

Comment

PACKER AND COLLEAGUES' MODEL OF
MENOPAUSE FOR HUMANS

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Packer, Tartar, and Collins (1998) recently reported a variety of analyses concerning longevity, fertility decline, "grandmother helping," and menopause in baboons and lions, similar to those we carried out previously using data on Ache hunter-gatherers (Hill and Hurtado 1991, 1996). Their goal was to determine whether the helping provided by elderly females to their offspring and grandoffspring could provide an adaptive explanation for why females cease reproducing in old age. This "grandmother hypothesis" of menopause (see Hawkes et al. 1989, 1998 for summary) suggests that older females can increase genetic representation more through kin help than through direct reproduction, and thus reproductive senescence has evolved through natural selection. Packer and colleagues' study is important because it provides data on two species that do not show exceptionally early reproductive senescence relative to the lifespan. However, the authors also speculate on how these data might be relevant to understanding the evolution of human menopause. Following classic senescence theory (Charlesworth 1980; Hamilton 1966; Medawar 1952; Williams 1957), they propose that (1) since few individuals survive to the age of reproductive cessation in lions, baboons, and humans, selection is weak on fertility at advanced ages and reproductive senescence is expected; and (2) human menopause takes place long before other body

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systems senesce because the period of juvenile dependence is long, and since menopausal women will soon die, reproduction would be unsuccessful (and hence a waste) if it were to take place later in the lifespan. Packer and colleagues note that baboon reproductive senescence is likewise slightly earlier than that of lions because of the longer period of juvenile dependence in baboons.

Packer and coauthors' model for human menopause is based on speculation concerning the values of some important human life history parameters for which we have previously published measures among the Ache. Here we report again the values for the relevant life history traits and their implications for Packer and colleagues' model. The Ache data are important because they come from the pre-contact period of a group living as hunter-gatherers, making them a more appropriate model than most modern populations for understanding the natural history of an evolved human trait (menopause).

Similar to Packer and colleagues we have carried out several tests of the "grandmother" hypothesis and related alternative models of menopause (Hill and Hurtado 1991, 1996). Our database is larger and includes more statistical controls for other variables that affect fertility and mortality than does the Packer study. The model we tested assumes a tradeoff between expenditure of resources and time in direct reproduction vs. expenditure spent helping kin. Rogers (1993) also explored a similar theoretical model that further includes a time delay between the costs (lost fertility) and the benefits (kin help) of alternative phenotypes. The fitness for each of the two alternatives in our model, reproduce or help kin, was assessed by calculating the rate of increase (r in the Euler Lotka equation) for populations characterized by two alternative life tables representing the two alternative life histories. Effects of maternal mortality and grandmother help were measured statistically using hazards models. Our analyses (Hill and Hurtado 1996:419–451) showed that 50-year-old Ache women could achieve six times greater genetic contribution through direct reproduction (if reproductive senescence did not occur) than they do through helping kin. Helping kin does not produce more gene copies (scaled to a reproductive value of 1; Hill and Hurtado 1996:430) than direct reproduction would, and thus, diversion of resources to kin help cannot explain the evolutionary maintenance of reproductive senescence.

Our model takes into account all relevant factors mentioned by Packer and coauthors, such as adult female survival around the age of menopause, mortality of offspring if mother dies, the measured impacts of "grandmother" investment on grandoffspring survival and the fertility of sons and daughters, and a small female fertility decline with age. Although in our model we *do* take into account female mortality after age 50 and the loss of dependent offspring if mother dies (Packer et al.'s hypoth-

esis for human menopause), menopause is still not favored! The impact of mothers on their offspring survival after age 5 is small (see below), and women are unlikely to die soon after menopause in any case. Our data support Packer and colleagues' conclusion that the impact of "grandmothers" on their close kin is small; however, it is not zero, and it could not be—otherwise there would be no evolutionary explanation for why older females help kin at all. In this sense we agree with the hypothesis (Hawkes et al. 1998) that women survive well beyond reproductive years because their activities during those years increase genetic contribution. Indeed, our data suggest that postreproductive Ache women are producing about 0.05 offspring equivalents per year through their kin-directed assistance.

Two criticisms of our 1996 study have been voiced. First, we may have underestimated the helping effect of grandmothers by comparing the survival of grandoffspring with and without a living grandmother or the fertility of adults with and without a surviving mother. This is because other related individuals may provide compensating help if the "grandmother" dies, thus dampening the effect of her absence (Hawkes first pointed this out to us after our 1991 article). This criticism could also be leveled at Packer et al.'s study. However, in the Ache case we have clearly shown that (1) the presence or absence of some other kin does affect fertility and mortality quite strongly; (2) even grandfathers have an effect larger than grandmothers on the demographic traits of some kin; and (3) older women have a larger impact on their sons' fitness than on their daughters' fitness (counter to the current formulation of the grandmother hypothesis in which mothers help daughters). Thus, while not perfectly measured, the grandmother effect is relatively weak, and unlikely to produce more gene copies than direct reproduction.

The second criticism of our study is that our data come from only one group of hunter-gatherers. This is true, just as Packer and others' study includes only one population of baboons and one population of lions. Of course it is better to use data that measure parameters from one study than simply to speculate on values. When the relevant parameters are measured in other human populations we will see whether the Ache case is exceptional. Regardless of the outcome from other studies, menopause exists among Ache women and is probably under stabilizing selection. The Ache data are therefore relevant to understanding why menopause is maintained *among the Ache*. There is no support for the proposition that Ache women experience menopause because they achieve higher fitness later in life through kin help than they could through reproduction.

We agree with Packer, Tartar, and Collins's conclusion that for baboons and lions, reproductive senescence is best seen as just general senescence. Females lose reproductive function in old age because selection is very

weak for continued reproduction at those ages. Very few baboon and lion females survive to the age of reproductive cessation. This not true for humans, however, and Packer and coauthors' hypothesis does not appear to explain human menopause.

Human females, unlike lions and baboons, show reproductive senescence long before other physiological senescence sets in (Hill and Hurtado 1996: fig. 13.2). Reproduction function in Ache females declines from the early 30s (fig. 10.7) and terminates in last birth at an average age of 42 (fig. 8.6). Women who continue reproducing into older age show longer interbirth intervals with age (fig. 8.12). During the fertility decline, age of mother is positively associated with offspring mortality (1996:299) but the absolute effect is very small. Loss of mother also increases offspring mortality up to age 10 (1996: table 13.1) but the impact is small after age 5 (Hill and Hurtado 1991: fig. 4) and paternal loss is more important to offspring survival than maternal loss after age 5 (unpublished analysis). Most important, 83% of all women alive at age 18 (first birth) are still alive at age 42 when the average woman has born her last child, and at that age women have a mean life expectancy of an additional 24 years (Hill and Hurtado 1996: table 6.1).

Thus, several facts of human life history are inconsistent with Packer and coworkers' model of human menopause. (1) Most women who survive to adulthood are still alive when reproduction ceases; thus selection is still potentially strong when menopause is expressed. Half of all Ache females ever born, and more than 80% who reach sexual maturity, survive to the mean age of last reproduction! (2) At the mean age of last reproduction Ache women have a 92% probability of surviving another 5 years and an 81% probability of surviving another 10 years. Even allowing a tenfold increase in female mortality related to childbirth for older women has little impact on these survival rates (Hill and Hurtado 1991:329). (3) The impact of maternal survival on offspring survival is very small after age 5. (4) Adult male mortality rates are higher than those for females, and paternal impact on offspring mortality is almost as great as maternal (and greater after age 5), yet male reproductive function does not cease at an early age.

Packer and colleagues' hypothesis for human menopause combines the scenario of senescence of a trait expressed late in the lifespan (Hamilton 1966) with one discussed by Hawkes et al. (1989) suggesting that women should stop reproducing at some age because they are increasingly more likely to die, and the extra offspring produced will also die if mother dies. We have shown *all* the assumptions of this hypothesis to be false using field data on a real population. Our further work has failed to support the "grandmother" hypothesis in its "opportunity costs" form, although we continue to agree with Hawkes and her coauthors that it probably explains why women survive long after reproductive cessation. We also see no ev-

idence for a "risky childbirth" hypothesis since observed rates of maternal death in childbirth are too low to overcome the advantages of reproduction (Hill and Hurtado 1991).

So why does human female fertility decline early in the adult lifespan and cease completely when the expected lifespan is still almost five times the period of high juvenile dependency on mother? Why do human females lose more than half their viable primordial follicles before reaching sexual maturity if reproduction ultimately ceases when primordial follicle number falls too low? Menopause continues to be an intriguing topic, and we are exploring new ideas that integrate menopause with other aspects of the human life history trajectory (Hill and Kaplan 1999; Kaplan et al. 1999). Continuing dialogue on this issue should be fascinating for all.

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