A Critique of the Grandmother Hypotheses: Old and New

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ABSTRACT   The singularity of reproductive senescence in human females has led many investigators to consider menopause an adaptation permitting increased maternal investment in existing progeny. Much of the focus has been on the grandmother hypothesis—the notion that aging women gain an inclusive fitness advantage from investing in their grandchildren. This hypothesis has evolved from an explanation for menopause into an explanation for the exceptionally long postreproductive lifespan in human females. In the old grandmother hypothesis, menopause is an adaptation facilitating grandmothering; it is about stopping early in order to create a postreproductive lifespan. In the new grandmother hypothesis, grandmothering is an adaptation facilitating increased longevity, and menopause is a byproduct. This paper reviews and critically evaluates the evidence for and against both hypotheses, focusing on key predictions of each. If menopause is the result of selection for increased maternal investment, this involved mainly mothers, not grandmothers. Am. J. Hum. Biol. 13:434–452, 2001.   © 2001 Wiley-Liss, Inc.

Defined as a nonfacultative and irreversible cessation of fertility which occurs in all female conspecifics well before the senescence of other somatic systems and the end of the average adult lifespan, menopause is virtually unique to human females (Marsh and Kasuya, 1984; Hill and Hurtado, 1991; Pavelka and Fedigan, 1991). 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 lifespan to permit increased maternal investment in existing progeny (Williams, 1957; Hamilton, 1966; Lancaster and Lancaster, 1983; Hawkes et al., 1989; Hill and Hurtado, 1991; Peccei, 1995a,b). Investigation of the adaptation hypotheses has focused mainly on the inclusive fitness which older women gain from investing in reproductive-aged daughters and their offspring (Hawkes et al., 1989, 1997, 1998; Hill and Hurtado, 1991; Rogers, 1993). Other researchers suggest that premature reproductive senescence is the result of selection for efficient early reproduction (Pavelka and Fedigan, 1991; O'Rourke and Ellison, 1993; Leidy, 1999; Wood et al., 1999; Peccei, 2001), whereas yet others view menopause as the byproduct of increases in lifespan or life expectancies (Washburn, 1981; Weiss, 1981; Wood, 1994a). This paper reviews the evidence for the adaptation theory and examines and evaluates evidence for and against the grandmother hypotheses.

THE ORIGIN OF MENOPAUSE: ADAPTATION

The adaptive explanation for menopause is straightforward. At some point in human evolution females who ceased to reproduce before the end of their lives gained a fitness advantage over their still-fertile sisters because they could direct their remaining reproductive effort more profitably toward enhancing the reproductive success of existing progeny (Williams, 1957; Lancaster and Lancaster, 1983; Hawkes et al., 1989; Hill and Hurtado, 1991). The fitness trade-off between investing more in offspring already born as opposed to producing more offspring amounted to a quality versus quantity trade-off. Researchers have speculated that menopause is a very old trait (Hill, 1982; Foley, 1987). Menopause possibly appeared during a time of rapid encephalization in the hominine line, which brought with it increased secondary altriciality of hominid infants and greater and more prolonged offspring dependence (Trevathan, 1987; Diamond, 1992; Peccei, 1995a,b; Bogin and Smith, 1996; Turke, 1997). Exactly when these life history changes occurred is still debated. Many researchers believe major anatomical and behavioral changes began with *Homo erectus* around 1.6 mya (Leonard

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and Robertson, 1992; Smith and Tompkins, 1995; cf. Ruff et al., 1997; Clegg and Aiello, 1999; O'Connell et al., 1999; Wood and Collard, 1999). It is likely that increases in adult brain size and secondary altriciality as well as changes in rates of development kept pace during human evolution (Smith and Tompkins, 1995), and it 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.

ADAPTATION HYPOTHESES

There are actually three adaptation hypotheses: the grandmother hypotheses—old and new—and the mother hypothesis. The grandmother hypotheses are about *inclusive* fitness. The benefit of early reproductive cessation comes from increasing the fertility of adult daughters and nieces and the survivorship of their offspring. Principal proponents are Hawkes, Blurton Jones, and O'Connell (Hawkes et al., 1989, 1997, 1998), although others have dealt with different aspects of the grandmother hypothesis (Turke, 1988; Hames, 1988; Hill and Kaplan, 1988; Hill and Hurtado, 1991, 1996; Pavelka and Fedigan, 1991; Rogers, 1993; Kaplan et al., 2000). The mother hypothesis, in contrast, is about increasing the survivorship and potential fertility of one's own subadult offspring (Williams, 1957; Gaulin, 1980; Lancaster and King, 1983; Lancaster and Lancaster, 1983; Peccei, 1995a,b). The original grandmother hypothesis and the mother hypothesis are not necessarily mutually exclusive, since both are about stopping early.

The grandmother hypothesis of Hawkes et al. (1989, 1997, 1998) has evolved in recent years. Hawkes and colleagues no longer invoke the fitness benefits of grandmothering to explain *menopause* as an adaptation. Rather, *human longevity* is viewed as the result of the inclusive fitness garnered by hardworking postreproductive females. In hypothesizing that the ability to provide difficult-to-find-and-process food allowed females to outlive their reproductive capacity and maintain reproductive value, the postreproductive lifespan is still an adaptation, but menopause itself has become an epiphenomenon of increased longevity. Menopause is no longer about stopping early in the new grandmother hypothesis. No explanation is proffered as to why female reproductive capacity did not keep up with increases in lifespan.

According to evolutionary theories of senescence, there should be no selection for postreproductive individuals (Williams, 1957). Hence, all three hypotheses have the burden of demonstrating that a female postreproductive lifespan is fitness-enhancing. The stopping early hypotheses say nothing about how human lifespans and life expectancies increased, although many potential explanations follow from them. Ways in which premature reproductive senescence could have increased longevity have been suggested (Peccei, 1995a,b, 2001); these will be discussed later. The new grandmother hypothesis provides an explanation for human longevity and the long postreproductive lifespan in women, but it has various problems. Both grandmother hypotheses overestimate the importance of older women, past and present, in the energetics of human reproduction. If menopause is an adaptation, it most likely involved mothers, not grandmothers.

MENOPAUSE AS AN ADAPTATION

Before the relative merits of the grandmother and mother hypotheses are discussed, it is useful to review the evidence for menopause and/or the female postreproductive lifespan as an adaptation. Although there are a number of positive features, there are also real questions as to whether menopause is in fact an adaptation for prolonged maternal investment. These questions and the evidence are summarized (see Peccei, 2001, for a more detailed discussion).

There is evidence supporting the adaptation explanation. Two reasons can be advanced for a trade-off between prolonged fertility and increased maternal investment in the evolutionary past: (1) ecological pressures stemming from cooling and drying trends in the Plio-Pleistocene [1.7–1.9 mya], which necessitated changes in diet, including an increasing reliance on meat and difficult-to-access plant foods (Isaac, 1978; Hill, 1982; Vrba, 1985; Foley, 1987; Foley and Lee, 1991; Kingston et al., 1994; Plummer and Bishop, 1994; Sikes, 1994; Asfaw et al., 1999; deHeinselin et al., 1999; O'Connell et al., 1999); and (2) the secondary altriciality of hominid infants due to cranial expan-

sion and pelvic constraints, which occurred in the Early Pleistocene (Martin, 1983; Stanley, 1992). In addition, if a protracted period of postreproductive maternal investment was the response to this unique set of socioecological, anatomical and developmental pressures, menopause should be unique to human females, and there should be clear differences between the reproductive patterns of human females and those of human males and females in other primate species. The evidence strongly supports this prediction (Harman and Talbert, 1985; Pavelka and Fedigan, 1991; Wood, 1994a; Hill and Hurtado, 1996). For example, half of all chimpanzee mothers in Gombe and Mahale never outlive their reproductive capacity, whereas among the Ache, hunter-gatherers in Paraguay, half of all reproductive-aged women live at least 18 years after reproductive cessation (Hill and Hurtado, 1996). When fertility declines in human males, in most cases this is a function of advanced overall senescence, ill health, or sociocultural and economic factors (Harman and Talbert, 1985; Wood, 1994a). Further, there also appears to be a trade-off between fertility and longevity in human females, but only when the relevant data are considered over a period of centuries (Westerndorp and Kirkwood, 1998).

The adaptation explanation also has troubling features. If the postreproductive lifespan in human females is an adaptation permitting increased maternal or grandmaternal investment, postmenopausal women should be contributing in a major way to the nutrition and safety of their offspring and grandoffspring in order to maintain reproductive value. It might even be expected that postreproductive women work harder and produce more than males and reproductive-aged women. This notion is the premise for a good deal of research on the value of menopause (Hawkes et al., 1989, 1997; Blurton Jones et al., 1989; O'Connell et al., 1999). However, new evidence of meat consumption as far back as 2.5 mya (deHeinselin et al., 1999), and new evidence on the nutritional contribution of males in extant foraging societies (Kaplan et al., 2000) seems to belie this point. Without getting into precisely who consumes the food that men acquire and how subsistence contributions affect the reproductive success of males in extant foraging societies, it appears that, in general, women do not con-

tribute more than males to the nutrition of offspring before or after menopause and that postreproductive women do not produce more than reproductive-aged women (Kaplan et al., 2000). The production of women hunter-gatherers may be dedicated mostly to their own offspring, but less of what children eat is provided by both mothers and grandmothers. This does not imply that women are shirkers. It merely suggests that one cannot overlook male investment in an explanation of menopause, as Hawkes et al. (1998) do. If men contribute more than women to the nutrition of offspring, it is because women must allocate their energetic resources to other reproduction-related requirements.

If menopause is an adaptation for a postreproductive lifespan, there should be fitness costs and benefits associated with prolonged and curtailed fertility, respectively. Fitness costs to prolonged fertility in the form of reduced infant survivorship have been found, but neither reduced offspring survivorship due to premature maternal death nor fertility costs to offspring resulting from mothers' fertility are supported (Naeye, 1983; Spellacy et al., 1986; Hill and Kaplan, 1988; Gaulden, 1992; Wood, 1994a; Fretts et al., 1995; Hill and Hurtado, 1996). Evidence of fitness benefits associated with cessation of reproduction before death is mixed. For example, among the Hadza, hunter-gatherers in Tanzania, variation in children's weight is correlated with grandmothers' foraging time (Hawkes et al., 1998). In contrast, for the Ache during the forest-dwelling period, fertility of sons and daughters and survivorship of grandchildren is not significantly increased by the presence of postmenopausal mothers/grandmothers (Hill and Hurtado, 1991, 1996). Both the costs and the benefits of reproductive cessation are probably difficult to establish in extant populations because menopause already exists.

If the postreproductive lifespan is supposed to enhance female fitness, postmenopausal women should suffer no negative health consequences beyond "normal" aging-related deterioration. Evidence for negative health consequences of estrogen withdrawal is mixed. Although estrogen replacement therapy appears to slow bone loss, prevent cardiovascular disease [CVD] in some women, and alleviate symptoms of Alzheimer's disease, prolonged estrogen

production or replacement is associated with increases in certain cancers (Colditz et al., 1987; Avioli, 1994; Kuller et al., 1994; Paganini-Hill and Henderson, 1994; Kannel et al., 1996; Hill, 1996; Brace and McCauley, 1997; Henderson, 1997; Byington et al., 1999). As Leidy (1999) 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 CVD or osteoporotic fractures (Khaw, 1992). For example, consideration of the incidence of osteoporosis in historical and cross-cultural perspectives indicates that diet and exercise level are important variables determining the rate at which bone is lost (Lees et al., 1993).

On balance, when the adaptation hypotheses are considered together, the evidence for selection for menopause and/or a post-reproductive lifespan to increase and prolong maternal investment in existing progeny is inconclusive. To further investigate menopause as an adaptation it is useful to examine the evidence for and against the individual hypotheses separately. The grandmother hypotheses are the primary focus here. The mother hypothesis has been considered in some detail elsewhere (Peccei, 1995a,b, 2001).

Troubling points common to both grandmother hypotheses

Four issues that present problems for both grandmother hypotheses are considered:

(1) Cross-population qualitative and quantitative evidence for helpful grandmothers is not compelling.
(2) Mathematical models do not support the fitness advantage of reproductive cessation in favor of increased investment in adult daughters and grandchildren.
(3) Both hypotheses require female philopatry, which does not appear likely in the hominine line.
(4) The role of males and siblings is ignored.

Cross-population qualitative and quantitative evidence

In some traditional societies, e.g., the Hadza in Tanzania, the !Kung in Southern Africa, the Ye'kwana in Venezuela, and the residents of the Micronesian atoll Ifaluk,

evidence supports the proposition that by providing nutritional support and services postreproductive women enable their daughters to raise more offspring, thereby contributing to their daughters' reproductive success and their own inclusive fitness (Biesele and Howell, 1981; Hames, 1988; Turke, 1988; Hawkes et al., 1989). However, hardworking grandmothers, like Hadza women aged 45–60 years, who bring in approximately 1,000 more calories on a daily basis than the average person consumes, appear to be exceptional (Kaplan et al., 2000). Among the Ache and Hiwi, foragers in Paraguay and Venezuela, respectively, women, never produce surplus calories (Kaplan et al., 2000). In addition, even Hadza grandmothers provide less than 6% of the average daily protein intake.

Nutritional support does not constitute the sole contribution of postmenopausal women to their daughters' reproductive success in traditional societies, and it is a mistake to assume that investigators like Kaplan et al. (2000) discount the importance of postreproductive females on the basis of their caloric contributions. Grandmothers are frequent providers of childcare and shelter (Draper, 1976; Biesele and Howell, 1981; Sharp, 1981; Hames, 1988; Blurton Jones et al., 1989). Older females also acquire authority and are eligible for special status (Brown, 1985). 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, organizing food preparation and the distribution and delegating and assigning subsistence tasks to younger women (Brown, 1985).

This administrative role has the potential fitness-enhancing value of "nondepreciable" care, which benefits all present grandchildren simultaneously and at minimal incremental cost per child (Borgerhoff Mulder, 1992). In this context, it is easy to appreciate that the interests of grandmothers and mothers do not always coincide and that it is reasonable to expect grandmothers to invest more in the kinds of assistance that benefit many grandchildren equally when socioecological conditions permit.

Finally, although Turke (1988) has shown that on Ifaluk there is a significant positive correlation between the number of offspring

an individual can produce and the number of living parents that individual still has, a favorable trade-off between prolonged fertility and increased grandparental investment has yet to be documented empirically as a result of the sorts of quantitative investigation described above. The trade-offs involving nondepreciable care are especially difficult to quantify. Still, Turke (1988:185) reminds us that "parents should not have evolved postreproductive lifespans if throughout human evolutionary history elderly parents generally have been net service and resource drains on their offspring."

Mathematical models

Desire to quantify the fitness trade-off implied by the original grandmother hypothesis stimulated the mathematical models of Hill and Hurtado (1991, 1996) and Rogers (1993). The models actually deal with the maintenance of menopause, not its origin. None of these models were able to unequivocally account for the maintenance of menopause as a female life history trait. These models are fundamentally about grandmothering, in that enhancing the fertility of grown daughters results in more grandchildren; they are based on Hamilton's (1964) rule [$br > c$], according to which the cost in terms of foregone fertility must be smaller than the benefit of grandmothering to the recipients divided by the coefficient of relationship.

To test whether the inclusive fitness benefits of reproductive senescence are sufficient for the maintenance of menopause, Hill and Hurtado (1991, 1996) created a demographic model that allowed them to predict when women should cease reproduction in order to maximize fitness by diverting time and energy to increasing the reproductive success of close junior kin. Using Ache demographic data to estimate the effect of maternal presence on the fertility of adult sons and daughters and the survivorship of grandchildren, Hill and Hurtado found no support for the grandmother hypothesis, and only qualified support for a mother effect.[1]

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[1]The grandmother effect calculates the benefit in terms of the increased number of grandchildren who survive to age 5 or 10, when the grandmother survives these intervals, with the assumption that she provides assistance. The mother effect measures the number of additional children that an Ache son or daughter can expect if his or her mother survives to the end of a given 5-year interval.

Also considering primarily the effects of maternal care on fertility of offspring and survival of grandoffspring, Rogers (1993) formulated two different models to ascertain whether the benefits of menopause are sufficient to retain a hypothetical allele for menopause. Using demographic data from the 1906 population of Taiwan (Hamilton, 1966), Rogers found that death in childbirth [Model I] cannot account for menopause. Model II, which assumes menopause facilitates maternal care, could not be rejected. However, Rogers (1993) suggests that the assumed values of the "effectiveness coefficients" are unrealistically high and that more realistic estimates of how much assistance a woman can provide might reduce the benefit side of the inequality by a factor of 10.

Male philopatry in the hominine line

The presence of maternal grandmothers had to have been very widespread for some considerable time during human evolution for the grandmother hypotheses to account for menopause and/or the postreproductive lifespan. Thus the grandmother hypotheses require a history of female philopatry and male dispersal in the hominine line. Most extant foraging societies are patrilocal (Kano, 1997; White and Churchill, 1997). Only 17.3% [$n = 185$] of modern hunter-gatherer societies have a consistent pattern of male transfer (White and Churchill, 1997; data from Murdock, 1967), implying that in the vast majority of these societies grandmothers and senior females are not generally available to help junior female kin. Citing Ember (1978), Hawkes et al. (1998:1338) argue that among hunter-gatherers "the tendency toward matrilocality increases with women's relative contribution to subsistence and (separately) with increased dependence on gathering" [parentheses theirs]. If this is the case, the fact that men provide more food energy per day than women in 80% of the foraging societies for which there are quantitative data [$n = 10$] (Kaplan et al., 2000) suggests that matrilocality would be present in less than 20% of foraging societies. Men also provide the vast majority of protein, primarily through hunting. There is a good deal of evidence of meat eating over the past 2.5 million years, and

although direct archeological evidence is still lacking, *H. erectus* was probably obtaining meat through hunting by the time of the species' dispersal to Eurasia 1 mya (Isaac, 1978; Hill, 1982; Potts, 1984; Bunn and Kroll, 1986; Shipman, 1986; Klein, 1989; de-Heinselin et al., 1999).[2] Given that hunting tends to be associated with male philopatry, it is unlikely that female philopatry was the ancestral trait. Kano (1997) also reminds us that both chimpanzees and bonobos, our closest pongid relatives, are patrilocal and that none of the other pongids are matrilocal.[3] The preponderance of evidence suggests that male philopatry and female dispersal were the ancestral traits.

Role of males and older siblilngs

Helping adult daughters and their children is not exclusive to older females. The grandmother hypotheses ignore the role of males and, to a lesser extent, that of older siblings. The important role of males throughout human evolution should be clear. This applies to older males as well as to young males. In general, older males are probably more useful than older females as providers of nutritional resources and status. Among the Ache, Hiwi, and Hadza, men provide large surpluses of calories well into their sixties (Kaplan et al., 2000). Hadza men aged 15–60 years produce 8,000 calories per day and 96% of the average daily protein intake. Older siblings provide childcare and help with other domestic chores (Hames, 1988; Hill and Kaplan, 1988). They may also provide certain kinds of foods to younger siblings, despite the fact that hunter-gatherer juveniles are energetically dependent until they reach sexual maturity.

Summary: problems common to both grandmother hypotheses

Cross-population qualitative and quantitative evidence does not support the notion that helpful grandmothers are the subsidizers of human reproduction. Empirical investigations in foraging societies suggest that in some cases grandmothers contribute to offspring fitness. However, these studies do not demonstrate that grandmothering maintains menopause, and mathematical models using real data have not been successful in showing that menopause is maintained through inclusive fitness. This is in part because of the difficulty of showing that menopause and the postreproductive lifespan afford an advantage when all females will have it if they live long enough. The main problem with the old grandmother hypothesis is that it presupposes menopause—at least, as it is investigated in foraging societies, like the Hadza. Hadza grandmothers are not "choosing" between having another baby and helping daughters. They already have menopause.

In contrast to the supposed socioecological homogeneity when menopause and/or the postreproductive lifespan first arose, one should not expect to find helpful grandmothers now in all foraging or horticultural societies, where socioenvironmental conditions vary and change over time. The kind of help that grandmothers give and the importance of the help vary with local factors, including the availability of other helpers and the feasibility and necessity of grandmothers' help. For instance, among the Ache shared bounty of men's hunting provides most of the food for the band. This fact is probably responsible for the reality that Ache grandmothers are generally less helpful and less essential to daughters' fitness (Hill and Kaplan, 1988). As a corollary, it should be expected that children's contribution to their own nutrition will vary. Hawkes et al. (1998:1338) say as much when they write, "[S]tudies of modern hunter-gatherers indicate wide variation in ages at which children begin to forage, even in broadly similar ecological conditions."

With grandmothers' help varying in importance and kind according to local conditions, one would also expect to find that the median age of menopause increases as socioecological conditions improve over time, if helpful grandmothers are the reason for menopause and/or the postreproductive lifespan. Yet, there is no upward secular trend over the past 150 years, a period during which we know tremendous improvements in living conditions have occurred (Gray, 1976; Flint, 1978, 1997; McKinlay et al., 1985). Indeed, it appears that the age of menopause has not increased over the past

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[2]Recently published evidence of the smaller-brained *H. ergaster* in the Republic of Georgia 1.7 mya may imply increased reliance on animal protein, but hunting has not been suggested (Gabunia et al., 2000).

[3]Dispersal patterns may not be as rigid as once assumed, particularly for chimpanzees at Gombe, where 50% of females returned to their natal groups (Pusey et al., 1997). In nonprovisioned populations, however, virtually 100% of females leave their native groups permanently.

1,000 years (Amundsen and Diers, 1970, 1973; Post, 1971).[4]

Further, there is no reason that mother–offspring foodsharing should benefit only daughters (Gurven and Hill, 1997). It is equally possible that the female postreproductive lifespan evolved because mothers gained fitness through helping grown sons, as some have suggested based on observation of the supportive role of pygmy chimpanzee paternal grandmothers (Wrangham, 1996; White and Churchill, 1997).[5] Moreover, grandfathers may be just as helpful as grandmothers (Gurven and Hill, 1997). Finally, it does not require hardworking grandmothers to produce a postreproductive lifespan in females. If the prolongation of fertility is constrained, only selection for longer lifespans is necessary. The reason for such selection could be anything, e.g., the value of longevous males, as Marlowe (2000) hypothesizes. The fact that senescent mortality may have been steeper in paleopopulations (Gage, 1988; Gage et al., 1989) suggests that old people of either sex would have been scarce and, as Diamond (1997) proposes, accordingly treasured and revered for the knowledge that they possessed and could pass on.[6]

An anecdotal argument casts further doubt on the grandmother hypothesis. It comes from the reproductive ecology of East African elephants (Douglas-Hamilton and Douglas-Hamilton, 1975; Laws et al., 1975; Moss, 1988). Female elephants live in extended family units, led and protected by a matriarch. Accordingly, females assist young daughters in delivering their first calves and behave in a parental manner toward younger sisters and nieces, while the matriarch will sometimes neglect her own newborn in favor of watching over her extended family (Douglas-Hamilton and Douglas-Hamilton, 1975). Moss (1988) writes of a 58-year-old female at Amboseli giving birth to an apparently healthy calf after an interbirth interval of 9 years. The calf died a few weeks later, which was not surprising, since the survival probability of calves of older females is reduced. What is astonishing is that living in the group with this old female were her daughter, her granddaughter, and her greatgranddaughter. Here is a case of male dispersal and close intergenerational cooperation between females and yet no menopause. Females reproduce, albeit with increasing interbirth intervals and decreasing success, until the end of their lives. It is difficult to overlook the possibility that it is precisely in female-bonded societies that females have no need for a protracted postreproductive lifespan. With the ever-present opportunity to increase one's inclusive fitness, females do not have to avoid metabolically costly late births, even if the offspring's chances of survival are small.

THE NEW GRANDMOTHER HYPOTHESIS

The new grandmother hypothesis shares all of the difficulties discussed in the previous section. In addition it has a number of problems of its own. In this hypothesis, grandmothers are the reason for a whole array of human traits: younger age at weaning, longer childhoods, longer lifespans, and more offspring than their hominoid relatives (Hawkes et al., 1998). These divergent traits are due to a change in diet, specifically, to the availability of weaning foods, which young juveniles cannot provide for themselves but which hardworking grandmothers can provide for them. Hawkes et al. (1998) claim that the critical role of grandmothers in the evolution of human life histories is supported when these exceptional traits are examined in terms of Charnov's (1993) dimensionless "assembly rules" for mammalian life histories.[7]

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[4]The fact is we should not be looking at the present for reasons for the origin or even the maintenance of menopause. When we consider the present, the only phenomenon we should investigate is variation in the age of menopause and the fitness implications of this variation.

[5]White and Churchill (1997) hypothesize that mothers increase sons' reproductive success by increasing access to mates and supporting dominance rank.

[6]Support for the importance of both maternal and paternal grandparents comes from the !Kung, a bilateral society with strong relationships between affines. According to Lee (1979:82), if parents and married offspring live far apart, they will usually exchange yearly month-long visits. Sometimes the entire family of the groom takes up residence with the bride's family. Lee's data on the amounts of subsistence work done by visitors do not suggest that grandparents are traveling to subsidize directly the energy requirements of grand offspring, although care of grandchildren and assistance in their socialization are important results of these visits (Shostak, 1983; Biesele and Howell, 1985). Perhaps more important than any kind of day-to-day assistance, grandparents are simply maintaining the relationships between affines which provide a valuable safety net for themselves and their offspring in times of scarcity and local strife.

[7]In order to understand and evaluate the arguments that Hawkes et al. (1998) make, the reader needs some familiarity with Charnov's Model (1993) (see also Charnov, 1991; Charnov and Berrigan, 1991, 1993). The following is a brief and simplified

The evolution of human longevity was surely a complex dynamic process, involving feedback between instantaneous adult and infant mortalities, size at weaning, age at maturity, annual fertility, hominid physioanatomy, and paleoecology. Still, it is useful to try to understand how the increase in longevity might have begun and how other life history traits interacted to achieve this increase. However, the assertion that all the distinctive features of human life history "could be systematic adjustments on the primate pattern that follow from grandmothering" makes several problematic assumptions (Hawkes et al., 1998:1338). The new grandmother hypothesis also has inconsistencies and omissions.

Troubling points with the new grandmother hypothesis

A number of arguments and assumptions made by Hawkes et al. (1997, 1998) are evaluated here. Many of these points have also been discussed by others.

(1) Long lifespans, early weaning, late age at maturity, and high fertility in humans are due to grandmothering.
(2) Longer childhoods permit development of larger brains.
(3) Postreproductive grandmothers probably existed in paleopopulations.
(4) There is wide variation among hunter-gatherers in the age at which juveniles begin to forage.
(5) The grandmother hypothesis "avoids problematic assumptions about men's foraging goals" (Hawkes et al., 1998: 1338).

Long lifespans, early weaning, late age at maturity due to grandmothers. Although the economic contribution of hardworking postmenopausal Hadza women is consistent with long lifespan, late maturity, small size at weaning, and high fertility, grandmothering is not necessarily the reason for these traits.

Longevity. The relationship between adult instantaneous mortality rate [M], average adult lifespan [$1/M$], oldest observed age [T_{max}], and maximum potential lifespan [MPL] must be considered. M is the probability of dying at any given time during adulthood. It is determined by $1/M$, which in turn is determined by T_{max}.[8] All of these related quantities, according to Charnov (1993), are determined by the environment. In contrast, MPL is determined by the biology of an organism and is species-specific (Cutler, 1976). Thus, MPL and T_{max} appear to be quite different life history traits. As part of the environment, helpful grandmothers can decrease M and increase $1/M$ and T_{max}. How grandmothers can affect MPL is not indicated.

It is probable that extrinsic environmental sources of mortality were more important during most of the evolution of human longevity than is presently the case in many traditional societies. This would have made adult mortality rates higher and the increase in senescent mortality more rapid as a function of age (Gage, 1987; Gage et al., 1988; cf. Konigsberg and Frankenberg, 1992), but both rates would also have been more amenable to improvement through external influences, such as the assistance of kin. However, it can be questioned how M, $1/M$, and T_{max} can be wholly determined by the environment or taxonomically independent. Many of the physiological factors that determine MPL must affect M, $1/M$, and T_{max} to some degree. For example, brain size correlates with MPL, either because the brain is the "organ of longevity" biologically or neurochemically governing the rate of aging, or because brain size co-evolved with MPL due to the dynamic interaction between intelligence and longevity (Sacher, 1959; Cutler, 1975a,b, 1976). Either way, brain size and consequent cognitive abilities must play a role in determining $1/M$ and T_{max}.

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description of the model. Charnov has identified critical trade-offs in mammalian life histories, from the approximate invariance of the products of particular traits, thereby revealing the basic rules responsible for the fact that values of traits are similar for organisms at some taxonomic level and different from those of organisms outside the taxonomic boundary. Increases in age at maturity allow for a longer growth period and greater adult size, which creates greater production potential. Realization of this potential depends on adult longevity. Therefore adult instantaneous mortality [M] must decrease if age at maturity is to increase. Hence the period of independent growth from weaning to age at maturity [α], and instantaneous adult mortality rate [M] vary inversely, such that αM is approximately invariant. Size at weaning relative to adult size [δ] scales isometrically, whereas production rates increase more slowly with adult size than weaning size. To continue producing offspring of the same relative size at weaning, larger mothers reduce annual fecundity [b]. b also varies inversely with α, and αb is another approximate invariant.

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[8]T_{max} is computed according to Charnov's (1993) formula: $1/M = 0.4T_{max} - 0.1$.

TABLE 1. *Average values for selected life history variables given by Hawkes et al. (1998)[a]*

	AW	AM	α	$1/M$	T_{max}	αM	δ	b	αb
Orangutans	6.0	14.3	8.3	17.9	44.5	0.46	0.28	0.063	0.52
Gorillas	3.0	9.3	6.3	13.9	34.5	0.45	0.21	0.126	0.79
Chimpanzees	4.8	13.0	8.2	17.9	44.5	0.46	0.27	0.087	0.70
Humans	2.8	17.3	14.5	32.9	82.3	0.44	0.21	0.142	2.05

[a]AW = age at weaning; AM = age at maturity; α = period of independent growth from weaning to age at maturity; $1/M$ = average adult lifespan; T_{max} = maximum observed lifespan [$1/M = 0.4T_{max} - 0.1$]; αM = an invariant; δ = size at weaning relative to size at maturity; b = annual fecundity; αb = another invariant.

Extension of lifespan by grandmothering led to later maturity. Based on Charnov's (1993) model, Hawkes et al. (1998:1337) write, "If human longevity has been extended by grandmothering, then age at maturity [α] should be delayed accordingly."[9] [As does Charnov (1993), Hawkes et al. (1998) interpret and use α both as period of independent growth and age at maturity.] The fact that the period of independent growth [α] is adjusted to the whole lifespan is suggested as evidence that gains from growing longer before reproducing extend through the postreproductive years. This could be, but it does not mean grandmothering is responsible for increases in longevity or α. Since αM is an invariant according to Charnov's (1993) model, the extension of human longevity for whatever reason should lead to an increase in α. All that is shown is that humans fit the allometric relationship. It is equally plausible that grandmothering is the result of increases in M and α. Moreover, even though Charnov draws a causative arrow from M to α, in reality the relationship between M and α may be reversed or reciprocal. White and Churchill (1997) favor the notion that a long lifespan may be a direct consequence of selection for extended childhood: the result of mothers', not grandmothers', increased investment in the survival and viability of their own offspring.

Hawkes et al. (1998) also suggest that if the grandmother hypothesis is correct, than age at maturity and the period of independent growth [α] should be high in humans compared to the great apes, and they are (Table 1). However, this is due to Charnov's (1993) formula, since α/T_{max} is also an approximate invariant (Table 2). Large T_{max} signifies a large α. It has little to do with the grandmother hypothesis itself.

Furthermore, there is a problem with us-

TABLE 2. *Values of life history traits AM and α given by Hawkes et al. (1998) relative to M and T_{max} respectively*

	$AM \cdot M$	αM	AM/T_{max}	α/T_{max}
Orangutans	0.79	0.46	0.32	0.19
Gorillas	0.66	0.45	0.27	0.18
Chimpanzees	0.73	0.46	0.29	0.18
Humans	0.52	0.44	0.21	0.18

ing α as a comparative measure across species. Although α and age at maturity may be used interchangeably in humans, because humans are weaned so young, for orangutans, gorillas, and chimpanzees weaning is late relative to age at maturity, making their α relatively small compared to age at maturity. However, if α really signifies the period of independent growth, it is surely not correct to assume as with other species that α equals age at maturity minus age at weaning for humans. Humans have a period of childhood dependence, nutritional and otherwise, between weaning and the juvenile period (Bogin and Smith, 1996), whereas for other hominoid species α really represents a period of independent growth. According to Bogin and Smith (1996), independent growth in humans does not begin until approximately 7 years of age. In fact, hunter-gatherer children begin to acquire food at different ages and in different amounts depending on availability, difficulty of acquisition, and dangers involved.[10] For example, by age 5, Hadza children can supply up to 50% of their daily nutritional requirements in some seasons (O'Connell et al., 1999). Ache toddlers are quite proficient at collecting small fruits from the ground, but acquisition of more important foods does not begin until after age 10 (Kaplan et al., 2000). Hunter-gatherer children remain energetically dependent on older individuals

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[9]I assume that "longevity" refers to T_{max}.

[10]There are also large seasonal variations in children's daily contribution to their own nutritional requirements, because they tend to do best at collecting fruits (Kaplan et al., 1999).

to some extent until their late teens (Kaplan et al., 2000). Hence even when juveniles are relatively proficient foragers, a true α would be very small for humans.

If human offspring are being nutritionally subsidized after weaning, which allows both for earlier weaning and prolonged nutritional dependence, regardless of who is doing the provisioning, the more correct life history variable to compare across species would be age at maturity [AM], not α. While the quantities αM and $\alpha/T_{\max}$ are invariant for the four species, AM $\cdot M$ and AM/$T_{\max}$ vary considerably (Table 2). Moreover, for humans the quantities AM $\cdot M$ and AM/$T_{\max}$ are low compared to the great apes, although the difference is not significant.

High fertility and late age at maturity due to grandmothers. If the new grandmother hypothesis is correct, Hawkes et al. (1998:1338) predict that "[g]randmother's contribution must increase daughters' annual fecundity", and human annual fecundity [b] should be higher than that of other hominoid species. The data given by the authors do not show humans with meaningfully higher b than all of the other great apes (Table 1). The annual birthrate in human hunter-gatherer populations, though higher than that of orangutans and chimpanzees, is similar to that of gorillas. The claim that, compared to the great apes, human annual fecundity is uniquely high is as arguable as why this should be attributable to grandmothering.

The higher human annual fertility of humans compared to chimpanzees and orangutans, for example, could be a function of greater foraging skill resulting from increased cognitive and communicative abilities following encephalization. However, it is not known how long human fertility has been high compared to chimpanzees. There is presently no evidence that high fertility is an ancestral trait. Cross-population variation in total fertility rates, nutritional status and mortality rates would seem to suggest that big increases in fertility are to be the result of relatively recent changes in diet, stemming from the switch from hunting and gathering to agriculture and animal husbandry (Bongaarts, 1980; Wood, 1994a; Hill and Hurtado, 1996).

Another life history invariant in Charnov's (1993) model is the product of αb; annual fecundity rate b should *decrease* as the age of maturity α increases (Table 1). Com-

pared to αM, this "invariant" does not seem to work very well for the four species, especially humans. For human females the product of the period of independent growth and annual fecundity [αb] is more than double that of the great apes. However, to argue, as Hawkes et al. (1998:1338) do, that this happens because αb "incorporates the production of both mothers and grandmothers" through nutritional subsidization of earlier weaning or accelerated infant growth resulting in earlier weaning seems to stretch the point. As argued above, the "true" period of independent growth is probably very different for humans than the 14.5 years cited by Hawkes et al. (1998). Depending on what age one chooses for nutritional independence, α could be comparatively very much smaller for humans or roughly the same for all four species.

Although human infants are weaned comparatively early relative to age at maturity, size at weaning relative to adult size [δ] is similar for all four species. Indeed, δ is the same for humans and gorillas.[11] However, early weaning *and* "normal" size at weaning do not necessarily imply grandmothering. Both could be the result of the excessive metabolic cost of rapid postnatal brain growth and ecological changes which provided better, but difficult-to-find-and-process, weaning foods. Both early AW and normal δ could also be the result of mothers redirecting maternal investment from costly lactation to greater foraging and food processing effort, or enlisting the help of anyone available, including older siblings or males. Bogin and Smith (1996) suggest that the introduction of a postweaning period of nutritional dependence reduced the reproductive strain on early *Homo* females, allowing for prolonged provisioning necessary to sustain costly postnatal brain growth while permitting shorter interbirth intervals. The problem of how to feed weaned children was solved by spreading the responsibility (Bogin and Smith, 1996). αb could be comparatively high for humans because it incorporates a change in maternal investment strategy, alone or in conjunction with the help of adult males (Gurven and Hill, 1998; Kano, 1998). Food sharing may

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[11]An interesting question is why do gorillas and humans have similar annual fecundity, as well as similar age and relative size [δ] at weaning.

be ancestral, but grandmothers doing it may be recent (Kano, 1998).

Longer childhoods permitted larger brains

Hawkes et al. (1998:1338) draw "the causal arrow from long childhood to learning". The more logical and common view is that development of larger brains and the need for more learning time *required* an extended childhood, not that longer childhood permitted development of larger brains. Arguing that adult lifespan, not brain size and learning time, predicts age at maturity, Hawkes et al. (1998) assert that late age at maturity relative to adult size occurs in animals with small brains and that juvenile periods in nonhuman primates are longer than needed to acquire survival skills (see Charnov and Berrigan, 1991, for examples). Other investigators do not agree (Bogin and Smith, 1996; Kaplan et al., 2000). According to Bogin and Smith (1996), the addition of an adolescent period before full sexual maturity affords an opportunity for young males and females to practice complex social skills, which for females pays off later in the form of increased infant survivorship.

Age structures in paleopopulations and the likelihood of postreproductive grandmothers

Hawkes et al. (1998) maintain that life history changes initiated by grandmothering should be marked by evidence for increases in age at maturity and the existence of a postmenopausal lifespan. Three possible points in time during human evolution are suggested for the occurrence of these changes. These alternatives are discussed only as they relate to delayed maturity, not to increases in longevity. The three points are 1.8 mya (*H. erectus*), 600 kya (early archaic *H. sapiens*), 50 kya (anatomically modern *H. sapiens*). With the new grandmother hypothesis, alternative times are unnecessary. Because termination of fertility is not about stopping early in the new hypothesis, it implies that the human female postreproductive lifespan arose when maximum lifespan exceeded the current age of menopause of about 50 years.

Hawkes and colleagues have since identified the relevant changes in hominid life history with the appearance of *H. erectus* (O'Connell et al., 1999). "The appearance of this form is marked by shifts in brain size, dental eruption schedules and adult body weight, all read to indicate increased lon-

gevity and delayed maturity" (O'Connell et al., 1999:468). Recent studies agree that maximum lifespan has exceeded 50 years since early *H. erectus* (Bogin and Smith, 1996; Hammer and Foley, 1996), which could indicate that menopause is 1.8 myo.[12] In contrast, early *H. erectus* [*H. ergaster*] is not generally associated with delayed maturity (Smith, 1991, 1993; Bogin and Smith, 1996; cf. Clegg and Aiello, 1999). The first appearance of delayed maturity is estimated at 1.5 mya (Smith, 1993).

Whether or not menopause is that old, the determining role of postreproductive grandmothers in the evolution of human longevity remains questionable because this would imply that females have lived a number of years past menopause with some frequency for a very long time.[13] Age structures in early societies do not support either of the grandmother hypotheses. In contrast to many extant foraging societies, such as the !Kung, Hadza, and Ache, where a large number of women live well beyond 50 years even if life expectancy at birth was less than 50 years, it is unlikely that many women survived to become postmenopausal even as recently as 50,000 years ago. Evidence for this iconoclastic suggestion comes from comparison of population parameters of two paleopopulations (a Late Woodland North American population, 800 AD; and a Christian Nubian population, 1100 AD) and a Coale and Demeny (1988) Western model life table with *similar life expectancy at birth* (Gage, 1988; Gage et al., 1989). Infant mortality is lower in paleopopulations, but the rate of decline in immature mortality is slower, adult mortality is greater, and rate of increase in senescent mortality is steeper. For example, expectation of life from age 15 (sexes combined) was 18.25 years for the Late Woodland population and 16.83 years for the Nubian population (Gage, 1988:436). For the Late Woodland population, the probabilities of dying between ages 45–49 and 50–54 were 0.865 and 0.988, respectively; for the Nubian population these probabilities were 0.778 and 0.945, respectively (Gage, 1998:436). These results sug-

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[12]Eventually conclusive evidence for the evolution of menopause may be recoverable from changes in bone mass and tubular bone associated with estrogen withdrawal in females fossils (Bogin and Smith, 1996).
[13]If menopause is about mothering and/or the age of onset was earlier, it could be that old.

gest that few women survived to become postmenopausal even as late as 1,000 years ago, and when they did, postmenopausal lifespans were short (Gage, 1988). Life expectancies in extant foraging populations are considerably above the range of pre-industrial hunter-gatherer populations, falling in the range of contemporary developing populations (Gage et al., 1989). For example, for the !Kung expectation of life from age 15 is 42 years.

Wide variation in juvenile foraging age

Hawkes et al. (1998:1338) state that among hunter-gatherers there is a wide variation in the age at which juveniles begin to forage and that there is no indication that large differences in time spent "practicing" affect adult performance. This supports the "rule" that age at maturity is determined by adult mortality, not by the need to provide sufficient time for juveniles to learn survival skills. Data on the daily energy production of Ache, Hiwi, and Hadza men and women do not support the generalized notion that the children of human foragers have more time than necessary to learn all the skills they will need as adults (Kaplan et al., 2000). For the Ache and Hiwi, except for gathering small fruits, food acquisition returns do not peak until well into adulthood. The Hadza appear to be an exception with foraging skills, including hunting and extraction production, reaching a peak before age 20 in both sexes.

The principal tenet of the new grandmother hypothesis is that an increase in offspring provisioning, specifically protracted food sharing of difficult-to-find-and-process food, may be responsible for the postreproductive lifespan in human females and an array of other life history traits, all of which are "systematic adjustments on the primate pattern that follow from grandmothering" (Hawkes et al., 1998:1338). However, to argue that there is a wide variation in the age at which children begin to forage undermines the whole point of using data from any extant foraging society to support the grandmother hypothesis, especially the Hadza where grandmothers are so hardworking. Forgetting for a moment the questions regarding the interpretation of the values of various human life history traits, it seems that since the values of these traits are species-specific, they must be a response to universal conditions at some point in our

past. If children in extant foraging societies can feed themselves at different ages, the need for helpful grandmothers also varies. It can probably be assumed that the role of grandmothers has varied over the past, as well. This being the case, is should not be assumed that human longevity and the female postreproductive lifespan evolved from grandmothers' provisioning of adult daughters and grandchildren. The necessity for mothers to engage in longer provisioning, which they could accomplish with the help of males and adolescents, would have been sufficient to change the nonhuman primate pattern. Moreover, if human societies are used as models, groups in which children's post-weaning nutritional dependence requires extensive input from grandmothers should not be used. Otherwise, some human groups where grandmothers are not so crucial have "systematic adjustments" that they do not really need!

Grandmother hypothesis avoids problematic assumptions about males

The grandmother hypothesis "avoids problematic assumptions about men's foraging goals" (Hawkes et al., 1998:1338). This is not an acceptable argument. A hypothesis about the origin of the female postreproductive lifespan cannot fail to discuss the role of males, or siblings for that matter. If Hawkes et al. (1998) believe males make a smaller contribution than females to the energetics of human reproduction, reasons supporting this view need to be provided.

Summary: new grandmother hypothesis

In summary, the new grandmother hypothesis has a variety of problems. A number emerge from the interpretations Hawkes et al. (1998) make of the fit between Charnov's model and their data. Neither the model nor the data are questioned; rather the inferences drawn can be questioned. (1) The notion that M, $1/M$, and T_{max} are determined entirely by the environment is incomplete. These life history traits must also be influenced by phylogenetic and physiological factors, as well. (2) Length of lifespan need not necessarily determine the period of independent growth; it could be the other way around. (3) Compared to chimpanzees, gorillas, and orangutans, age at maturity is not late relative to average adult lifespan or maximum observed lifespan. In fact, according to the data of Hawkes et al. (1998:1337),

human AM is comparatively early (see AM · M and AM/$T_{\max}$, Table 2). (4) The fact that αM is invariant for all four species does not mean that age at maturity is adjusted to the whole lifespan in humans as in other primates. For human females it is not a period of independent growth and, therefore, should not be used *mutatis mutandis* for age at maturity. AM · M shows clearly that human children are indeed different from the other great apes; they mature early relative to average adult lifespan. (5) Humans do not have higher annual fecundity than all of the large-bodied apes. According to the data of Hawkes et al. (1998), b for human females is similar to that of gorillas. Moreover, it is not known how long b has been high relative to chimpanzees, our closest relatives. (6) Large α in humans, which is due to an extremely early age at weaning, is responsible for the fact that αb is so large in humans. (7) Relative size at weaning [δ] is similar in humans, chimpanzees, gorillas, and orangutans, but even if δ were comparatively small for humans, there is not sufficient evidence to conclude that helpful grandmothers are responsible. (8) Most researchers are not persuaded that longer childhoods led to larger brains; the reality is probably the reverse.

Several issues not related to Charnov's (1993) model also need to be considered. (1) According to the new grandmother hypothesis the female postreproductive lifespan arose when lifespan exceeded 50 years. Paleoanthropological evidence suggests that this occurred approximately 1.5 mya. However, a causal relationship between increased lifespan and grandmothering is belied by age structures and life expectancies in paleopopulations, which suggest that very few women survived to age 50. (2) The new grandmother hypothesis avoids any discussion of the role of males in increasing human longevity. (3) Lastly, the Hadza, who are at the extreme end of the spectrum when it comes to the nutritional contribution of postreproductive women, are probably an inappropriate model to illustrate that hardworking grandmothers are responsible for the extraordinarily long lifespans of humans.

DISCUSSION

Hawkes et al. (1998) assert that, in their scenario, menopause is definitely not premature reproductive senescence. If repro-

ductive senescence begins before the decline of other somatic processes, it can always be considered premature. However, calling reproductive senescence in human females premature is not merely a matter of definition. If we compare humans to other mammals, it may look as though long lifespan, not menopause, is the derived trait, but the acceleration in follicular atresia that occurs before age 40 in human females must be considered (Richardson et al., 1987; Richardson and Nelson, 1990; Faddy et al., 1992; Gougeon et al., 1994; Faddy and Gosden, 1996; cf. Leidy et al., 1998). Human females are born with enough oocytes to last 70 years if the rate of atresia remains constant; instead females run out of oocytes at about age 50 years (Faddy et al., 1992; Gosden and Faddy, 1994). Why the rate of atresia accelerates in humans is still poorly understood, and although the follicular depletion system seems to be "highly conserved" between humans and chimpanzees (Wood et al., 1999), it is unknown whether chimpanzee females experience this acceleration. The acceleration of follicular loss could be the result of selection for efficient early reproduction and/or factors constraining the prolongation of the female reproductive lifespan (Peccei, 2001). Estimated heritabilities for the age of menopause vary between 0.4 and 0.6 (Snieder et al., 1998; Peccei, 1999), and the lack of a discernible upward trend in the age of menopause provides further evidence that something more than the prolongation of human lifespans through inclusive fitness is required to explain menopause.

Finally, to emphasize that menopause is "not about stopping early" presents a peculiar and particular problem for the new grandmother hypothesis. In a population where reproductive capacity and lifespan were originally in synchrony, selection for a postreproductive lifespan due to grandmothering implies that some females simply outlived their fertility. In this scenario, reproductive cessation is, in effect, decoupled from increased lifespan. This somehow makes the special role of postreproductive females in selection for longer lifespan much less compelling.

Mothering versus grandmothering

In the altriciality–lifespan hypothesis, it is postulated that *if* menopause is an ancestral trait, it must have involved selection for premature reproductive senescence in re-

sponse to the increased altriciality of hominid infants (Peccei, 1995a,b).[14] As cranial capacities increased, the challenges facing our hominine ancestors included, along with the risk of the birth process itself, high metabolic costs of gestation and lactation, and a postulated likelihood of increasing maternal depletion as a function of parity and age (Jelliffe and Maddocks, 1964; Martin, 1983; Merchant and Martorell, 1988; Johnson et al., 1990; Merchant et al., 1990a,b; Tracer, 1991; Stanley, 1992; Wood, 1994b, 1990; Smith and Tompkins, 1995; Ruff et al., 1997; Wood and Collard, 1999).

Because of secondary altriciality and resultant prolonged dependence of human infants, mothers must care for more than one dependent offspring at a time or risk losing precious reproductive opportunities. The expedience of overlapping childcare further increased the relative cost of each successive offspring. With prolonged fertility negatively affecting maternal well-being and maternal mortality negatively affecting juvenile survivorship, it is reasonable to posit that premature reproductive senescence and the postreproductive lifespan in human females evolved to ensure prolonged investment in offspring who were already born. Only females who reproduced early and relatively rapidly had an advantage. Perhaps males and adolescents subsidized the energetics of reproduction by providing a nutritional surplus which made up for the chronic energy deficit of reproductive-aged females and juveniles. At any rate, the response involved *mothering,* not grandmothering. The hypothesis also suggests that premature reproductive senescence and mothering played a role in increasing maximum potential lifespans and life expectancies by permitting further cranial expansion and by increasing offspring viability (Peccei, 1995a,b). The fitness garnered in the female postreproductive lifespan selected for genes of longer-lived, presumably heartier individuals. Clearly, the inclusive fitness garnered by longevous males, who increased

the viability of offspring through provisioning, lending status, and sharing stored knowledge, would also have selected for longer lifespans.

There are two evolutionary theories for aging: mutation–selection balance and intertemporal trade-offs in reproductive effort (Medawar, 1952; Williams, 1957). According to the first, the strength of selection decreases with age because fewer individuals remain, allowing deleterious mutations to accumulate in later life. According to the second, high fertility at younger ages results in rapid aging, whereas slower aging usually implies reduced fertility at younger ages. Hawkes et al. (1998) suggest that regular mother–child provisioning perturbs the standard equilibrium in humans, permitting both high fertility early in life and slower aging, and thus grandmothering works either way to slow aging; so would mothering.

It is certainly not clear that postmenopausal lifespan evolved because of inclusive fitness gained from provisioning grandchildren. According to Hamilton's (1964) rule, the cost in terms of foregone fertility must be smaller than the benefit of grandmothering divided by four, or the benefit of mothering divided by 2 [$b/4 > c$ or $b/2 > c,$ respectively]. For grandmothering to be more profitable than mothering, the costs of grandmothers would have to be less than half those of mothers. At present, the costs of grandmothers would certainly be smaller than those of mothers due to declining fertility. However, it may not be reasonable to assume that these costs would differ by more than a factor of 2 in a hypothetical population where premature reproductive senescence did not previously exist. In real life, as well as in modeling menopause, there is greater gain in enhancing the survival and increasing the potential fertility of one's own offspring (Peccei, 1995a,b; Peccei and Peccei, unpublished, 1995). Moreover, even if menopause is sustained by grandmothering, it is unlikely that grandmothering triggered menopause, given the relatively recent improvement in life expectancies and the difficulty of increasing grandoffspring fitness enough to warrant premature reproductive senescence. More likely, mothering was instrumental in the origin of menopause (Peccei, 1995a,b; White and Churchill, 1997). In addition, it could be argued that grandmothering requires a long

[14]Originally it was also postulated that *if* menopause is a hominine trait, it would have occurred at an earlier age because estimated maximum potential lifespans for early *Homo* species were lower than the current age of menopause. In view of recent increased estimates of hominine maximum potential lifespans (Hammer and Foley, 1996; Bogin and Smith, 1996), it is unnecessary to hypothesize an earlier age of onset, unless one assumes that no individuals lived out the maximum potential lifespan.

postreproductive lifespan, whereas mothering does not, and therefore a postreproductive lifespan via mothering was easier to establish.

Another way to look at the grandmothering versus mothering debate is to ask who would have benefited from menopause first: mothers with still-dependent offspring, or mothers with reproductive-aged daughters? In species with male philopatry, the answer is always mothers with still-dependent offspring because there would be no mature daughters present. In species where females remain in their natal groups after maturity, the answer is both, because an older female is probably both a mother and a grandmother at the same time. Now, imagine that the benefit could come either from mothering or grandmothering; which is more likely to cause menopause to evolve? From a logical perspective, the answer is clear: when the first female experienced premature reproductive cessation, she would have had her own still-dependent offspring to care for.

Packer et al. (1999) also indicate that it is mothers with young offspring who benefit the most from menopause. According to Packer et al. (1999), lions and olive baboons are good candidates for menopause as an adaptation for grandmothering because they remain with female kin after maturity and exhibit kin-directed cooperative behavior. Female lions even nurse daughters' cubs. However, once they become postreproductive, female lions do not engage in any grandmothering; they do not increase the fertility of offspring or survivorship of grandoffspring. The suggested reason for the absence of postreproductive investment in both lions and olive baboons is that maternal mortality affects only the youngest offspring. Juvenile survivorship is not affected by mother's death or future reproduction.

In addition, neither species experiences breeding costs, i.e., there is no increase in mortality because of reproduction. Indeed, the age-specific fertility of most female mammals shows a pattern of relative constancy over a long period of time, followed by a rapid decline in fertility (Laws et al., 1975; Paul et al., 1993; Packer et al., 1998). With a rapid decline in age-specific fertility accompanied by generalized senescence, maintaining reproductive capacity in older females offers little additional fitness

(Packer et al., 1998), whereas successfully bringing last offspring through infancy does. This speaks for mothering as the reason for a postreproductive lifespan, albeit a very short one, in lions and olive baboons and reinforces the notion that among our ancestors' mothers were the first to benefit from menopause.

Packer et al. (1998) also suggest that the timing of menopause is set by maternal survivorship and the duration of offspring dependency, such that postreproductive lifespan should be longer in species with longer dependency. In addition, increasing reproductive costs should lead to an even greater gap between menopause and expected lifespans in species in which interbirth intervals are shorter than dependency (Packer et al., 1998). This may explain the longer postreproductive lifespan of human females in foraging societies relative to nonhuman primates. Infant dependency also explains why in mammals reproductive cessation is more striking in females than in males, as well as why the sex difference in postreproductive lifespan is so much more pronounced in humans (Packer et al., 1998).

CONCLUSIONS

The evidence for menopause as an adaptation via the old grandmother hypothesis is not compelling, and the acceleration in follicular atresia preceding menopause, which is still thought to be unique to humans, is not consistent with the new grandmother hypothesis, which makes menopause the simple byproduct of increased longevity. Still, menopause could be the by-product of increased longevity (for whatever reason), if female reproductive lifespan could not be prolonged because of existing selection for efficient early reproduction. In the face of increasing lifespans and life expectancies, selection for early fertility becomes a fitness-enhancing adaptation in its own right (Peccei, 2001). There is undoubtedly also an element of adaptation for increased maternal investment in the origin of menopause. Given ecologically-induced changes in diet beginning in the Plio-Pleistocene, the secondary altriciality of hominine infants, low life expectancies in paleopopulations, and the unlikelihood of female philopatry, as well as Hamilton's rule and the logical inference that when fertility ceases a woman still has her own still-dependent offspring to care for, the evidence suggests that the

mother hypothesis works better than the grandmother hypothesis, with selection for early fertility via antagonistic pleiotropy. In this scenario, by permitting prolonged juvenile dependence, menopause allows for cranial expansion, which leads to increases in maximum lifespan. Once a postreproductive lifespan of some length was established, grandmothering could be useful if grandmothers were living and present. As lifespans continued to increase, there may have been an increase in the AOM.

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LITERATURE CITED

Amundsen DW, Diers CJ. 1970. The age of menopause in Classical Greece and Rome. Hum Biol 42:79–86.

Amundsen DW, Diers CJ. 1973. The age of menopause in medieval Europe. Hum Biol 45(4):605–612.

Asfaw B, White T, Lovejoy O, Latimer B, Simpton S, Suwa G. 1999. *Australopithecus garhi:* a new species of early hominid from Ethiopia. Science 284(5414): 629–635.

Avioli LV. 1994. Impact of the menopause on skeletal metabolism and osteoporotic syndromes. Exp Gerontol 29(3/4):391–415.

Biesele M, Howell N. 1981. "The old people give you life: Aging among !Kung hunter-gatherers. In: Amoss PT, Harrell S, editors. Other ways of growing old. Stanford: Stanford University Press. p 77–98.

Blurton Jones NG, Hawkes K, O'Connell JF. 1989. Modelling and measuring costs of children in two foraging societies. In: Standen V, Foley RA, editors. Comparative socioecology. Oxford: Blackwell Scientific Publishing. p 367–390.

Bogin B, Smith BH. 1996. Evolution of the human life cycle. Am J Hum Biol 8:703–716.

Bongaarts J. 1980. Does malnutrition affect fecundity? A summary of evidence. Science 208:564–569.

Borgerhoff Mulder M. 1988. Reproductive decisions. In: Smith EA, Winterhalder B, editors. Evolutionary ecology and human behavior. New York: Aldine de Gruyter. p 339–374.

Brace M, McCauley E. 1997. Oestrogens and psychological well-being. Ann Med 29(4):283–290.

Brown JK. 1985. Introduction. In: Brown JK, Kerns V, editors. In her prime. South Hadley, MA: Bergin & Garvey Publishers, Inc. p 1–11.

Bunn H, Kroll EM. 1986. Systematic butchery by Plio-Pleistocene hominids. Curr Anthropol 27(5):431–452.

Byington RP, Furberg CD, Riley W, Applegate W, Herd A, Herrington D, Hunninglake D, Lowery M. 1999. Effect of estrogen + progestin on progression of carotid atherosclerosis in postmenopausal women with documented heart disease: HERS B-mode substudy. Am Coll Cardiol 33:265A.

Charnov EL. 1991. Evolution of life history variation among female mammals. Proc Natl Acad Sci U S A 88:1134–1137.

Charnov EL. 1993. Life history invariants: some explorations of symmetry in evolutionary ecology. Oxford: Oxford University Press.

Charnov EL, Berrigan D. 1991. Dimensionless numbers and the assembly rules for life histories. Phil Trans R Soc London B 33:241–248.

Charnov EL, Berrigan D. 1993. Why do female primates have such long lifespans and so few babies? or life in the slow lane. Evol Anthropol 1:191–194.

Clegg M, Aiello LC. 1999. A comparison of the Nariokotome *Homo erectus* with juveniles from a modern human population. Am J Phys Anthropol 110:81–93.

Coale AJ, Demeny P. 1966. Regional model life tables and stable populations. Princeton: Princeton University Press.

Colditz GA, Willett WC, Stampfer MJ, Posner B, Speizer FC, Hennekens CH. 1987. Menopause and the risk of coronary heart disease in women. New Engl J Med 316:1105–1110.

Cutler RG. 1975a. Evolution of longevity in primates. J Hum Evol 5:169–202.

Cutler RG. 1975b. Evolution of human longevity and the genetic complexity governing aging rate. Proc Natl Acad Sci U S A 72(11):4664–4668.

Cutler RG. 1976. Nature of aging and life maintenance processes. Interdiscipl Top Gerontol 9:83–133.

deHeinselin J, Clark D, White T, Hart W, Renne P, WoldeGabriel G, Beyene Y, Vrba E. 1999. Environment and behavior of 2.5-million-year-old Bouri Hominids. Science 284(5414):625–629.

Diamond J. 1992. The third chimpanzee: the evolution and future of the human animal. New York: Harper-Collins Publishers.

Diamond J. 1997. Making more by making less: the evolution of female menopause. Why is sex fun? New York: Basic Books. p 103–125.

Douglas-Hamilton I, Douglas-Hamilton O. 1975. Among the elephants. New York: Bantam Books.

Draper P. 1976. Social and economic constraints on child life. In: Lee RB, DeVore I, editors. Kalahari hunter-gatherers. Cambridge: Cambridge University Press.

Ember CR. 1978. Myths about hunter-gatherers. Ethnology 17:439–448.

Faddy MJ, Gosden RG. 1996. A model conforming the decline in follicle numbers to the age of menopause in women. Hum Reprod 11(7):1484–1486.

Faddy MJ, Gosden RG, Gougeon A, Richardson SJ, Nelson JF. 1992. Accelerated disappearance of ovarian follicles in mid-life: implications for forecasting menopause. Hum Reprod 7:1342–1346.

Flint M. 1978. Is there a secular trend in age of menopause? Maturitas 1:133–139.

Flint M. 1997. Secular trends in menopause age. J Psychosom Obstet Gynecol 18(2):65–72.

Foley RA. 1987. Another unique species. New York: Longman Scientific and Technical.

Foley RA, Lee PC. 1991. Ecology and energetics of encephalization in hominid evolution. Phil Trans R Soc London B 334:223–232.

Fretts RC, Schmittdiel J, McLean FH, Usher RH, Goldman MB. 1995. Increased maternal age and the risk of fetal death. New Engl J Med 333(15):953–957.

Gabunia L, Vekua A, Lordkipanidze D, Swisher CC, Ferring R, Justus A, Nioradze M. Tvalchrelidze M, Anton SC, Bosinski G, Joeris O, deLumley MA, Majsuradze G, Mouskhelishvili A. 2000. Earliest Pleistocene hominid cranial remains from Dmanisi, Republic

of Georgia: Taxonomy, geological setting, and age. Science 288:1019–1025.

Gage TB. 1988. Mathematical hazard models of mortality: an alternative to model life tables. Am J Phys Anthropol 76:429–441.

Gage TB, McCullough JM, Weitz CA, Dutt JS, Abelson A. 1989. Demographic studies and human population biology. In: Little MA, Haas JD, editors. Human population biology: a transdisciplinary science. New York: Oxford University Press. p 45–65.

Gaulden ME. 1992. Maternal age effect: the enigma of Down Syndrome and other trisomic conditions. Mutat Res 296:69–88.

Gaulin SJC. 1980. Sexual dimorphism in the human post-reproductive lifespan: possible causes. J Hum Evol 9:227–232.

Gosden RG, Faddy MJ. 1994. Ovarian aging, follicular depletion, and steroidogenesis. Exp Gerontol 29(3/4):265–274.

Gosden RG, Spears N. 1997. Programmed cell death in the reproductive system. Br Med Bull 52(3):644–661.

Gosden RG, Faddy MJ. 1998. Biological bases of premature ovarian failure. Reprod Fertil Dev 10(1):73–78.

Gougeon A, Ecochard R, Thalabard JC. 1994. Age-related changes of the population of human ovarian follicles: increase in the disappearance rate of non-growing and early-growing follicles in aging women. Biol Reprod 50:653–663.

Gray RH. 1976. The menopause—epidemiological and demographic considerations. In: Beard RJ, editor. The menopause: a guide to current research and practice. Baltimore: University Park Press. p 25–40.

Gurven M, Hill K. 1997. Cited by: Hawkes K, O'Connell JF, Blurton Jones NG. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long post-menopausal life spans. Curr Anthropol 38(4):551–577.

Hames RB. 1988. The allocation of parental care among the Ye'kwana. In Betzig L, Borgerhoff Mulder M, Turke P, editors. Human reproductive behavior. Cambridge, UK: Cambridge University Press. p 237–251.

Hamilton WD. 1964. The genetical evolution of social behaviour. I, II. J Theor Biol 7:1–52.

Hamilton WD. 1966. The moulding of senescence by natural selection. J Theor Biol 12:12–45.

Hammer MLA, Foley RA. 1996. Longevity and life history in hominid evolution. Hum Evol 11(1):61–66.

Harman SM, Talbert GB. 1985. Reproductive aging. In: Finch CE, Schneider EL, editors. Handbook of the biology of aging. New York: Van Nostrand Reinhold. p 457–510.

Hawkes K, O'Connell JF, Blurton Jones NG. 1989. Hardworking Hadza grandmothers. In: Standen V, Foley RA, editors. Comparative socioecology. Oxford: Blackwell Scientific Press. p 341–366.

Hawkes K, O'Connell JF, Blurton Jones NG. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long post-menopausal life spans. Curr Anthropol 38(4):551–577.

Hawkes K, O'Connell JF, Blurton Jones NG, Alvarez H, Charnov EL. 1998. Grandmothering, menopause, and the evolution of human life histories. Proc Natl Acad Sci U S A 95:1336–1339.

Henderson VW. 1997. Estrogen, cognition, and a woman's risk of Alzheimer's disease. Am J Med 103(3, Part A):11S–18S.

Hill K. 1982. Hunting and human evolution. J Hum Evol 11:521–544.

Hill K. 1996. The demography of menopause. Matusitas 23:113–127.

Hill K, Kaplan H. 1988. Tradeoffs in male and female reproductive strategies among the Ache: parts 1 and 2. In: Betzig L, Borgerhoff Mulder M, Turke P, editors. Human reproductive behavior. Cambridge, UK: Cambridge University Press. p 277–305.

Hill K, Hurtado AM. 1991. The evolution of premature reproductive senescence and menopause in human females: an evaluation of the "grandmother hypothesis". Hum Nat 2:313–350.

Hill K, Hurtado AM. 1996. Ache life history: the ecology and demography of a foraging people. New York: Aldine de Gruyter.

Isaac GLl. 1978. Traces of Pleistocene hunters: and East African example. In: Lee RB, DeVore I, editors. Man the hunter. Chicago: Aldine Publishing Company. p 254–261.

Jelliffe DB, Maddocks I. 1964. Ecologic malnutrition in the New Guinea highlands. Clin Pediatr 3(7):432–438.

Johnson PL, Wood JW, Weinstein M. 1990. Female fecundity in highland Papua New Guinea. Soc Biol 37(1–2):26–43.

Kannel WB, Hjortland MC, McNamara PM, Gordon T. 1976. Menopause and risk of cardiovascular disease. Ann Intern Med 85:447–452.

Kano T. 1997. Cited by: Hawkes K, O'Connell JF, Blurton Jones NG. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long post-menopausal life spans. Curr Anthropol 38(4):551–577.

Kaplan H, Hill K, Lancaster J, Hurtado AM. 2000. A theory of human life history evolution: brains, learning and longevity. Evol Anthropol 9:156–185.

Khaw KT. 1992. Epidemiology of the menopause. Br Med Bull 48:249–261.

Kingston JD, Marino BD, Hill A. 1994. Isotopic evidence for Neogene hominid paleoenvironments in the Kenya Rift Valley. Science 264:955–959.

Klein RG. 1989. The human career. Chicago: University of Chicago Press. p 183–223.

Konigsberg LW, Frankenberg SR. 1992. Estimation of age structure in anthropological demography. Am J Phys Anthropol 89:235–256.

Kuller LH, Meilahn EN, Cauley JA, Gutai JP, Matthews KA. 1994. Epidemiologic studies of menopause: changes in risk factors and disease. Exp Gerontol 29:495–509.

Lancaster JB, Lancaster CS. 1983. Parental investment: the hominid adaptation. In: Ortner D, editor. How humans adapt: a biocultural odyssey. Washington, DC: Smithsonian Institution Press. p 33–66.

Lancaster JB, King BJ. 1985. An evolutionary perspective on menopause. In: Brown JK, Kerns V, editors. In her prime. South Hadley, MA: Bergin & Garvey Publishers, Inc. p 13–20.

Laws RM, Parker ISC, Johnstone RCB. 1975. Reproduction. Elephants and their habitats. Oxford: Clarendon Press. p 204–227.

Lee RB. 1979. The !Kung San: men, women, and work in a foraging society. Cambridge: Cambridge University Press.

Lees B, Molleson T, Anrett TR, Stevenson JC. 1993. Differences in proximal femur bone density over two centuries. Lancet 341(8846):673–675.

Leidy LE. 1994. Biological aspects of menopause: across the lifespan. Annu Rev Anthropol 23:231–253.

Leidy LE. 1999. Menopause in evolutionary perspective. In: Trevathan W, McKenna J, Smith EO, editors. Evolutionary medicine. Oxford: Oxford University Press. p 407–427.

Leidy LE, Godfrey LR, Sutherland MR. 1998. Is follicular atresia biphasic? Fertil Steril 79(5):851–859.

Leonard WR, Robertson ML. 1992. Nutritional requirements and human evolution: a bioenergetics model. Am J Hum Biol 4:179–195.

Marlowe F. 2000. The patriarch hypothesis: an alternative explanation of menopause. Hum Nat 11(1):27–42.

Marsh H, Kasuya T. 1984. Changes in the ovaries of the short-finned pilot whale, *Globicephala macrorhychus,* with age and reproductive activity. In: Perrin WF, et al., editors. Reproduction in whales, dolphins and porpoises. Cambridge, MA: Report of the International Whaling Commission. p 311–335.

Martin RD. 1983. Human brain evolution in an ecological context. 52nd James Arthur Lecture on the Evolution of the Human Brain. New York: American Museum of Natural History.

McKinlay SM, Bifano NL, McKinlay J. 1985. Smoking and age at menopause in women. Ann Intern Med 103:350–356.

Medawar PB. 1952. An unsolved problem of biology. London: HK Lewis.

Merchant K, Martorell R. 1988. Frequent reproductive cycling: does it lead to nutritional depletion of mothers? Prog Food Nutr Sci 12:339–369.

Merchant K, Martorell R, Haas JD. 1990a. Consequences for maternal nutrition of reproductive stress across consecutive pregnancies. Am J Clin Nutr 52: 616–620.

Merchant K, Martorell R, Haas JD. 1990b. Maternal and fetal responses to the stresses of lactation concurrent with pregnancy and of short recuperative intervals. Am J Clin Nutr 52:280–288.

Miller JE, Huss-Ashmore R. 1989. Do reproductive patterns affect maternal nutritional status? An analysis of maternal depletion in Lesotho. Am J Hum Biol 1: 409–419.

Moss C. 1988. Births and babies. Elephant memories. New York: William Morrow and Company, Inc.

Naeye RL. 1983. Maternal age, obstetric complications, and the outcome of pregnancy. Obstet Gynecol 61(2): 210–216.

O'Connell JF, Hawkes K, Blurton Jones NG. 1999. Grandmothering and the evolution of *Homo erectus.* J Hum Evol 36:461–485.

O'Rourke MT, Ellison PT. 1993. Menopause and ovarian senescence in human females. Am J Phys Anthropol Suppl 16:154.

Packer C, Tatar M, Collins A. 1999. Reproductive cessation in female mammals. Nature 392:807–811.

Paganini-Hill A, Henderson VW. 1994. Estrogen deficiency and risk of Alzheimer's disease in women. Am J Epidemiol 140(3):256–261.

Paul A, Kuester J, Podzuweit D. 1993. Reproductive senescence and terminal investment in female Barbary macaques (*Macaca sylvanus*) at Salem. Int J Primatol 14(1):105–124.

Pavelka MSM, Fedigan LM. 1991. Menopause: a comparative life-history perspective. Yrbk Phys Anthropol 34:13–38.

Pavelka MSM, Fedigan LM. 1999. Reproductive termination in female Japanese monkeys: a comparative life history perspective. Am J Phys Anthropol 109: 455–464.

Peccei JS. 1995a. A hypothesis for the origin and evolution of menopause. Maturitas 21(2):83–89.

Peccei JS. 1995b. The origin and evolution of menopause: the altriciality–lifespan hypothesis. Ethol Sociobiol 16:425–449.

Peccei JS. 1999. First estimates of heritability in the age of menopause. Curr Anthropol 40(4):553–558.

Peccei JS. 2001. Menopause: adaptation or epiphenomenon. Evol Anthropol 10(2):43–57.

Plummer TW, Bishop LC. 1994. Hominid paleoecology at Olduvai Gorge Tanzania as indicated by antelope remains. J Hum Evol 27:477–475.

Post JB. 1971. Ages at menarche and menopause: some mediaeval authorities. Pop Stud 25:83–87.

Potts R. 1984. Hominid hunters? Problems of identifying the earliest hunter-gatherers. In: Foley R, editor. Hominid evolution and community ecology. London: Academic Press Inc. p 129–159.

Pusey A, Williams J, Goodall J. 1997. The influence of dominance rank on the reproductive success of female chimpanzees. Science 277:828–831.

Richardson SJ, Nelson JF. 1990. Follicular depletion during the menopausal transition. Ann NY Acad Sci A592:13–20.

Richardson SJ, Senikas V, Nelson JF. 1987. Follicular depletion during the menopausal transition: evidence for accelerated loss and ultimate exhaustion. J Clin Endocrinol Metab 65(6):1231–1237.

Rogers AR. 1993. Why menopause? Evol Ecol 7:406–420.

Ruff CB, Trinkhaus E, Holliday TW. 1997. Body mass and encephalization in Pleistocene *Homo.* Nature 387:173–176.

Sacher GA. 1959. Relation of lifespan to brain weight in mammals. In: Wolstenholme GE, O'Connor M, editors. Ciba Foundation Colloquia on Ageing, Vol 5. Boston: Little Brown. p 115–133.

Sharp HT. 1981. Old age among the Chipewyan. In: Amoss PT, Harrell S, editors. Stanford, CA: Stanford University Press. p 99–109.

Shipman P. 1986. Scavenging or hunting in early hominids: theoretical frame work and tests. Am Anthropol 88:27–42.

Shostak M. 1983. Nisa. New York: Random House.

Sikes NE. 1994. Early hominid habitat preferences in East Africa: paleosol carbon isotopic evidence. J Hum Evol 27:25–45.

Smith BH. 1993. The physiological age of KNM-WT 15000. In: Walker A, Leakey R, editors. The Nariokotome *Homo erectus* skeleton. Cambridge, MA: Harvard University Press. p 195–220.

Smith BH. 1991. Dental development and the evolution of life history in Hominidae. Am J Phys Anthropol 86:157–174.

Snieder H, MacGregor AJ, Spector TD. 1998. Genes control the cessation of a woman's reproductive life: a twin study of hysterectomy and age at menopause. J Clin Endocrinol Metab 83(6):1875–1880.

Spellacy WN, Miller SJ, Winegar A. 1986. Pregnancy after 40 years of age. Obstet Gynecol 68(4):452–454.

Stanford JL, Haartge P, Brinton, LA, Hoover RN, Brookmeyer R. 1987. Factors influencing the age at natural menopause. Int J Res Scholar Clin Epidemiol Chron Illness 40(11):995–1002.

Stanley SM. 1992. An ecological theory for the origin of *Homo.* Paleobiology 18(3):237–257.

Tracer DP. 1991. Fertility-related changes in maternal body composition among the Au of Papua New Guinea. Am J Phys Anthropol 85:393–405.

Trevathan WR. 1987. Human birth. New York: Aldine de Gruyter.

Turke PW. 1988. Helpers at the nest: childcare networks of Ifaluk. In: Betzig L, Borgerhoff Mulder M, Turke P, editors. Human reproductive behavior. Cambridge, UK: Cambridge University Press. p 173–188.

Turke PW. 1997. Did menopause evolve to discourage infanticide and encourage continued investment by agnates? Evol Hum Behav 18(1):3–13.

Vrba ES. 1985. Ecological and adaptive changes associated with early hominid evolution. In: Delson E, editor. Ancestors: the hard evidence. New York: Alan R. Liss, Inc. p 63–71.

Washburn SL. 1981. Longevity in primates. In: March J, McGaugh J, editors. Aging, biology and behavior. New York: Academic Press. p 11–29.

Weiss KM. 1981. Evolutionary perspectives on human aging. In Amoss PT, Harrell S, editors. Other ways of growing old. Stanford, CA: Stanford University Press. p 25–28.

Westendorp RG, Kirkwood TB. 1998. Human longevity at the cost of reproductive success. Nature 396:743–744.

White FJ, Churchill SE. 1997. Hawkes K, O'Connell JF, Blurton Jones NG. 1997. Hadza women's time allocation, offspring provisioning, and the evolution of long post-menopausal life spans. Curr Anthropol 38(4): 551–577.

Williams GC. 1957. Pleiotropy, natural selection and the evolution of senescence. Evolution 11:398–411.

Wood B, Collard M. 1999. The human genus. Science 284:65–71.

Wood JW. 1990. Fertility in anthropological populations. Annu Rev Anthropol 19:211–242.

Wood JW. 1994a. Dynamics of human reproduction: biology, biometry, demography. New York: Aldine De Gruyter.

Wood JW. 1994b. Maternal nutrition and reproduction: why demographers and physiologist disagree about a fundamental relationship. Ann NY Acad Sci 709:101–116.

Wood JW, O'Connor KA, Holman D. 1999. Biodemographic models of menopause. Hum Biol Assoc Abstr 19:211–242.

Wrangham R, Peterson D. 1996. Demonic males. New York: Houghton Mifflin Company. p 206–207.