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Review

An evolutionary perspective on the origin and ontogeny of menopause

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Abstract

The "grandmother hypothesis" proposes that menopause evolved because ancestral middle-aged women gained greater reproductive success from investing in extant genetic relatives than from continuing to reproduce [Williams GC. Pleiotropy, natural selection, and the evolution of senescence. Evolution 1957;11:398–411]. Because middle-aged women faced greater risks of maternal death during pregnancy and their offspring's infancy than did younger women, offspring of middle-aged women may not have received the needed level of prolonged maternal investment to survive to reproductive age. I put forward the "absent father hypothesis" proposing that reduced *paternal* investment linked with increasing maternal age was an additional impetus for the evolution of menopause. Reduced paternal investment was linked with increasing maternal age because men died at a younger age than their mates and because some men were increasingly likely to defect from their mateships as their mates aged. The absent father hypothesis is not an alternative to the grandmother hypothesis but rather a complement. It outlines an additional cost—reduced paternal investment—associated with continued reproduction by ancestral middle-aged women that could have been an additional impetus for the evolution of menopause. After reviewing additional explanations for the origin of menopause ("patriarch hypothesis," "lifespan-artifact" hypotheses), I close by proposing a novel hypothesis for the ontogeny of menopause. According to the "adaptive onset hypothesis," the developmental timing of menopause is a conditional reproductive strategy in which a woman's age at onset is influenced by the likelihood that any children she could produce would survive to reproductive age. Twelve variables predicted to be associated with age at onset and evidence that bears upon the predictions is discussed.

Keywords: Menopause; Evolution; Age; Onset; Timing; Paternal investment; Absent father hypothesis; Adaptive onset hypothesis

Contents

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Unlike all other primates, women become incapable of reproducing well before the end of their life largely due to oocyte depletion [1–3]. The short-finned pilot whale (*Globicephala macrorhynchus*) [4] and the Asian elephant (*Elephas maximus*) [5] are the only other known species whose females have a comparably long postreproductive lifespan. Cross-culturally, most women experience menopause around age 50 [6] and live many years in a postreproductive state [7]. The average American woman, for example, lives 79.2 years [8], roughly 30 of which are postreproductive. Without the apparent acceleration in follicular attrition around age 40, women would have enough oocytes to last 70 years [9]. Why didn't natural selection favor a larger oocyte supply or one that became depleted more slowly so that women, like most other female animals, could reproduce throughout the majority of their adult lives?

1. On the origin of menopause

Explanations of the origin of menopause fall into two camps: adaptationist hypotheses or byproduct hypotheses. Adaptationist hypotheses view menopause as a naturally selected characteristic that increased our ancestors' reproductive success. Byproduct hypotheses do not view menopause as having been reproductively beneficial; rather, menopause is viewed as a byproduct (i.e., artifact or side effect) of other characteristics or occurrences.

The "lifespan-artifact" hypothesis views menopause as an artifact that was "uncovered" by a very recent increase in women's lifespans [10–12]. This byproduct hypothesis has conceptual and empirical problems. Conceptually, the lifespan-artifact hypothesis cannot explain why women's reproductive senescence occurs so much earlier than all other female body functions. Nor can it explain why women's, but not men's, reproductive function should terminate prematurely ([13], Fig. 1). Empirically, there is no compelling evidence that our human ancestors had a significantly shorter maximum lifespan than we do today [14]. Our maximum lifespan has remained constant for at least the past 100,000 years [15,16]. Based on body and brain masses of a catarrhine comparison group, Judge and Carey [17] predict a human lifespan of between 72 and 91 years in early *Homo* (see also Refs. [18,72]). Moreover, a substantial portion of females from all observed modern populations, including hunter-gatherer and isolated technologically primitive populations, experience a long postmenopausal life [1,19].

Several authors have hypothesized that menopause is a side effect of selective pressure to increase female lifespan [20–23]. According to this byproduct hypothesis, the derived feature in humans is not a short fertile span but an unusually long lifespan that uncovered menopause. Women's long postreproductive lifespan evolved because older women are hypothesized to have increased their reproductive success through postreproductive mother–child food sharing [21]. This hypothesis also has a host of conceptual and empirical problems [24]. For example, it is unclear how this hypothesis can explain why men typically live nearly as long as women [25] and why selection did not increase women's fertile span in conjunction with their lifespan.

The "patriarch hypothesis" views menopause as an artifact that was uncovered by an increase in *men's* lifespan [26]. Longevity is proposed to have evolved in men once they became "successful reproducers beyond their peak vigor" ([26], p. 29). According to this byproduct hypothesis, women's longevity also increased because the relevant "longevity allele" was not located on the Y chromosome. Women's longevity

was "dragged along" with male longevity, which uncovered menopause due to oocyte depletion. The merit of the patriarch hypothesis depends on the plausibility of at least two conditions: that selection could not favor a larger egg supply or one that became depleted more slowly and that the relevant longevity promoting genes were not on the Y chromosome. At present, there is little evidence for either condition.

Menopause exhibits "design features" that suggest it may have been naturally selected (G.C. Williams, personal communication, June 2000). First, menopause reliably occurs in all women who live long enough to experience it. Second, women's reproductive senescence occurs so much earlier than many other female body functions, including cardiac function, breathing capacity, renal plasma flow, and vital capacity ([13], Fig. 1, [73]). And third, women's, but not men's, reproductive function terminates prematurely. The shortcomings of the byproduct hypotheses coupled with the evidence of design that menopause exhibits have prompted many investigators to explore adaptationist explanations. The most prominent, the "grandmother hypothesis," posits that menopause evolved because it increased middle-aged women's chances of surviving to care for extant children and grandchildren by decreasing women's chances of death during pregnancy and their offspring's infancy [27–32]. In the absence of menopause, extant offspring would not have received the necessary level of prolonged maternal investment to survive to reproductive age [30–34].

Turke [35] expanded upon the grandmother hypothesis by proposing that menopause also benefited middle-aged women by reducing the risk of infanticide and by encouraging continued investment in children by fathers' kin (agnates). Due to male death, a woman who does not experience menopause "invites the presence of a new male, which in turn threatens the existence of extant offspring (especially the youngest) and also threatens to disrupt any nepotistic relationship that may have existed with the primary mate's family" ([35], p. 3).

1.1. The absent father hypothesis

In emphasizing the roles of decreased maternal and agnatic investment, previous adaptationist accounts of menopause have failed to consider how reduced *paternal* investment may have been an additional cost associated with continued reproduction by ancestral women. The "absent father hypothesis" proposes that reduced paternal investment linked with increasing maternal age was an additional impetus for the evolution of menopause. Reduced paternal investment may have been linked with increasing maternal age for two reasons: men's defection from their middle-aged mates and men's relatively earlier death. The absent father hypothesis is not an alternative to the grandmother hypothesis but rather a complement. It outlines an additional cost—reduced paternal investment—associated with continued reproduction by ancestral middle-aged women that could have been an additional impetus for the evolution of menopause.

1.1.1. Reduced paternal investment due to men's defection

Because their reproductive success historically was dependent upon their ability to gain sexual access to reproductively valuable women, men have evolved preferences for women with characteristics indicative of high reproductive value [10,36]. Men tend to prefer to mate with young women, for example, because on average younger women have higher reproductive value than older women [10,36,37]. Women's decline in reproductive value with age existed even before the origin of menopause because actuarially, a younger woman's future reproduction was always on average higher than an older woman's. Younger women always had a higher reproductive value because on average they always had (1) more eggs available for fertilization, (2) higher fertility, (3) lower risks of chromosomal abnormalities, and (4) more years available for their eggs to be fertilized, gestated, born, and reared to reproductive age.

Men's evolved preference for youth could have prompted some ancestral men to pursue younger women—either in place of or in addition to their mates—once their mates approached the end of their lives. Evidence that as men get older, they tend to prefer and marry women who are increasingly younger than they are supports this notion [38,39]. Additionally, older men's tendency to acquire status and resources could have increased their ability to attract younger women, who tend to prefer status and resources in mates [38]. Although the attractiveness of older men may have been tempered by their increased probability

of dying before children born to their younger partners reached reproductive age, the benefits that some older men can provide (e.g., personal and kin resources, protection, and status) may have outweighed the costs of their potential death. Additionally, high-status older men may have been especially attractive to younger women as extra-pair partners because women's intra-pair partners could have been duped into providing parental investment.

Ancestral mated men who defected from their middle-aged mates by mating with younger women in addition to, or in place of them, would likely have partially or wholly reallocated their resources and protection to the younger women. In polygynous mating systems, ancestral men who mated with younger women in addition to their middle-aged mates may not technically have been "defecting" on them. Nevertheless, such middle-aged women may have incurred a reduction of paternal investment because their mates may have partially or wholly reallocated their resources and protection to the younger women. As such, many ancestral middle-aged women who continued to reproduce may have lacked the necessary level of paternal investment to rear newborns to reproductive age. Evidence that fathers who leave their wives and children invest less in those children than fathers who remain with their wives and children supports this suggestion [40].

1.1.2. Reduced paternal investment due to men's early death

Cross-culturally, men tend to mate with women who are younger than they are [37] and die several years before women [7]. This combination results in a male tendency to die several years before their mates [35]. American men, for example, tend to mate with women 2.7 years younger than they are [37] and die 5.6 years before American women [8]. Thus, on average American men die 8.3 years before their mates. Higher male mortality coupled with men's tendency to mate with women younger than they are likely was a recurrent feature of our evolutionary history [35]. Thus, men's earlier death would have prevented them from protecting and investing in their mates and any offspring their mates bore near the time of their death. Middle-aged ancestral women who continued to reproduce may have lacked the necessary level of paternal investment because their mates were likely to have died before these offspring reached reproductive age.

1.1.3. Reduced paternal investment as an additional impetus for the evolution of menopause

One selective pressure associated with the high level of paternal investment in humans compared to other primates is the relatively helpless nature of human infants [14,34,41]. Without prolonged investment from both mothers and fathers or related kin, infants and young children were more likely to die before they reached reproductive age [42]. Ache children whose fathers die, for example, suffer a death rate that is 2.6 times higher than children with fathers [43]. However, as father absence was found to only marginally increase the likelihood of children's death among the Hiwi, the generality of this effect outside of the Ache (a group of forager-horticulturalists from Eastern Paraguay) is uncertain [43]. Nevertheless, "with new evidence of meat consumption during the Plio-Pleistocene and new evidence of males' nutritional contribution in extant foraging societies, it is difficult to dismiss the importance of male investment in human reproduction" ([24], p. 48).

Despite the hypothesized historical importance of paternal investment in offspring's ability to survive to reproductive age, most adaptationist explanations for menopause have emphasized solely the role of reduced *maternal* investment during middle age (reviewed in Refs. [44,45]). If paternal investment was integral to child rearing, it follows that reduced paternal investment linked with increasing maternal age added fitness benefits for the evolution of menopause. Combining the absent father hypothesis with previous adaptationist accounts suggests that menopause evolved, in part, because terminating the ability to reproduce increased ancestral women's ability to care for extant offspring by decreasing the likelihood of having to care for newborns and infants who, due to reduced investment from *both* parents, may not have survived to reproductive age.

1.1.4. Implications for tests of the adaptive nature of menopause

In their pioneering test of the grandmother hypothesis's ability to account for the current maintenance of menopause using Ache demographic data, Hill and Hurtado [13] found that the fitness benefits of

menopause and investment in kin did not outweigh the estimated fitness benefits of continued reproduction. An Ache woman's effect on her offspring's and other kin's reproductive success was not greater than the estimated number of offspring's she could successfully rear in the absence of menopause. To estimate the number of offspring a middle-aged Ache woman could produce if she did not experience menopause, Hill and Hurtado considered only the woman's expected survivorship if she continued to reproduce and her expected fertility. They did not consider the degree to which a father would be investing in the children. Because paternal investment is central in Ache child rearing [42] and is predicted to decrease with increasing maternal age, incorporating any reduction of paternal investment a middle-aged Ache woman received may significantly reduce the estimated number of children that she could rear successfully. This reduction may then reveal that postmenopausal Ache women gained more reproductive success by investing in extant children and grandchildren than they could have by continuing to reproduce.

More generally, investigations into the adaptive nature of menopause should consider multiple impetuses for its evolution including infanticide [35], infant-altriciality [31,32], and as argued here, paternal death and defection with increasing maternal age. The inconsistent findings on the adaptive nature of menopause [13,42,46–51] may be due, in part, to an underestimation of the myriad negative fitness effects incurred by ancestral middle-aged women who continued to reproduce.

2. On the ontogeny of menopause

The crux of the grandmother and absent father hypotheses is that: (1) menopause evolved to prevent reproduction and prompt investment in extant offspring because (2) reduced investment from *both* parents associated with increasing maternal age (3) decreased the likelihood that children born to middle-aged women would survive to reproductive age. However, the likelihood that children born to middle-aged women would survive to reproductive age varied. Additionally, ample variation exists in the age at onset of menopause [6,52,53]. To avoid the permanent cessation of menstruation when reproduction could still be successful, selection could have linked the timing of menopause (age at onset) with the likelihood that any children a woman could produce would survive to reproductive age. When an ancestral middle-aged woman could produce children who were *likely* to survive to reproductive age, greater reproductive success could have been gained by continuing to reproduce and experiencing menopause relatively late (compared to the mean age at onset in the local population). Conversely, when an ancestral middle-aged woman could produce children who were *unlikely* to survive to reproductive age, greater reproductive success could have been gained by experiencing menopause relatively early and investing in extant genetic relatives. However, if there were no extant genetic relatives to invest in, the best reproductive strategy would have been to reproduce and to experience menopause relatively late regardless of a future child's prospects of surviving to reproductive age. Thus, age at menopause may be a conditional female reproductive strategy attuned to the likelihood that any children they could produce in middle age would survive to reproductive age. Selection could have designed a psychological mechanism that was sensitive to: (1) the investment needs of extant children and grandchildren and (2) the ability to successfully rear future children to reproductive age. If the needs of extant dependent kin exceeded the ability to successfully rear future children, the mechanism could increase women's follicle-stimulating hormone levels, which would increase the rate of follicular attrition [9,54,74] and result in an early onset of menopause. I have termed this the adaptive onset hypothesis (AOH).

2.1. Adaptive onset hypothesis

AOH explains variation in the age at onset of menopause and not the existence of menopause itself. Evidence supporting the hypothesis would, however, provide indirect support that menopause is an adaptation because the hypothesis is derived conceptually from adaptationist accounts of menopause. As discussed earlier, adaptationist accounts of menopause have three principles. AOH draws upon principle three in suggesting that mechanisms have evolved to: (1) gauge the likelihood that children born to middle-aged women would survive to reproductive age and (2) influence the onset of menopause through manipulation of follicle-stimulating hormone levels. The potential

value of AOH is two-fold: support for the hypothesis would suggest that age at menopause is adaptively patterned (and hence predictable) and that menopause is an adaptation.

AOH can be tested by investigating if variables relevant to the investment needs of extant and future dependent kin are associated with age at onset of menopause. When conditions are such that additional reproduction is likely to jeopardize extant offspring and is unlikely to be fruitful, greater reproductive success can be gained by experiencing menopause relatively early. Conversely, when conditions are such that additional reproduction is *unlikely* to jeopardize extant offspring and is likely to be fruitful, greater reproductive success can be gained by experiencing menopause relatively late and continuing to reproduce. All things being equal, AOH predicts that the following 10 variables are associated with relatively early onset of menopause:

(1) Having poor health.
(2) Having a husband whose health is poor.
(3) Having a husband with a history of extra-pair relationships (and hence an increased likelihood of diverting resources and protection to other women in the future).
(4) Having a husband who is considerably older (and hence increasingly likely to die in the near future).
(5) The absence of an investing husband.
(6) A fat store that is below the threshold necessary to conceive and sustain pregnancy.
(7) Having extant dependents who require several years of investment to reach reproductive age.
(8) A history of high-risk pregnancies.
(9) The absence of extant kin to help raise future offspring.
(10) Having relatively limited monetary resources.

All things being equal, AOH predicts that the following two variables are associated with relatively late onset of menopause.

(11) Having a husband who is likely to protect and to invest in future offspring.
(12) Having no children or grandchildren to invest in.

Mixed support exists for an association between some of these variables and age at menopause. With regards to prediction six (fat store level), many studies find that a lower body mass index (an indicator of body fat quantity) is associated with an earlier age at menopause [55–60]. In support of prediction five (absence of an investing husband), Hidayet et al. [58] found that divorced or separated women reported an earlier onset than married women (see also Refs. [59,61–65]). In support of prediction 10 (having relatively limited monetary resources), not being employed [59], and low adult socioeconomic status are usually associated with early onset of menopause (see Refs. [66,67]) and with increased rate of entry into the perimenopause [68].

Identifying predictors of age at menopause is important because early or late onset may be associated with many postmenopausal health problems, including increased risks for ovarian cancer, breast cancer, colorectal cancer, cardiovascular disease, and osteoporosis [69]. Additionally, the ability to predict age at onset has significant implications for family planning, sterility treatment, and for the decision to pursue hormone replacement therapy [70].

Aside from smoking [59] and genetics [71], few variables significantly and consistently predict age at menopause across populations [63]. The menopause literature is fraught with conflicting effects of the "usual suspects" (parity, height, race, education level, oral contraceptive use, menstrual cycle length, and age at menarche) on age at onset. The lack of robust findings is due in part to variation in: (1) populations studied, (2) definitions used, (3) statistical techniques employed, (4) range of variables studied, (5) confounded variables controlled for, and (6) manner in which age at menopause was calculated. The unreliable effects may also indicate that the wrong variables are being investigated. Given the conflicting findings of the usual suspects, it may be worthwhile to explore whether other variables are associated with age at onset. The aforementioned 12 variables that can affect a woman's ability to rear extant and future children to reproductive age are ripe for fruitful exploration.

3. Conclusion

If the costs associated with continued reproduction were sufficiently great, natural selection could have favored the evolution of menopause [30]. Previous theorists have hypothesized that reduced maternal and

agnatic investment and an increased risk of infanticide were costs associated with continued reproduction by ancestral women [27,28,30,35]. The absent father hypothesis proposes that reduced paternal investment linked with increasing maternal age was an additional cost associated with continued reproduction. However, continued reproduction can sometimes be fruitful. According to the adaptive onset hypothesis, to avoid the permanent cessation of menstruation when reproduction could still be successful, selection could have linked the timing of menopause with the likelihood that any children a middle-aged woman could produce would survive to reproductive age. Research into the relation between variables that affect women's abilities to successfully rear extant and future children and women's age at menopause may indicate that menopause is an adaptation and that its timing is a conditional reproductive strategy.

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