Why Menopause?

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Abstract

George Williams proposed in 1957 that menopause evolved because, late in life, women have more to gain from child care than from continued fertility. I develop here a quantitative model of this idea in order to determine whether the proposed benefit is in fact larger than the cost.

To make this work possible, I introduce an age-structured theory of kin selection that allows for a time delay between an act of altruism and the benefit it provides. Using this theory, I show that in age-structured populations, conventional inclusive fitness calculations are justified for effects on fertility only when either the population is stationary or there is no delay between cost and benefit. For effects on mortality, conventional calculations also require that donor and recipient be affected at the same age.

I then introduce two versions of Williams's idea. Model I assumes that menopause is maintained because it reduces the risk of mortality during childbirth, thus increasing the provision of parental care. The analysis demonstrates that this model is incapable of accounting for menopause. Model II assumes that menopause facilitates parental care by reducing the time during which a woman is partially incapacitated by the demands of pregnancy and infant care. This model could not be rejected. However, a definitive test will require parameter estimates that are not yet available.

Women who reach age 45 can expect to live several additional decades, but are extremely unlikely to produce additional children. This is puzzling. Would selection not favor women who continued reproducing into their forties and fifties? Some have suggested that this discrepancy is an artifact of a recent evolutionary change in lifespan (Weiss, 1981). But this is merely to look at the puzzle from a different direction. The puzzle becomes that of explaining how selection could have favored a longer life, given that reproduction stops by age 45. According to the evolutionary theory of senescence (Hamilton, 1966; Charlesworth, 1980), selection does not oppose mutations that increase mortality beyond the age of last reproduction. Thus, deleterious mutations accumulate late in life and the post-reproductive lifespan should be short (Williams, 1957). This seems to be true of most organisms (Williams, 1957; Hill and Hurtado, 1991) but is manifestly not true of human females.

Williams (1957) proposed a solution to this puzzle. He suggested that late in life a woman has more to gain from child care than from continued fertility. Menopause eliminates the risk that a woman will die in childbirth, and be thus unable to care for existing offspring. This increases the survival or fertility of existing offspring, thereby compensating for the foregone fertility. Menopause does not happen earlier because the reduction in risk provided by menopause is not important to young women, whose risk of mortality during childbirth is comparatively low, and who have few children to care for anyway. It has not evolved in other species because the human pattern of intense, long-lasting parental care is rare in nature.

Several authors have elaborated on this theme (Hamilton, 1966; Gaulin, 1980; Lancaster and King, 1985; Alexander, 1990; Pavelka and Fedigan, 1991). Hawkes *et al.* (1989) suggest a variant of Williams's

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hypothesis: Menopause allows mothers to devote more time to provisioning their grown daughters, thus increasing the rate at which these daughters reproduce. The older women might also achieve the same end by caring for their daughters' children (Pennington, 1991; Pennington and Harpending, 1993).

Few attempts have been made to test any of these hypotheses. Mayer (1982) suggests that if post-reproductive life is indeed advantageous, then women who live beyond menopause should have higher inclusive fitness than those who do not. Mayer must have had in mind a comparison between women who die at the age of menopause and those who live beyond. However, the comparison he actually makes is different. Instead of estimating the inclusive fitness of women who died at the age of menopause, he estimates that of women who died at any earlier age. This biases his result because women who die early in life are likely to produce fewer children simply because of their shortened opportunity for reproduction. This bias may account for Mayer's main finding—that women who live beyond menopause have higher fitness. In addition Mayer uses an erroneous formula (Dawkins, 1982, pp. 185–186) in calculating inclusive fitness. Thus, his study provides no support for Williams's hypothesis.

Hill and Hurtado (1991) perform a different test. They estimate the effect of maternal care on children's fertility, and conclude that this benefit is not large enough to compensate for the cost of menopause. However, they consider only one benefit of child care: an increased fertility of grown sons and daughters owing to assistance from their mothers.

In this paper I develop a more comprehensive model incorporating effects on survival as well as fertility, and on several generations of descendants. In order to determine whether menopause is evolutionarily stable, I consider a hypothetical rare allele that increases a mother's fertility at the age of menopause. If menopause is stable, then this allele must have an adverse effect on fitness. Thus, the problem is that of determining the conditions under which such an allele would be selected against. I will show that mortality during childbirth cannot account for the evolution of menopause, but that the opportunity cost of fertility may.

Age-structured kin selection with time delays

I employ a model of age-structured kin-selection with time delays. Kin-selection is relevant because the costs and benefits of child care accrue to different individuals: the parent and a child, grandchild, or other descendant. The model must be age-structured because parents and children are of disparate ages. Time delays are relevant because the cost and benefits are not simultaneous. A reduction in the mother's mortality at age x increases her probability of surviving to ages $x+1, x+2, x+3, \ldots$, and benefits accrue to offspring at each of these later ages. Previous age-structured models of kin selection (Charlesworth and Charnov, 1981; Morris et al., 1987; Taylor, 1990), do not allow for any time delay between the altruistic act and its resulting benefit. Thus, a new model is developed in appendix 1.

In the text, I make three simplifying assumptions regarding sex that are not made in the appendix. A mother can help her young children of both sexes, but the same may not be true of grown children and grandchildren. For example, in a matrilocal society a mother could help her grown daughters but not her grown sons, since the sons move away as they marry. For simplicity, I ignore this distinction. Second, I assume that equal numbers of daughters and sons are produced. Finally, I ignore the difference between male and female schedules of survival and fertility. By definition, $m(x)$ is the rate at which daughters are produced by women of age x. My assumption implies that $2m(x)$ is the rate at which parents of either sex produce offspring of both sexes.

Table 1 summarizes the effects on fitness of a rare altruist allele. The fate of the allele is determined by the sum of the products of the entries in the four rows. The allele will increase in frequency if this sum is positive, and decrease if it is negative. The argument underlying table 1 is given in appendix 1. Here, I

Table 1: Contributions to Fitness of Altruist Allele
The altruist allele will increase (decrease) in frequency if the sum of row products is positive (negative).

1	2	3	4	5	6	7
Effect on	Prob. of act	E[# affected]	Addi-tive effect	Repro-ductive value	Discount factor	Rel'n to donor
			Donor			
A. fert.	$l(x_1)$	1	$\delta m^0(x_1)$	1	$e^{-\rho x_1}$	1
B. mort.	$l(x_1)$	1	$\delta P^0(x_1)$	$v(x_1+1)$	$e^{-\rho(x_1+1)}$	1
			Recip. (y,τ,g)			
C. fert.	$l(x_1)$	$n_g(y,\tau)$	$\delta m^g(y,\tau)$	1	$e^{-\rho(x_1+\tau)}$	r_g
D. mort.	$l(x_1)$	$n_g(y,\tau)$	$\delta P^g(y,\tau)$	$v(y+1)$	$e^{-\rho(x_1+\tau+1)}$	r_g

present a heuristic justification of the table and illustrate its use.

Column 1 identifies the table's four rows. An act of altruism can affect the fertility (row A) and mortality (row B) of the donor, as well as the fertility (row C) and mortality (row D) of recipients. Recipients are indexed by symbols (y,τ,g), which indicate the recipient's age (y) when the benefit is received, the delay (τ) between cost and benefit, and the generation (g) to which the recipient belongs. The donor herself constitutes generation 0, her children generation 1, her grandchildren generation 2, and so forth.

The altruist allele is expressed only if the donor survives to perform her altruistic act at age x_1. Thus, column 2 shows that the allele's effect is proportional to the probability, $l(x_1)$, of surviving to this age.

Column 3 gives the expected number of individuals affected by an act of altruism, given that it occurs. Rows A and B reflect the fact that each act of altruism affects just one individual as donor. In rows C and D, the symbol $n_g(y,\tau)$ represents the conditionally expected number of recipients of generation g and age y affected when the donor is of age $x_1+\tau$, given that the donor lives at least to age x_1. An algorithm for calculating $n_g(y,\tau)$ is given in appendix 2. The product of columns 2 and 3 gives the unconditionally expected number of individuals affected by an individual with the altruist genotype.

Next, column 4 multiplies this product by the additive effect of altruism on age-specific fertility or survival. The additive effect on fertility at age x is the amount by which altruism increases fertility $(m(x))$ at that age. For example, altruism changes the donor's fertility at age x_1 from $m(x_1)$ to $m(x_1)+\delta m^0(x_1)$. The superscript 0 indicates that $\delta m^0(x_1)$ is an effect on generation 0, the donor. Similarly, altruism changes from $P(x_1)$ to $P(x_1)+\delta P^0(x_1)$ the donor's probability of surviving from x_1 to x_1+1. Finally, $\delta m^g(y,\tau)$ and $\delta P^g(y,\tau)$ are additive effects on the fertility and survival, respectively, of recipients of generation g who are aged y after delay τ. If an act is truly altruistic, then additive effects on the donor must be negative, and those on recipients positive. However, the results in table 1 hold regardless of the sign of these effects. The product of columns 2–4 gives the aggregate effect of an altruist allele on fertilities and mortalities.

Multiplying by column 5 re-expresses these effects in a single currency: births. This makes the entries in rows A and C (for fertility effects) very simple: a unit change in fertility contributes one birth to our sum. Rows B and D (for mortality effects) are less simple. Effects on survival at age x increase (or decrease) the chance of surviving to age $x+1$, in effect adding (or subtracting) individuals of reproductive value $v(x+1)$. The reproductive value can be thought of as a sum of the future births that this individual is expected to produce, with births at age y discounted by $e^{-\rho y}$.

Similar discount factors are listed in column 6, and can be understood as follows. The birth of an altruist

at time y increases the frequency of the altruist allele by $1/2N(y)$, where $N(y)$ is the size of the population at time y. Thus, we must weight each birth by $1/2N(y)$. This is equivalent to weighting by $e^{-\rho y}$ since our assumptions imply that the population is increasing exponentially at rate ρ. The product of columns 2–6 gives the expected effect of an altruist gene on births, appropriately discounted.

However, we are not interested in all births, but only in the births of new altruist individuals. Thus, each birth must be multiplied by the probability that the newborn carries the altruist allele. This is accomplished by column 7, which lists the coefficient of relationship between the affected individual and the donor. It equals 1 when the affected individual is the donor herself, and $r_g = 1/2^g$ when descendants of generation g are affected. When selection is weak and the inheritance system is diploid, the coefficient of relationship can be interpreted as the probability that the recipient has the altruist allele given that the donor does.

To illustrate the use of table 1, I shall now derive extensions of Hamilton's rule (Hamilton, 1975) that incorporate the effects of age structure and time delays. In each case I assume that there is exactly one recipient so that $n_g(y,\tau) = 1$. First, consider the case in which altruism affects mortality only so that $\delta m^0(x_1) = \delta m^g(y,\tau) = 0$. The benefit is received by a recipient of age y after a delay of τ units of time. The altruist allele is favored by selection only if the sum of row products from table 1 is positive, i.e. if

$$\begin{aligned} 0 \;<\; & l(x_1)\delta P^0(x_1)v(x_1+1)e^{-\rho(x_1+1)} \\ & + l(x_1)\delta P^g(y,\tau)v(y+1)e^{-\rho(x_1+\tau+1)} \end{aligned}$$

This is equivalent to

$$r_g b e^{-\rho\tau} v(y+1) > c v(x_1+1) \tag{1}$$

where $c = -\delta P(x_1)$ is the cost of altruism to the donor, and $b = \delta P^g(y,\tau)$ is the benefit to the recipient.

On the other hand, if altruism affects fertility but not mortality, then $\delta P^0(x_1) = \delta P^g(y,\tau) = 0$. Summing row products A and C from table 1 shows that the altruist allele is favored if

$$r_g b e^{-\rho\tau} > c \tag{2}$$

where $c = -\delta m^0(x_1)$ is the cost of altruism, and $b = \delta m^g(y,\tau)$ the benefit.

Equations 1 and 2 extend "Hamilton's rule" (Hamilton, 1975) to deal with age-structure and time delays. Comparison with the original rule, $rb > c$, shows that the original rule applies only where either there is no delay ($\tau = 0$) or else the population is stationary ($\rho = 0$). For mortality effects, the original version also requires that donor and recipient each be affected at the same age so that $v(x_1+1) = v(y+1)$.

Time delays appear not only in life history evolution, but also in contexts ranging from foraging ecology (Stephens, 1990) to evolutionary economics (Rogers, 1994). Inclusive fitness models usually ignore these delays, calculating inclusive fitness as a weighted sum of effects on self and relatives irrespective of the timing of these effects. The present results show that this procedure is justified only in stationary populations.

Two Models of Menopause

This section uses the results of the preceding section to develop two models of menopause. Both models consider the fate of a hypothetical mutant allele that increases the fertility of a woman whom I shall call Ego. The increase occurs at the age, x_1, of menopause. Each model proposes a different mechanism by which this increased fertility reduces Ego's ability to care for existing children. In each case, the goal is to specify the conditions under which the mutant allele will be selected against, i.e. the conditions under which

Table 2: Components of fitness under Model I

Row	Row Product
A	$l(x_1)\delta m^0(x_1)$
B	$l(x_1)\delta P^0(x_1)v(x_1+1)$
C	$l(x_1)\sum_{\tau=1}^{\infty}\sum_{g=1}^{\infty} r_g \sum_{y=0}^{x_1+\tau-1} n_g(y,\tau)\delta m^g(y,\tau)$
D	$l(x_1)\sum_{\tau=1}^{\infty}\sum_{g=1}^{\infty} r_g \sum_{y=0}^{x_1+\tau-1} n_g(y,\tau)\delta P^g(y,\tau)v(y+1)$

menopause is evolutionarily stable. Throughout, I assume that the population is stationary so that $\rho = 0$ and column 6 of table 1 can be ignored. This seems reasonable since density dependent population regulation probably kept growth rates near zero throughout most of our evolutionary history.

Model I: Death in childbirth

In model I, the cost of fertility is an increased risk of death during childbirth. Such deaths, of course, end any further possibility of child care, thus reducing the expected fertility and survival of Ego's descendants when she is aged $x_1 + 1$, $x_1 + 2$, and so on. Each of these later ages contributes a term to Ego's fitness.

These effects are summarized in table 2, which presents the row products from table 1 that model I implies. The row products A and B are straightforward. The sums in row products C and D include entries for all the categories of recipients that would be affected by Ego's death. The delay, τ, runs from 1 to ∞ to account for the fact that Ego's death at age x_1 would affect descendants at each future age. These effects, of course, grow rapidly smaller so that in practice it is not necessary to sum to infinity. These row products also sum over the generations (g) and ages (y) of the descendants that are affected at each value of τ.

Notice that since x_1 is the age of menopause, $v(x_1 + 1) = 0$ and the entry for row B disappears. Thus, menopause is evolutionarily stable provided that the sum of the entries for rows A, C, and D is negative.

The next step is to incorporate into the model the trade-off between Ego's fertility at age x_1 and the subsequent survival and fertility of her descendants. This is done in appendix 3, which relates the additive effects from column 4 of table 1 to two sets of parameters, α_g and β_g, which measure proportional effects of Ego's care on descendants' fertility and mortality, respectively. By definition, Ego's care increases the fertility of descendants of generation g and age y from $m(y)$ to $m(y)(1 + \alpha_g)$. Ego's care has no effect on the mortality of children older than age x_0, but reduces from $1 - P(y)$ to $(1 - P(y))(1 - \beta_g)$ the mortality of descendants of age $y \le x_0$ and generation g. Appendix 3 shows that menopause is evolutionarily stable under model I provided that

$$\frac{1-\mu}{\mu} < \sum_{g=1}^{\infty}(\alpha_g A_g + \beta_g B_g) \tag{3}$$

where μ is the probability of death during childbirth. The coefficients A_g and B_g are defined by

$$\begin{aligned} A_g &= 2r_g \sum_{\tau=1}^{\infty} l(x_1+\tau) \sum_{y=0}^{x_1+\tau-1} n_g(y,\tau)m(y) \\ B_g &= 2r_g \sum_{\tau=1}^{\infty} l(x_1+\tau) \sum_{y=0}^{x_0} n_g(y,\tau)v(y+1)(1-P(y)) \end{aligned}$$

A_g measures the potential aggregate effect of a woman's child care after age x_1 on the fertility of her descendants, weighted by coefficients of relationship. Similarly, B_g measures her potential aggregate effect

Table 3: Components of fitness under Model II

Row	Row Product
A	$l(x_1)\delta m^0(x_1)$
B	0
C	$l(x_1)\Delta t \sum_{g=1}^{\infty} r_g \sum_{y=0}^{x_1-1} n_g(y,0)\delta m^g(y,0)$
D	$l(x_1)\Delta t \sum_{g=1}^{\infty} r_g \sum_{y=0}^{x_1-1} n_g(y,0)\delta P^g(y,0)v(y+1)$

on descendants' mortality. Whereas α_g depends on behavior but not on demographic parameters, the reverse is true of A_g. α_g measures the effect of maternal care on the fertility of individual descendants. A_g can be thought of as measuring the numbers of these descendants. The same comments apply to the distinction between β_g and B_g.

Before this inequality can be used, a decision must be made about the time units to be used. The five-year intervals in which demographic data are conventionally tabulated would be inappropriate here: their use would imply that a mother's death has no effect on the child care she provides until five years later. A better approximation is obtained by assuming the time units to be instantaneous so that $v(x+1) \approx v(x)$, the sum from $\tau = 1$ is approximately equivalent to a sum from $\tau = 0$, and the expressions above become

$$A_g \approx 2r_g \sum_{\tau=0}^{\infty} l(x_1+\tau) \sum_{y=0}^{x_1+\tau} n_g(y,\tau)m(y) \tag{4}$$

$$B_g \approx 2r_g \sum_{\tau=0}^{\infty} l(x_1+\tau) \sum_{y=0}^{x_0} n_g(y,\tau)v(y)(1-P(y)) \tag{5}$$

In spite of the instantaneous time units, A_g and B_g can be approximated by applying equations 4 and 5 directly to demographic data tabulated in conventional five-year intervals.

Model II: Opportunity cost of fertility

Model II deals with another cost of fertility. During late pregnancy a woman cannot work as hard as usual. Even after her child is born, her ability to work is reduced for several months by the need to care for a young infant. Throughout this period, she is less able to provision other dependents (Hurtado et al., 1985). This may reduce these dependents' survival and fertility. Borrowing a term from economics, I refer to this as the *opportunity cost* of fertility.

The row products for this model are shown in table 3. Row product B is zero because in model II fertility does not affect Ego's survival. The care that Ego provides is diminished during a relatively brief interval from x_1 to $x_1 + \Delta t$. Since descendants are affected only during a brief interval, we can ignore variation within this interval in $m(x)$, $l(x)$, and $v(x)$. The sum over τ that appeared in Model I can thus be replaced by the product of the value at $\tau = 0$ times the width Δt of the interval.

The next step is to express the additive effects on descendant fertility and survival in terms of $\delta m^0(x_1)$, the increase in Ego's fertility at the age of menopause. I assume that the effects of absent maternal care on the fertility and mortality of an average descendant of generation g and age y are proportional to the change in their ancestor's total (male plus female) birth rate, $2\delta m^0(x_1)$. Thus,

$$\delta m^g(y,0) = -2\alpha_g^* m(y)\delta m^0(x_1) \tag{6}$$

$$\delta P^g(y,0) = \begin{cases} -2\beta_g^*(1-P(y))\delta m^0(x_1) & \text{if } y \le x_0 \\ 0 & \text{if } y > x_0 \end{cases} \tag{7}$$

Table 4: Coefficients for Model I

A_g and B_g are weights given by inequality 3 to effects on fertility and mortality, respectively, of descendants of generation g.

Generation	Fertility	Survival	
g	A_g	B_g	
		$x_0 = 5$ yrs	$x_0 = 10$ yrs
1	1.590	0.026	0.056
2	0.418	0.686	0.742
3	0.018	0.168	0.175
4	0.000	0.007	0.007

where α_g^* and β_g^* are analogous to the parameters α_g and β_g of model I. As with Model I, $\delta P^g(y,0)$ is assumed to be proportional to mortality, $1 - P(y)$, rather than to survival, $P(y)$. Substituting equations 6 and 7 into table 3 and setting the resulting sum < 0 shows that Model II can account for menopause provided that

$$1 < \Delta t \sum_{g=1}^{\infty} (\alpha_g^* A_g^* + \beta_g^* B_g^*) \tag{8}$$

where

$$A_g^* = 2r_g \sum_{y=0}^{x_1} n_g(y,0) m(y) \tag{9}$$

$$B_g^* = 2r_g \sum_{y=0}^{x_0} n_g(y,0) v(y)(1 - P(y)) \tag{10}$$

Coefficients A_g^* and B_g^* summarize the effects of all demographic parameters, while the effects of behavior are summarized in $\Delta t\, \alpha_g$ and $\Delta t\, \beta_g$. I have again assumed that the units of time are small. A_g^* and B_g^* can be approximated as before by applying the formulae directly to conventionally tabulated demographic data.

Evaluating the Models

This section estimates several of the parameters of the two models, and makes conservative guesses at the rest in order to decide whether either can account for menopause.

Model I

I evaluated equations 4–5 using data from the 1906 population of Taiwan (Hamilton, 1966), with two different assumptions about x_0, the age beyond which offspring mortality is not reduced much by Ego's presence. The results (table 4) measure the potential effect of various kinds of child care on the fitness of a menopausal woman who provides that care. This potential is realized only if the woman in fact cares for these descendants in ways that enhance their fertility or survival.

The results show that the greatest potential effects of child care are on the fertility of children (generation 1) and the survival of grandchildren (generation 2). The potential effect through survival of younger children outweighs that through fertility of grown sons and daughters.

For children, potential effects through fertility outweigh those through mortality, yet the reverse is true for grandchildren. Presumably, this is because fertility is low in the years preceding menopause, so that menopausal woman usually have more offspring of reproductive age than they have very young children. However, most of a menopausal woman's grandchildren are likely to be pre-reproductive. Thus, the largest potential effects of child care by menopausal women are on the fertility of their children and the survival of their grandchildren.

To evaluate Model I, we also need an estimate of of the probability, μ, of death during childbirth. This probability is now extremely low in the US and western Europe, but its decline did not begin until the middle 1930s. In the decade preceding this decline, the rate of death during childbirth averaged about 6 per thousand (still and live) births in New York State (Rothman and Rothman, 1987, p. 5), about 6 per thousand live births in the US as a whole, and about 10 per thousand live births among non-whites in the US (Shapiro et al., 1968, fig. 7.1). Thus, it seems reasonable to take $\mu = 1/100$ as an estimate of the rate of death during childbirth prior to the recent decline in this rate. It is possible that this rate is somewhat higher among women at the age of menopause. As we shall see, however, minor adjustments of this sort will not affect the answer.

Model I can account for menopause only if inequality 3 is satisfied. This requires information about several parameters that have not been estimated. In place of estimates, I have tried to make guesses that will tend to exaggerate the benefits of menopause. To begin with, I assume that $x_0 = 10$, that is, that child care affects survival until children are 10 years old. Second, I assume that living women double the fertility of their descendants in each generation so that $\alpha_g = 1$ for all g. Finally, I assume that $\beta_g = 1$, which implies that child care reduces the mortality of all descendants to zero. These assumptions should all exaggerate the likelihood that inequality 3 will be satisfied. Nonetheless, that inequality becomes

$$99 < 3.01$$

which is false. This means that Model I cannot account for menopause. The rate of death during childbirth is too small to outweigh the obvious benefit of continued fertility.

With realistic estimates of α_g and β_g, the right-hand side would be considerably smaller, making the situation even worse. The conclusion would not change even if we learned that μ rose by the age of menopause to 10 times the value assumed here. If menopause is advantageous it must be for some other reason.

Model II

I calculated A^* and B^* from demographic data for the 1906 population of Taiwan, as shown in table 5. As before, the main potential effects of care are on the fertility of children and the survival of grandchildren.

As before, I make guesses about parameters that have not been estimated so as to exaggerate the benefit of menopause. First, I assume as before that $x_0 = 10$, that is, that child care affects survival until children are 10 years old. Next, I assume that $\alpha_g^* = \beta_g^* = 1$, just as in the evaluation of Model I. Finally, I assume that the opportunity cost of fertility lasts three years years, since children begin to be independently mobile by about this age. This corresponds to a value of $\Delta t = 3/5$ when 5-year time units are employed. Inequality 8 becomes

$$1 < 1.13 \tag{11}$$

which is satisfied, suggesting that menopause may be evolutionarily stable under this model. However, my guesses at the values of α_g^* and β_g^* are certainly inflated, and realistic estimates might well reduce the right hand side by a factor of 10. Thus, it is not clear whether Model II can account for menopause either. It may do so, however, and better parameter estimates are needed to decide the question.

Table 5: Coefficients for Model II
A_g^* and B_g^* are weights given by inequality 8 to effects on fertility and mortality, respectively, of descendants of generation g.

Generation	Fertility	Survival	
g	A_g^*	B_g^*	
		$x_0 = 5$ yrs	$x_0 = 10$ yrs
1	1.229	0.076	0.144
2	0.004	0.498	0.512
3	0.000	0.002	0.002
4	0.000	0.000	0.000

Combining the two models

The two models can also be combined into a single model, incorporating both effects. With my assumptions, the inequality for this aggregate model is

$$1 - \mu < 1.13 + 3.01\mu$$

Since $\mu \approx 1/100$, this inequality is essentially equivalent to (11). The effect of mortality during childbirth is negligible compared with that of opportunity cost. This finding echoes that of Charnov (1992), who finds no evidence of any mortality cost to reproduction in female mammals.

Discussion

The models developed here provide a basis for evaluating Williams's (1957) hypothesis concerning the evolution of menopause. Model I attributes menopause to the effect of mortality during childbirth on the provision of parental care. This model is clearly incapable of accounting for the evolution of menopause.

Model II attributes menopause to the opportunity cost of fertility, that is, to the reduction in a mother's work capacity caused by pregnancy and infant care. It is not clear whether this model can account for menopause. A definitive answer must await estimates of several parameters. This will require research into the effects of maternal care on the mortality of young children and the fertility of grown ones. These effects can be estimated either by comparing the fertility and mortality of children with living mothers to that of children whose mothers are dead (Hill and Hurtado, 1991), or by comparing the children of mothers who are and are not caring for infants. I plan to introduce statistical methods for this purpose in a later publication.

In future work it may also prove useful to explore other ways of implementing Williams's hypothesis about menopause. In particular, neither of the models developed here takes proper account of competition between siblings for parental care. Because of this competition, a child's survival or fertility may decrease with the number of his or her siblings. Model I ignores this problem entirely. Model II allows care of small infants to interfere with care of older siblings, but takes no further account this problem.

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Appendix 1: Age-structured Kin Selection with Time Delays

This section outlines an age structured theory of kin selection in which one individual (the "donor") suffers a cost in return for a benefit to be received by a relative (the "recipient") after a delay of τ units of time. When the coefficient of relationship, r, between donor and recipient is unity, this model becomes a special case of the age structured models described by Charlesworth (1980). On the other hand, when the delay approaches zero, this theory reduces to the model of age-structured kin selection presented by Charlesworth and Charnov (1981). The argument follows that of Charlesworth and Charnov very closely, and the notation is changed only slightly. My goal here is to derive conditions under which a rare altruist allele will increase in frequency.

Consider an allele, A_2, that is a rare variant in a population dominated by allele A_1. Nearly all carriers of A_2 will be A_1A_2 heterozygotes, who will nearly always mate with the common genotype A_1A_1. Thus, other genotypes and mating types can be neglected. Subscripts m and f will be used to indicate males and females, respectively. Let

s_i = the proportion of sex i among newborns, $i \in \{m, f\}$,

$B(t)$ = the number of births at time t,

$B_2(t)$ = the number of A_1A_2 individuals born at time t,

ρ = the exponential growth rate of a pure population of A_1A_1 individuals,

$p_2(t)$ = the frequency of A_2 among individuals born at time t.

In addition, for individuals of genotype A_1A_i and sex g, where $g \in \{m, f\}$, define

$P_{ig}(x)$ = the probability of surviving from age x to age $x+1$,

$l_{ig}(x)$ = $\prod_{u=0}^{x-1} P_{ig}(u)$, the probability of surviving from birth to age x,

$m_{ig}(x)$ = the expected number of newborns of sex g produced per parent of sex g at age x,

$k_{ig}(x)$ = $l_{ig}(x)m_{ig}(x)$, the net reproductive function

$v_g(x)$ = $\left(l_{1g}(x)e^{-\rho x}\right)^{-1} \sum_{y=x} e^{-\rho y} k_{1g}(y)$, the "reproductive value" (Fisher, 1958) of A_1A_1 individuals of sex g and age x.

If A_2 is rare enough that terms of order $p_2^2(t)$ are negligible, then (Charlesworth, 1980, p. 169) its asymptotic rate of increase is given approximately by the real root, z, of the characteristic equation,

$$1 = \frac{1}{2} \sum_{x=0} e^{-(z+\rho)x} [k_{2f}(x) + k_{2m}(x)] \tag{12}$$

The altruist allele is favored by selection if $z > 0$. Our problem is thus to determine the sign of z. Define $\delta k(x)$ by

$$k_{2f}(x) + k_{2m}(x) = k_{1f}(x) + k_{1m}(x) + \delta k(x)$$

so that equation 12 becomes

$$1 = \frac{1}{2} \sum_{x=0} e^{-(z+\rho)x} [k_{1f}(x) + k_{1m}(x) + \delta k(x)] \tag{13}$$

The quantity in square brackets is guaranteed to be non-negative for all x. Therefore, if the quantity $e^{-(z+\rho)x}$ is arbitrarily replaced by $e^{-\rho x}$, the right hand side is > 1 if $z > 0$, and < 1 if $z < 0$. Because ρ is itself the solution of $1 = \frac{1}{2}\sum_x e^{-\rho x}[k_{1f}(x) + k_{1m}(x)]$, it follows that the sign of z is the same as that of $\Delta = \sum_{x=0} e^{-\rho x}\delta k(x)$. Thus, the altruist allele can invade if and only if $\Delta > 0$.

It will be useful here to establish the relationship between the net reproductive functions ($k_{2m}(x)$ and $k_{2f}(x)$) of genotype A_1A_2 and its birth rate, $B_2(t)$. First consider A_1A_2 births at time t due to females of age x. These mothers are the survivors of a cohort of $B_2(t-x)s_f$ females born at time $t-x$, of which a fraction $l_{2f}(x)$ still survives. These survivors will each have mated with males of genotype A_1A_1 (since A_2 is rare), and at age x will each produce $m_{2f}(x)/s_f$ offspring of both sexes of which 1/2 will be A_1A_2. Thus, a total of $\frac{1}{2}B_2(t-x)k_{2f}(x)$ offspring of genotype A_1A_2 are born at time t to mothers of age x. Similarly, $\frac{1}{2}B_2(t-x)k_{2m}(x)$ such offspring are born to fathers of age x. Summing over ages of parents,

$$B_2(t) = \frac{1}{2}\sum_x B_2(t-x)[k_{2f}(x) + k_{2m}(x)] \tag{14}$$

The contributions of males and females add because when A_2 is rare, matings between A_1A_2 males and females can be neglected. This formula is used several times below.

Although the effect of altruism on the donor is immediate, its effect on the recipient is delayed by τ units of time. Donors perform their altruism at age x_1, and recipients receive the benefits at age x_2. Let $\alpha = x_1 + \tau - x_2$ denote the age difference between donor and recipient, and denote by r the coefficient of relationship between donor and recipient (which can be interpreted as the probability that the recipient is of genotype A_1A_2, given that the donor is). Throughout, the subscripts D and R will be used to index the sex of donor and recipient, respectively. For example, s_D and s_R are, respectively, the proportion of the donor's and the recipient's sex among newborns. The effect of altruism turns out to depend on whether its costs and benefits affect mortality or fertility.

Mortality effects

Altruism increases the donor's risk of mortality at age x_1, and decreases that of the recipient at age x_2. The rate at which new A_1A_2 individuals are produced at time t is affected by altruism in two ways. There is (1) a reduction in the numbers of A_1A_2 parents of sex D who are older than x_1 because of the costs of altruism, and (2) an increase, owing to altruism's benefits, in the numbers of A_1A_2 parents of sex R who are older than x_2.

Mortality of donor

Altruism adds a negative quantity, $\delta P_{1D}(x_1)$, to the probability that the donor will survive from age x_1 to $x_1 + 1$. For ages $x > x_1$ this adds $\delta_1 l_{1D}(x)$ to the donor's survivorship, where $\delta_1 = \delta P_{1D}(x_1)/P_{1D}(x_1)$. Consequently $\delta_1 k_{1D}(x)$ is contributed to $\delta k(x)$ at all ages above x_1. The total contribution to Δ from mortality effects on the donor is

$$\begin{aligned}
\Delta_{DM} &= \delta_1 \sum_{x=x_1+1} e^{-\rho x} k_{1D}(x) \\
&= \delta_1 e^{-\rho(x_1+1)} l_{1D}(x_1+1) v_D(x_1+1) \\
&= \delta P_{1D}(x_1) l_{1D}(x_1) e^{-\rho(x_1+1)} v_D(x_1+1)
\end{aligned} \tag{15}$$

Row B of table 1 is obtained from this result by supressing the subscripts that indicate genotype.

Mortality of recipient

Each act of altruism adds $\delta P_{1R}(x_2)$ to the probability that a recipient will survive from x_2 to x_2+1. This increases the recipient's chances of surviving to each age x ($> x_2$) by $\delta_2 l_{1R}(x)$, where $\delta_2 = \delta P_{1R}(x_2)/P_{1R}(x_2)$. For $x > x_1, x_2$ there are additional terms of order $\delta_1\delta_2$, which can be ignored provided that selection is weak. In order to understand how all of this affects Δ it is helpful first to consider the effect on $B_2(t)$.

The number of altruistic acts at any given time depends on the number of donors of age x_1, and the combined beneficial effect of these acts adds

$$\frac{rs_D l_{2D}(x_1)\delta_2 e^{-\rho\alpha}}{2s_R l_{1R}(x_2)} \sum_{x=x_2+1} B_2(t-x) l_{1R}(x) m_{1R}(x) \tag{16}$$

to the number of A_1A_2 births at time t. Before explaining the relevance of this formula, I sketch its derivation. Recipients of age x ($> x_2$) at time t are affected by interactions with donors who were born at time $t-x-\alpha$. A total of $s_D B_2(t-x-\alpha) = s_D B_2(t-x)e^{-\rho\alpha}$ such donors were originally born, and of these a fraction $l_{2D}(x_1)$ survive to age x_1. I assume that a recipient exists for each act of altruism. Each act of altruism increased by $\delta_2 l_{1R}(x)/l_{1R}(x_2)$ the recipient's probability of surviving from age x_2 to age x. Of these recipients, a fraction r are themselves A_1A_2 heterozygotes, and each of these produces $m_{1R}(x)/s_R$ newborns (of both sexes and both genotypes) at time t, of which 1/2 are themselves A_1A_2.

Expression 16 is a contribution to the right hand side of equation 14. Equating terms in x shows that effects on recipients increase $k_{2R}(x)$ by an amount equal to the coefficient of $B_2(t-x)$ in (16). This is the contribution made to $\delta k(x)$ by effects on recipients. The contribution to Δ is therefore

$$\begin{aligned} \Delta_{RM} &= \frac{rs_D l_{1D}(x_1)\delta_2 l_{1R}(x_2+1) v_R(x_2+1)}{s_R l_{1R}(x_2) e^{\rho(\alpha+x_2+1)}} \\ &= l_{1D}(x_1) e^{-\rho(x_1+1)} r e^{-\rho\tau} \delta P_{1R}(x_2) v_R(x_2+1) s_D/s_R \end{aligned} \tag{17}$$

In the first line above I have substituted $l_{2D}(x_1)\delta_2 \approx l_{1D}(x_1)\delta_2$. This approximation is exact if $x_1 < x_2$, but otherwise ignores a term of order $\delta_1\delta_2$.

So far, I have assumed that exactly one recipient exists for each act of altruism. If instead the number of recipients is a random variable, then Δ_{RM} is inflated by a factor of n, where n is the conditionally expected number of recipients given that the act of altruism was performed. Row D of table 1 is obtained by supressing subscripts and assuming a balanced sex ratio so that $s_D = s_R = 1/2$.

Fertility effects

Let us now consider behaviors that lower the fertility of the donor, but raise that of the recipient.

Altruism adds a negative increment, $\delta m_{1D}(x_1)$, to the birth rate of each donor aged x_1. Thus, the contribution to $\delta k(x)$ is zero unless $x = x_1$, and the contribution to Δ of fertility effects on donors is

$$\Delta_{DF} = e^{-\rho x_1} l_{1D}(x_1)\delta m_{1D}(x_1) \tag{18}$$

Supressing the subscripts denoting genotype yields row A of table 1.

Altruism adds a positive increment, $\delta m_{1R}(x_2)$, to the birth rate of each recipient aged x_2. The rate of A_1A_2 births at time t is thus augmented by

$$\frac{1}{2} r B(t-x_1-\tau) l_{1D}(x_1)\delta m_{1R}(x_2).$$

Proceeding as in the case of mortality effects on recipients, we find that the contribution to Δ of fertility effects on recipients is

$$\Delta_{RF} = re^{-\rho(x_1+\tau)} l_{1D}(x_1)\delta m_{1R}(x_2) \tag{19}$$

This formula assumes that there is exactly one recipient per act of altruism. To deal with a variable number of recipients, this formula must be inflated by a factor of n, where n is the conditionally expected number of recipients, given that the act of altruism was performed. This is the basis of row C in table 1.

Appendix 2: Numbers of descendants

Let $s_i(y)$ denote the conditionally expected number of descendants of generation i born when the ancestor's age is y, given that the ancestor lives at least to age x_1. Children are in generation 1, and the conditionally expected number born at time y is

$$s_1(y) = \begin{cases} 2m(y) & \text{if } y \le x_1 \\ 2k(y)/l(x_1) & \text{if } y > x_1, \end{cases}$$

where $k(y) = l(y)m(y)$. Generations $2, 3, \ldots$ refer to grandchildren, great grandchildren, and so forth. Their expected numbers obey

$$s_{i+1}(y) = \int_0^y s_i(z)k(y-z)dz$$

The conditionally expected number of individuals of generation g and age z who are alive when the ancestor's age is $x_1 + \tau$, given her survival to x_1, is

$$n_g(z, \tau) = s_g(x_1 + \tau - z)l(z)$$

Appendix 3: Derivation of inequality 3

Let μ denote the rate of death during childbirth. I define the gross age-specific rate of female fertility, $\tilde{m}(x)$, to include all female births including those that lead to the mother's death. The net rate, $m(x) = (1-\mu)\tilde{m}(x)$, excludes these latter births since infants are unlikely to survive their mother's death. The mutant allele increases Ego's gross female fertility from $\tilde{m}(x_1)$ to $\tilde{m}(x_1) + \delta\tilde{m}^0(x_1)$ during the interval from age x_1 to age $x_1 + 1$. The net increase is

$$\delta m^0(x_1) = (1-\mu)\delta\tilde{m}^0(x_1) \tag{20}$$

Ego's increased fertility at age x_1 also reduces the probability of her survival from x_1 to $x_1 + 1$. If Ego's fertility were normal, this probability would be

$$P(x_1) = e^{-2\mu\tilde{m}(x_1)}\tilde{P}(x_1)$$

where $\tilde{P}(x_1)$ is the probability of survival in the absence of fertility and $e^{-2\mu\tilde{m}(x_1)}$ is the probability of surviving death during childbirth between ages x_1 and $x_1 + 1$. The factor of 2 appears because although only female births contribute to the sum from table 1, the mother's mortality rate is increased by births of both sexes. Because of her increased fertility, Ego's probability of survival from x_1 to $x_1 + 1$ is

$$P(x_1) + \delta P^0(x_1) = e^{-2\mu[\tilde{m}(x_1)+\delta\tilde{m}^0(x_1)]}\tilde{P}(x_1)$$

These equations imply that

$$\delta P^0(x_1) = \left(e^{-2\mu\delta\tilde{m}^0(x_1)} - 1\right) P(x_1) \approx -2\mu\delta\tilde{m}^0(x_1)P(x_1) \tag{21}$$

provided that $\mu\delta\tilde{m}^0(x_1)$ is small.

Additional indirect contributions appear because Ego's probability of surviving from birth to each age $x > x_1$ is decreased to

$$l(x) + \delta l(x) = l(x)\frac{P(x_1) + \delta P^0(x_1)}{P(x_1)} = l(x)[1 - 2\mu\delta\tilde{m}^0(x_1)]$$

The change in survival for $x > x_1$ is thus

$$\delta l(x) = -2l(x)\mu\delta\tilde{m}^0(x_1) \tag{22}$$

If Ego does not survive to age x, she will be unable to care for her children, grandchildren, and other descendants, thus reducing their fertility and survival.

To account for these effects on descendants, I assume that the mutant allele adds

$$\delta m^g(y,\tau) = \delta l(x_1 + \tau)\alpha_g m(y) \tag{23}$$

to the fertility of each generation-g descendant who is y years old when the ancestor is of age x. The parameter α_g measures the proportional effect of Ego's care on the fertility of these descendants.

Similarly, β_g measures the proportional effect of Ego on descendants' mortality. I assume that child care by Ego has no effect on the survival of children older than age x_0, but that it increases from $P(y)$ to $P(y) + \beta_g(1 - P(y))$ the age-specific survival of children of age $y \leq x_0$ and generation g. In expectation, the mutant allele adds

$$\delta P^g(y,\tau) = \begin{cases} \delta l(x_1 + \tau)\beta_g(1 - P(y)) & \text{if } y \leq x_0 \\ 0 & \text{otherwise} \end{cases} \tag{24}$$

to the survival of each of these descendants. Notice that the effect on survival is proportional to mortality, $1 - P(y)$, rather than to survival, $P(y)$.

Inequality 3 is obtained by substituting equations 20–24 into table 2 and setting the resulting sum < 0.

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