trade-offs and antagonistic pleiotropy would be the proximate mechanisms preventing increases in reproductive life span. Second, increases in life span and life expectancies may be partly the result of selection for early fertility if reproductive effort released by the cessation of fertility is directed toward increased somatic repair or if the cessation of childbirth increases life expectancies for mothers and offspring. The artifact and adaptation explanations are not mutually exclusive either. If the dissynchrony between reproductive and somatic senescence arose in human females because selection for a postreproductive life span was greater than selection for longer fertility, menopause would be more adaptation than artifact.

As an explanation for the origin of menopause, we can dismiss the hypothesis that a postmenopausal life span is the result of a recent increase in life expectancies due to improved sanitation and medical care. Some females have lived past menopause for at least 3,000 years, and during most of this time living conditions for most women were certainly not very good. This leaves us to explain why life span exceeded reproductive capacity.

Females of other long-lived species do not have menopause—recall elephants, tortoises, and most whales, which live to be 60, 80, and 90 years old, respectively. According to Diamond[53] there "is nothing evolutionarily inevitable about menopause from the perspective of mammals in general. Other species maintain fertility and viable oocytes longer than humans" (p. 116). Human females use up their supply of oocytes approximately half-way through the maximum life span.[2] In contrast, follicular depletion before the end of life is rare in other female mammals.[147] Females in other mammalian species rarely have postreproductive life spans; when they do, these are relatively short.[2]

According to evolutionary theories of senescence, there should be no selection for postreproductive individuals. Reproductive senescence is equivalent to death.[5] But no one is truly postreproductive. As Williams[5] put it, no one is truly postreproductive until the last child is self-sufficient. Diamond[53] goes further, stating that people are never truly postreproductive if they can influence the fitness of biological relatives. The notion that there can be no selection against mutations causing damaging effects in old people because they are postreproductive overlooks this fact. Especially in preliterate foraging societies, Diamond[53] notes, older folk are valued for their ability to share their vast store of knowledge, in particular their knowledge of matters concerning survival. Accordingly, older individuals, including postreproductive females, could have been valued even before the origin of spoken language, when communication still relied on vocalizations, signs, and facial expressions. This makes it possible that menopause is a very old trait indeed. As for how humans evolved longer life spans, Diamond[49] suggests that perhaps we live longer because we evolved better repair mechanisms as we gained more control of our environment.

If human females have the number of follicles predicted for a mammal of

our body weight at maturity, and if there is no evidence for a relatively high rate of follicular attrition, despite the acceleration in atresia,[15] why do we run out of oocytes prematurely? According to Gosden and Telfer,[15] the odd thing about human females is that, based on body weight, allometry predicts a life span of only 30 years. These researchers believe this constitutes "a *prima facie* case for arguing that menopause has arisen adventitiously during evolution" due to increases in life span (p. 174). In contrast, Judge and Carey[23] predict a human life span of 72 years based on body and brain mass of a catarrhine comparison group. Observing that this estimate exceeds average longevity in hunter-gatherers and the age of menopause by at least 20 years, Judge and Carey suggest "that a lifespan exceeding that of the female reproductive system is part of the phylogenetic legacy rather than a modern development related to uniquely human cultural innovations" (p. B205).

In the patriarch hypothesis Marlowe[17] posits that menopause is the by-product of selection for extension of maximum life span in males. The life span of females co-evolved with that of males, whereas commensurate increases in female reproductive life span were constrained by the depletion of viable oocytes. Without justifying the postreproductive life span, the artifact explanation is insufficient. However, menopause could be a result of life span outlasting female reproductive capacity if postreproductive females had indirect reproductive value.

Heritability in the Age of Menopause

Additional insight into the investigation of menopause and the adaptation-versus-epiphenomenon debate is provided by estimates of heritability in the age of menopause because heritability provides information about the history of selection on a trait.[141–151] Indeed, heritability estimates are particularly useful in this case because researchers have had to resort to a good deal of speculation about the origin of menopause, while empirical work on the maintenance of the trait is hindered by the fact that menopause is universal in our species. From a heritability estimate, we learn about the maintenance of menopause from the stability of the mean age of menopause over the relatively recent past—say, the last 2,000 years. We learn about the long-term maintenance of the trait from the amount of additive genetic variance (V_A).[152]

As discussed earlier, the evidence, though not conclusive, suggests that the age of menopause has remained relatively stable for a few thousand years throughout the world, despite tremendous socioeconomic and demographic change. With no upward secular trend in the mean age of menopause and heritablities as large as 40% to 60%,[25–27] there are three possible explanations. The first is that age of menopause has experienced upward movement that we have not detected. Lack of evidence for a secular trend is not definitive. The second is that age of menopause is selectively neutral. The third is that age of menopause is under some degree of stabilizing selection.

According to evolutionary theories of senescence, there should be no selection for postreproductive individuals. Reproductive senescence is equivalent to death. But no one is truly postreproductive.

High heritability and no discernible upward movement in the mean age of menopause in the presence of improving socioeconomic conditions suggests a cost to prolonged fertility now, in modern industrialized and traditional agricultural societies, which supports the third option. By implication, this cost has existed since the domestication of plants and animals about 10,000 years ago. Finding a cost to prolonging female reproductive life span now is significant because it changes the terms of the menopause debate. Previous investigation has focused on the socioecology of hunter-gatherers in an attempt to understand the selection pressures of the environment of evolutionary adaptedness. The fact that there is presently a cost, as many have suspected, means that there is nothing peculiar to the foraging way of life that makes premature reproductive senescence adaptive. This is not unexpected, given the preponderance of evidence suggesting that reproductive patterns in human females are remarkably consistent across populations and time, regardless of mode of subsistence.

The next question is whether stabilizing selection on the age of menopause is weak or strong. Strong stabilizing selection is usually associated with low heritabilities, because strong selection is generally expected to reduce additive genetic variation.[149,150,153,154; cf.151,155] (Under certain circumstances, however, high heritability can remain in the presence of strong stabilizing selection.[151,156–159]) This implies that a broad range of intermediate ages of menopause is favored, and that menstruating life span is not correlated with lifetime reproductive success.[149] In this case, menstruating life span would have a flat fitness profile, perhaps with selection only against very early or very late menopause. Over much of its range, age of menopause could be considered a neutral trait. Weak stabilizing selection on the age of menopause could be the result of selection for some other trait that affects the age of menopause, such as selection for early fertility or a postreproductive life span. The nearly universal age at last birth of approximately 40 years, regardless of median age of menopause, is consistent with this notion. To me this represents further support for the hypothesis that menopause is an epiphenomenon of selection for efficient early reproduction. In this scenario, menopause is both the result of selection for intensive reproduction early in the fertile period to accommodate prolonged dependence of offspring through overlapping child care and an adaptation for more effective maternal investment, including a modest postrepro-

ductive life span. The long postreproductive life spans now enjoyed by human females resulted from much later increases in life expectancies.

CONCLUSIONS

Undoubtedly all three explanations—adaptation, trade-off, and artifact—played some role in the origin of menopause. Indeed, selection for a postreproductive life span in human females was probably the result of a dynamic interplay among the need for prolonged offspring care, physiological trade-offs favoring easy fertility, and increasing lifespans and life expectancies. Supporting the adaptation explanation is the fact that a protracted postreproductive life span is unique to human females. Given this, it is reasonable to hypothesize that ecological pressures and increasing secondary altriciality of hominid infants provided compelling reasons for selection for intensified and protracted maternal investment in the environment of evolutionary adaptedness.

In support of the physiological trade-off explanation, there is strong evidence of selection for "front-loaded" fertility. Cross-populational and historical fertility patterns are remarkably consistent in showing that age-specific fecundability and fertility peaks when human females are in their mid-twenties and terminate around age 40, well before menopause and irrespective of the cross-populational variation in the age of menopause. This pattern is different from that of other mammalian females. Evidence of the continuing importance of efficient early fertility is provided by recent heritability estimates of 40% to 60% for the age of menopause and the apparent lack of upward secular trends. In the presence of so much additive genetic variation, lack of increase in the age of menopause suggests that there is a cost to prolonging fertility now. This in turn suggests that the cost of prolonged fertility is not exclusive to socioeconomic conditions of the environment of evolutionary adaptedness or to any particular mode of subsistence. The reason for premature reproductive senescence still exists. Also in favor of selection for early fertility is the fact that loss of oocytes accelerates when women are in their late thirties. In addition, selection for early fertility via antagonistic pleiotropy is also consistent with the greater cross-populational variation in median age of menopause than in mean age at last birth, suggesting greater selection on the end of fertility than the end of menstruation.

I find no support for the notion that menopause is the result of recent increases in life expectancies, given that it probably existed at least 3,000 years ago, long before the occurrence of significant increases in life expectancies. The fact that human females have enough oocytes to last 70 years also does not support the hypothesis that menopause is purely the result of increases in life span. Nevertheless, in the presence of selection for efficient early reproduction, increases in life span or life expectancies would have resulted in a postreproductive life span for human females. On the other hand, the prolonged postreproductive lifespan now enjoyed by virtually all human females who reach reproductive age is very likely the result of relatively recent increases in life expectancies.

If premature cessation of fertility and increased maternal investment resulted in selection for longer life span in females, my hunch is that it was primarily the result of mothering: Women were better off investing in the survival and fertility of their own subadult offspring than in grandchildren or nondescendant relatives. Given the contribution of males to the energetics of human reproduction, longer-lived males would also have enjoyed a fitness advantage. There is no need to invoke a correlated response. On the basis of the preceding, I argue that selection for efficient early reproduction was and still is a fitness-enhancing adaptation accommodating prolonged offspring dependence by facilitating overlapping child care. Thus, ultimately, premature reproductive senescence can also be viewed as an adaptation for prolonged maternal investment.

Obviously, the origin of menopause will never be explained by physiology alone, but because it is reasonable to assume that female reproductive physiology has remained basically the same since the origin of *Homo,* we may want to focus on the currently recognized physiological mechanisms governing declining fertility and menopause for what they can tell us about the environment of evolutionary adaptedness and the ultimate reasons for menopause. I believe the most important clue lies in the fact that human females have enough oocytes to last 70 years if the rate of atresia were to remain constant throughout adult life.

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