Rethinking the evolutionary theory of aging: fertility, mortality, and intergenerational transfers

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I use a new model in which population equilibrium depends on density, consumption and intergenerational transfers to rework Hamilton's classic evolutionary theory of aging. The age specific force of selection on mortality is a weighted average of the classic effect, proportional to remaining lifetime fertility, and a new transfer effect, proportional to cumulative net investment per offspring up to that age. The relative weight of the classic effect can be positive, negative, or zero. At an optimal equilibrium, the evolution of senescent mortality depends only on the transfer effect. The new theory explains mortality decline before sexual maturity as well as extended post-reproductive survival. A positive feedback loop can select for reduced fertility, higher consumption, greater investments in juveniles, and longer life. For many species, the evolution of aging is driven more by age patterns of transferring surplus food provisions than by remaining fertility. Human hunter-gatherer mortality corresponds poorly to the classic predictions, but matches closely the transfer effect predicted by this new theory.

Introduction

The classic theory of the evolution of mortality and fertility [1,2,3] was formalized by Hamilton[1], through analysis of the population renewal equation. The force of selection on a mutation that reduces mortality at some particular age *a* is proportional to *F(a)*, the lifetime reproduction that remains after age *a*. Since *F(a)* declines only after reproduction starts, selection should be equal at all ages up to sexual maturity[1,4,5]. If reproduction ends, *F(a)* is 0 and there is no selection for post-reproductive survival. The force of selection on a mutation that raises fertility at age *a* is proportional to the probability of surviving to that age. Although Hamilton's work has been extended and qualified, it is still the dominant paradigm for the evolution of aging[4,5].

The classic theory is simple and elegant, but it treats fertility and mortality as functions of age alone. If we consider the resource costs of mortality in addition to age, then we might expect that selection would rise at ages when cumulated parental investments were greater, rather than remaining constant across the pre-reproductive period[5], and that selection would favor post-reproductive parental survival if parents continue to provide care[3]. If a species has evolved an optimal strategy of low fertility and heavy parental investment, then higher fertility would reduce reproductive fitness rather than raise it, and we would not expect it to be positively selected.

As these considerations suggest, fertility and mortality are interdependent through resource constraints in ways that invalidate Hamilton's analysis of the renewal equation. Age-specific fertility and mortality cannot be treated as varying independently. Higher parental fertility might increase the mortality of juveniles, or reduce future parental fertility. Higher adult mortality might raise the mortality of dependent juveniles. Lower

juvenile mortality at one age could raise mortality of juveniles at other ages by increasing competition for food supplied by adults.

The theory proposed here is consistent with the large literature on the evolution of life histories that considers trade-offs among allocations of resources to growth, reproduction, repair and survival [6,7,8,10]. That literature focuses on trade-offs in a single organism. The theory proposed here instead emphasizes constraints, trade-offs and selective forces arising from the relations among members of a group of related organisms due to intergenerational resource transfers among them. Fertility is itself a resource transfer, so intergenerational transfers occur within all mother-offspring sets. However, with parental care or cooperative care, transfers have greater implications.

Here, I incorporate these ideas in a formal theory which builds on the classic theory by adding a resource balance constraint: The *population-weighted sum across all ages of net transfers of food must be zero*. This approach derives from economic and demographic models of intergenerational transfers[8] building on Samuelson's seminal work[9]. In the extended theory, the force of selection on mortality at age a is a weighted average of the classic force, remaining fertility, $F(a)$, and a new force, remaining transfers to others, $T(a)$ (or, equivalently, the cumulated net investment in an individual up to age a). With parental care, $T(a)$ first rises with age and then falls in adulthood, depending on transfer patterns in each species, and mortality should move inversely. The weight on $F(a)$ can be positive, zero or negative. In one important case, selection favors *lower* fertility and *higher* juvenile mortality along with lower adult mortality, so as to approach the optimal trade-off between numbers of offspring and investment in each. At this optimal point, the force of selection on fertility is zero, and the age pattern of selection on mortality depends solely on the transfer effect, $T(a)$.

To understand $T(a)$, consider a simplified example. Parents have resources W to invest in offspring, and invest $\tau(x)$ per surviving juvenile at age x. Fitness is the number of offspring surviving to maturity. Under these assumptions, the force of selection on mortality at age a is proportional to the cumulative survival-weighted net investment, $T(a)$, which rises with age, contrary to the classic theory. A death is more costly when more has been invested in the juvenile that dies, that is when $T(a)$ is greater.

The Basic Model: Population Renewal and Transfer Balance

Consumption of food at age x, $c(x,\gamma)$, depends on both age and the general level of consumption γ, with $dc(x,\gamma)/d\gamma>0$ for all x. This formulation allows complicated changes in consumption by age to be described by variations in a single parameter, γ. Two examples are $c(x,\gamma) = \gamma k(x)$ and $c(x,\gamma)=h(x)+\gamma k(x)$. In the first, consumption always varies proportionately at all ages. In the second, consumption might vary more or less than proportionately at some ages, and in particular food shortage could fall most severely on juveniles. Fertility, denoted $m(x,\gamma)$, depends on γ with $dm/d\gamma>0$. The force of mortality, denoted $\mu(x,\gamma)$, depends inversely on consumption of food: $d\mu/d\gamma<0$. Consequently, survivorship, $l(x)$, depends positively on γ. The form of $c(x,\gamma)$ affects the forms of all the functions that depend on γ, and this could be made explicit by writing them as functions of $c(x,\gamma)$ rather than γ alone.

Two equations must hold for a population in steady state. The first is Lotka's renewal equation.

$$(1.1) \quad 1=\int_0^{\omega} e^{-rx} l(x,\gamma) m(x,\gamma)\, dx .$$

I will call the integral R for Renewal. $R=1$ implicitly defines r, the stable population growth rate, as a function of γ, $r = r_R(\gamma)$ with $r_R'=dr_R/d\gamma>0$. I will call $r_R(\gamma)$ the renewal

curve. When consumption at all ages is greater, more organisms survive, and fertility is higher, so the population growth rate, r, is higher.

The second equation is key to all that follows. It states that provisioning (food acquired) minus consumption at each age, weighted by the stable population-age distribution, must sum to zero, assuming that all food acquired at a given time is also consumed at that time. Straightforward modification would allow wastage or storage[8].

Provisioning at age x depends on γ: $y(x,\gamma)$, $dy/d\gamma>0$. Food intake affects energy available for foraging, and lifetime consumption affects body size and age at attaining maturity, which in turn affects provisioning success. Provisioning also depends on population density through the function $\pi(A/N)$ which indicates the ease of acquiring food. A reflects the natural resource base in a specific area and N is the size of the population in this area, assuming negligible migration: $d\pi/dN<0$, $d\pi/dA>0$. For simplicity, population pressure depends on the total population size, but a complete treatment would include age-specificity in the denominator of A/N as well[10]. The full expression for age-specific provisioning is $\pi(A/N)y(x,\gamma)$ (or, more generally, $y(x,\pi,\gamma)$).

$$(1.2) \quad 0=\int_0^{\omega} e^{-rx} l(x,\gamma)\left[\pi\left(A/N\right) y(x,\gamma)-c(x,\gamma)\right]dx$$

I will call this integral B, for Balance. Lee[8] analyzes this equation more generally, and it enters Kaplan-Robson's[11]theory of human evolution, in both cases without density dependence or γ: $B=1$ implicitly defines $r=r_B(\gamma,\pi)$ as a function of $\gamma$and π, but I will often suppress the argument π, simply writing $r_B(\gamma)$. I call this the balance curve.

For given γ, higher population density reduces π, which reduces food provisioning at every age, requiring a higher ratio of adults to juveniles to achieve $B=0$. To achieve this higher ratio, the population growth rate must be lower. Therefore, greater population

size and density shift the balance curve $r_B(\gamma)$ downward, and $dr_B(\gamma,\pi(A/N))/dN = (dr_B/d\pi)*d\pi/dN<0$.

The net transfer made or received by a surviving individual at age x is $\tau(x,\gamma,\pi)=\pi(A/N)y(x,\gamma) - c(x,\gamma)$. For biologically relevant cases, $\tau(x)$ will be ≤ 0 up to a certain age of economic maturity, and positive thereafter. Only for humans does it sometimes turn negative again at old ages[12,13].

If there is no care of offspring, or if only the mother provides it, then the no-wastage, no-storage assumption locks together the fertility schedule and the mother's transfer schedule in a way that could not hold as γ varies, given the rigid specification of the age schedules in the model. In nature, this rigidity would be relaxed through varying maternal body reserves, differential treatment by birth order, varying infant mortality, and so on. A fuller model would allow for individual variations in effort and consumption in response to changing circumstances and allow bodily reserves to be built up or depleted. However, I believe that the conclusions of this simpler model would continue to hold.

The balance curve $r_B(\gamma,\pi)$ reflects many influences. π and γ jointly determine provisioning, consumption, and net transfers at each age via $\tau(x,\pi,\gamma)$. The value of γ also determines the death rate and survival function at each age. The survival function influences the population-age distribution: higher mortality means fewer adults per juvenile unless it is entirely limited to the juvenile ages, depending on the $\mu(x,\gamma)$ function. With all these functions fixed, we can solve for the population growth rate r that satisfies the balance equation. In a stable population, the age distribution is proportional to $e^{-rx}l(x)$. When r is high, the population is younger and the adults/juveniles ratio is lower. If net transfers are negative for juveniles and positive for adults, with no negative transfers in old age, then a unique r will satisfy $B=0$. In this way γ and π determine r, given implicitly by $r_B(\gamma,\pi)$.

For sufficiently high values of γ, $r_B' = dr_B/d\gamma < 0$. In this case, increases in the level of consumption require a higher adult/juvenile ratio, and therefore a lower population growth rate. For some species, this may be true across the entire range of γ, and I speculate this is true for opportunistic species. For other species there may be one or more lower ranges of γ in which $r_B' > 0$, so that higher levels of consumption can be accommodated by higher population growth rates with lower adult/juvenile ratios. In this case the balance curve, $r_B(\gamma)$, would be hump shaped over some range of γ, and at some $\gamma\#$, r would reach a (possibly local) maximum.

The balance curve might slope upward for two reasons. First, higher γ raises survival, particularly of juveniles, so wastage of resources is avoided, and with lower mortality the adult/juvenile ratio will rise. Second, higher γ raises productivity through larger body size and increased energy. Consequently, it may be possible to accommodate a higher population growth rate at a higher level of consumption. This will be true whenever there is an intermediate optimal brood size or birth interval, with corresponding optimal investment, which is the case for many species[14, 15, 11]. Optimality requires that slightly lower fertility and greater investment, or slightly higher fertility and lower investment would lead to a lower population growth rate. This requires that the balance curve have a hump. The hump summarizes the outcome of allocational trade-offs for the organism[11].

The equations apply to a particular species at a particular point in its evolution, for a particular region around the equilibrium values. General shapes of the curves might reflect broad features of taxa, such as primates or mammals, or ecological types, such as opportunistic or equilibrating species. Mutations may change the position and shape of the curves in ways discussed in later sections.

The model can be made sex-specific. In that case, (1.1) refers to one sex and the balance in (1.2) holds for the sum of both sexes, while the balance for males or females separately may be nonzero, albeit with identical r's.

Population Equilibrium: Density, Age Distribution, and Transfers

Figure 1 plots $r_R(\gamma)$ and $r_B(\gamma,\pi)$ for the case of a downward sloping balance curve, $r_B'<0$. For $\pi=\pi_1$, equations (1.1) and (1.2) determine steady state levels of r and γ at the (arbitrarily drawn) intersection of the solid lines at point X. Because $r>0$, the population increases over time to a higher density where $\pi = \pi_2$ is lower. Here, balance requires a higher ratio of adults to juveniles, so r at each level of γ must be lower. The balance curve now intersects the renewal curve at Z, where population equilibrates at $r=0$, $\gamma=\gamma^*$.

Z is simply where the renewal curve intersects the line $r=0$, $\gamma^*=r_R^{-1}(0)$, so its location is determined without reference to the balance curve, which therefore seems superfluous. However, without the balance equation we would not know the population size corresponding to $\gamma=\gamma^*$. This value depends on the distance that the balance curve must shift to intersect the renewal curve at $r=0$. The balance curve translates density into age-standardized consumption γ, reflecting the rich set of biological and demographic relationships incorporated in B. Sometimes, lower equilibrium γ is associated with higher density as we would expect. Other times, equilibrium γ varies while density remains constant, and sometimes γ and density increase together, as shown later. That is why the classic theory can mislead, and why the full model is essential for comparative statics, such as the force of selection on mutations.

The Age-Independent Component of Selection

Following Hamilton[1], I take r (the Malthusian parameter) to measure fitness, with the force of selection on a mutation assumed to be proportional to its effect on r. In many circumstances this assumption is incorrect[4,16] because reproduction typically involves two sexes. However, according to Rose[5] it is a "reasonable working assumption for the evolution of most populations." Further work on this theory is needed to deal better with diploid sexual reproduction in antagonistic pleiotropy, and with mutation accumulation and balance[5,16].

Let us consider the effect of a mutation that reduces mortality at some specific age (Figure 2). The original population equilibrates where the solid lines (superscript 1) cross at Z, with $r=0$ and $\gamma=\gamma_1^*$. The mutation raises the renewal curve for the mutant line to $r_R^2(\gamma)$ by the vertical difference between Z and V. The size of this vertical shift depends on the specific age and is equal to the force of selection at this age in Hamilton's analysis. However, in the extended theory, lower mortality also raises the balance curve for the mutant line to $r_B^2(\gamma)$, drawn here as a smaller shift. The intersection of the new curves, shown as dashed lines (superscript 2), occurs at point X, and the vertical distance between Z and X is the actual force of selection on this mutation (again depending on age). The balance equation constraint has reduced the Hamilton effect of the mutation on the growth rate, because the reduction in mortality makes the age distribution younger as the population growth rate rises (higher growth rate will typically dominate the countervailing effect of longer life on the age distribution). The younger age distribution requires lower consumption for the mutant line. The selection effect, equal to the vertical increase from Z to X, depends on both the size of the shifts in the two curves and the slopes of the two curves, as will be seen formally in the mathematical analysis later. The shifts in the curves need not be parallel as drawn here. Selection will also act on mutation-driven changes in the slopes, so both the shape and level respond to changes in the fertility and mortality functions.

The mutant and original populations each produces and consumes separately according to its own equations, but they share the resource base and provisioning ease; for both is subject to the same $\pi(A/N)$. N is the sum of the original population and the mutant population. At X, the mutant line is growing while the original population is stationary at Z. As the mutant population size and therefore total population increases, π falls, shifting the mutant balance curve down to $r_B^3(\gamma)$ (shorter dashes, change shown by arrow), where it intersects the mutant renewal curve $r_R^2(\gamma)$ at U, with $\gamma=\gamma_2^*$. The rising density also shifts the balance curve for the original population downward, so that it has negative growth. Eventually the mutant line becomes 100% of the population and reaches a new equilibrium at $r=0$ and γ_2^* at point U. A mutation raising fertility has similar effects, except that initially only the renewal curve is shifted while the balance curve remains in its original position. As density eventually increases, the balance curve will be shifted down in this case as well.

Although the balance curve in Figure 2 does not have a hump as drawn, we can imagine that intersections Z and X are on the right of a hump which has a peak out of sight to the left. Then selection on new mutations affecting mortality or fertility would eventually move the equilibrium to the peak of the hump, where $r_B'=0$. Density dependent adjustment of the balance curve would drop it down until the crest of the hump touched the line $r=0$ at a single point of tangency, which I call the optimum equilibrium.

Selection in the region to the left of the peak can be counterintuitive. In Figure 3, the life history of the species is such that $r_R^1(\gamma)$ and $r_B^1(\gamma)$ intersect on the left slope of a hump at equilibrium Z with $r=0$, $\gamma=\gamma^*$. A mutation that shifts the renewal curve upward or leftward (not shown in the figure) would intersect the original balance curve $r_B^1(\gamma)$ at a negative growth rate, and such a mutation would be selected out of the population. This effect shows that a mutation that raised fertility or lowered mortality would be negatively

selected. In other words, the classic selection results are reversed here. By contrast, a mutation that reduced fertility, shifting the renewal curve downward (or to the right), as shown by $r_R^2(\gamma)$ would raise r and be positively selected. A mutation that raises mortality would also be positively selected, to the extent that it shifted the renewal curve downward. However, it would be negatively selected to the extent that it shifts the balance curve downward. Selection favors higher juvenile mortality but lower adult mortality on the left of the hump (this point will be discussed in more detail below). A mutation that shifted the balance curve up to $r_B^2(\gamma)$ would be positively selected.

Through selection on fertility and mortality, the life history is guided toward the optimal equilibrium at the peak of the balance curve, which is therefore an evolutionary and ecologically stable equilibrium with $r_B'=0$. If mutations shift the renewal curve slightly upward, intersection with the balance curve moves slightly to the left, reducing γ for the mutant line although population density is unchanged. Then γ declines only because the mutation shifts the life history away from the optimal equilibrium, and reduced efficiency lowers γ for this mutant line at the given density level. Because its r is reduced, the mutant line goes extinct. The classic selection effects vanish at the optimal equilibrium. Nonetheless, mutations shifting the balance curve upward by reducing mortality will still be positively selected. Here, mortality selection is driven entirely by the transfer effects from the balance function.

As mutation and selection move the life history to the peak from the left, both γ and density increase. We know density is increasing because the selected mutations raise the population growth rate and the balance curve shifts down. Figure 3 shows that γ is increasing as well. Selection leads to a more efficient life history, permitting the species to equilibrate at a higher density by investing more in each offspring (higher γ), and in this way crowds out the original population even though it can achieve $r=0$ at a lower

level of γ. These strange and counterintuitive results to the left of a peak represent the positive feedback loop mentioned earlier, and may help explain the evolution of primates and other kinds of species with low fertility, heavy investment in offspring, and long adult life. This theory generates the positive feedback cycle under very general conditions.

The Age-Specific Force of Selection

I examine the effect on r of a mutation that increases fertility at age a by introducing an age-specific shifter term $\varepsilon(a)$ into the fertility equation, so that fertility is $m(x,\gamma)+\varepsilon(a)$. Because the change occurs only at age a, $\varepsilon(a)$ is 0 except at $x=a$. Now differentiate both (1.1) and (1.2) with respect to $\varepsilon(a)$. (View the renewal equation as a Stieltjes integral in which cumulative fertility has a step at age a.) Equating $d\gamma/d\varepsilon(a)$ from the two equations and solving for $dr/d\varepsilon(a)$, we arrive at equation (3.1), provided that the slopes of the renewal and balance curves are not equal (see Appendix). {OR (Supplementary material can be found at www.nature.com).}

$$(3.1)\qquad \frac{dr}{d\varepsilon(a)}=\frac{e^{-ra}l(a)}{A_f}\left(\frac{-r_B'}{r_R'-r_B'}\right).$$

The first ratio on the right is Hamilton's[1] force of selection on fertility at age a, where A_f is the average age of childbearing in the stable population. The second ratio is a weighting factor that is independent of age, the effect of which was explored in the preceding diagrams. If the balance curve has no hump, then its slope is always negative, and this weighting factor is always positive. In this case the force of selection is proportional to that in Hamilton's result. Now suppose the balance curve does have a hump, and denote consumption at its peak by $\gamma\#$. Then the weighting factor ranges from negative to positive values as γ varies, and passes through 0 at $\gamma\#$ where $r_B'=0$ by the first

order condition for a maximum. Left of the peak ($\gamma<\gamma\#$) selection favors reduced fertility in its approach to the optimum. At the peak ($\gamma=\gamma\#$) the force of selection is zero. Since higher fertility is selected to the right of the peak, and lower fertility to the left of the peak, selection will adjust fertility so as to reach the peak at $m(x,\gamma\#)$.

Now consider a mutation that reduces the force of mortality at age a by $\delta(a)$, so that the force of mortality is given by $\mu(x,\gamma)-\delta(a)$. Differentiate (1.1) and (1.2) with respect to $\delta(a)$, a procedure we can make formal by considering a step of $\delta(a)$ at age a in the cumulative mortality hazard. We then equate $d\gamma/d\delta(a)$ in the two equations, and solve for $dr/d\delta(a)$ (see Appendix) finding:

$$(3.2)\quad \frac{dr}{d\delta(a)}=\frac{F(a)}{A_f}\left(\frac{-r_B'}{r_R'-r_B'}\right)+\frac{T(a)}{C\left(A_y-A_c\right)}\left(\frac{r_R'}{r_R'-r_B'}\right).$$

The first ratio on the right is Hamilton's result, where $F(a)$ is the share of fertility remaining after age a, weighted by survival and discounted. This ratio is multiplied by the same factor as was the Hamilton effect for fertility in (3.1). The next ratio is a new selection effect that is proportional to $T(a)$, the transfer effect. $T(a)$ is the cumulative net investment (transfers) up to age a per birth or, equivalently, the remaining lifetime transfers made after age a per newborn. (Equivalence follows from the balance equation, which must integrate to 0, so the parts above and below any particular age must be equal, but of opposite sign.) It is divided by a number analogous to A_f, and which does not vary by age. C is the discounted and survival-weighted lifetime consumption per birth, equal to similarly defined πY when $B=0$. A_y and A_c are the average ages of producing and consuming in the stable population. When A_y is greater than A_c, the net direction of intergenerational transfers is downward across age, from older to younger, as is probably the case for all species with the exception of humans living in modern industrial

societies[13]. The full selection effect is the weighted average of the Hamilton effect and the transfer effect, with weights summing to unity.

There are five cases: a) The balance curve has no hump, so its slope is always negative, and consequently both weighting factors are always positive. Both are reflected in the total selection effect, with relative weights that vary with γ. b) On the right side of a hump ($\gamma > \gamma\#$) both weights are positive. Selection on both fertility and mortality moves the life history towards the peak at $\gamma\#$. The relative weight of the Hamilton effect goes to zero as the peak is approached, and that of the transfer effect goes to unity. c) On the left side of a hump ($\gamma < \gamma\#$), any intersection at which $\partial r_R/\partial\gamma \leq \partial r_B/\partial\gamma$ is unstable, because any mutation in fertility or mortality would lead to a non-intersection of the two curves in that region, and extinction of the lineage. There will also be a second intersection of the curves to the right, with $\partial r_R/\partial\gamma > \partial r_B/\partial\gamma$, which is the one that will be observed. It falls into either case b) or case d). d) On the left side of a hump ($\gamma < \gamma\#$) with $\partial r_R/\partial\gamma > \partial r_B/\partial\gamma$, the weight on the Hamilton effect is negative and the weight on the transfer effect is greater than unity. The negative weight on the Hamilton effect means an age pattern of mortality increase is selected so as to reduce the number of births, and to move the life history towards the optimum equilibrium at $\gamma\#$. Higher juvenile mortality is a poor substitute for lower fertility, but it nonetheless enhances fitness by raising r if equilibrium consumption is below the optimal level. At the same time, an age pattern of mortality reductions (proportional to $T(a)$) that economize on invested resources is positively selected; this effect also moves the life history toward the optimum consumption at $\gamma\#$. e) At the peak of the hump, $\partial r_B/\partial\gamma = 0$ (first order condition for a maximum of $r_B(\gamma)$). The Hamilton effect gets zero weight, and the age pattern of the force of selection on mortality is entirely determined by the transfer effect, $T(a)$. While there is zero selection on fertility at this optimum equilibrium, selection for lower mortality in proportion to $T(a)$ continues. I expect that this is the relevant case for many species.

The force of selection on mutations which alter the age-shape of $y(x,\gamma)$ and $c(x,\gamma)$ can be analyzed in exactly the same way. For $y(x,\gamma)$, let $\phi(a)$ be a perturbation at age a:

$$\frac{dr}{d\phi(a)} = \frac{e^{-ra}l(a)}{C\left(A_y - A_c\right)/\pi}\left(\frac{r'_R}{r'_R - r'_B}\right) \tag{3.3}$$

and similarly for $c(x,\gamma)$ but of opposite sign. When survival to older ages is low, there will be low selection to increase transfers from that age. As higher survivorship to a evolves, the selective pressure for making larger transfers at age a will increase. As these transfers increase, $T(a)$ is raised, and there is a feedback to greater pressures to select lower mortality at this age and above. The functions governing fertility, mortality, provisioning, consumption, and transfers all evolve in interactive and path-dependent ways.

Some Implications of the Theory

$T(a)$ can be interpreted either as transfers received up to age a, or as transfers made after age a, because the balance equation tells us these must be equal. Because transfers are often closely related to fertility itself, particularly for species without parental care, $T(a)$ will often look much like Hamilton's $F(a)$ and have similar implications for the intensity of selection on mortality across age. The greatest differences occur when there is parental care, which makes $T(a)$ rise after birth and remain positive post-reproductively so long as transfers continue. The theory predicts falling juvenile mortality and continuing post-reproductive survival in this case. Cooperative breeding[17] will also make $T(a)$ and $F(a)$ diverge. The average infant in an Efe hunter-gatherer group is cared for by 11 people in addition to its parents[18], and food sharing is common among hunter-gatherer groups.

$T(a)$, $F(a)$ and mortality in Figures 4 and 5 are calculated for the Ache, a well-studied human forager group in Paraguay[13,19,20]. The force of selection for mortality is a weighted average of the classic and transfer effects, shown in Figure 4 for a range of

weights. The bold curve is *T(a)*, corresponding to the force at the optimal equilibrium. *F(a)* has a positive weight if there is no hump and to the right of a hump, and a negative weight to the left. The curve for this last case shows the combination of positive selection for higher early juvenile mortality and lower late juvenile and adult mortality.

Species that have evolved an optimal balance between quantity-quality in reproduction and investment will be situated at the optimal equilibrium, so their age pattern of mortality should be driven entirely by *T(a)*. Since a high force of selection should correspond to low mortality, Figure 5 compares *T(a)* and *F(a)* with the inverse of the age-specific death rates. Inverse mortality for the Ache and (to a lesser degree) the 18th century Swedes correspond strikingly with *T(a)* and not with *F(a)*. The contrast is particularly strong for pre- and post-reproductive age ranges.

For non-humans, we can distinguish several types of cumulative transfer functions, that is, the sum to a of $\tau(x)$ before survival-weighting or discounting. a) Semelparous with no parental care (e.g., annual plants): The cumulative transfer functions are rectangular, receiving an investment all at once when born, and paying it back all at once when they reproduce. Constant force of mortality. b) Iteroparous with no parental care (e.g., perennials, trees, many animals): The cumulative transfer function starts with a step upward, then, after sexual maturity, returns towards zero in a series of smaller steps reflecting each year's investment in fertility. c) Rising force of mortality: Iteroparous with parental care up to maturity (e.g., many insects, birds, mammals, particularly primates). The cumulative transfer function rises prior to sexual or economic maturity, falls over the course of reproduction, and continues to fall as transfers to offspring continue even after biological reproduction has stopped. Force of mortality falls until sexual maturity, then rises gradually with no necessary acceleration towards end of reproduction. d) Cooperative breeding (e.g. some birds, humans): The cumulative transfer

function reflects transfers made to the offspring of others, including by younger siblings. This is similar to c), but depends on details of transfers.

In each case, predictions about the age pattern of mortality can be derived and, where life tables exist, tested. For species that make no investment in offspring after birth, the transfer function and the fertility function coincide, and the predictions of the classic theory and the present theory are identical (except for prepartum mortality).

Although the evolutionary implications of this theory might be difficult to test, cross-species empirical analysis could be useful[15]. The theory also has strong implications for relationships among observable age schedules of fertility, mortality, and transfers for any species. When age-specific birth and death rates are known, along with patterns of investment in young, comparisons like those for humans in Figure 5 might be revealing.

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Supplementary information is available on *Nature's* World-Wide-Web site (http://www.nature.com) or as paper copy from the London editorial office of *Nature.*

Mathematical Appendix

Let R′and B′ be partial derivatives with respect to γ, holding r constant; let r_R' and r_B' be partial derivatives with respect to γ, holding π constant for r_B'.

$$(4.1)\quad r_R' = \frac{R'}{A_f} = \frac{1}{A_f}\int_0^\omega e^{-rx}\left\{\frac{\partial l(x)}{\partial \gamma}m(x) + \frac{\partial m(x)}{\partial \gamma}l(x)\right\}dx$$

$$(4.2)\quad \begin{aligned} r_B' = {} & \frac{B'}{C(A_y - A_c)} = \frac{1}{C(A_y - A_c)}\int_0^\omega e^{-rx}\frac{\partial l(x,\gamma)}{\partial \gamma}\left[\pi\left(A/N\right)y(x,\gamma) - c(x,\gamma)\right]dx \\ & + \int_0^\omega e^{-rx}l(x,\gamma)\left[\pi\left(A/N\right)\frac{\partial y(x,\gamma)}{\partial \gamma} - \frac{\partial c(x,\gamma)}{\partial \gamma}\right]dx \end{aligned}$$

A_y and A_f are calculated A_c:

$$(4.3)\quad A_c = \frac{\int_0^\omega xe^{-rx}l(x,\gamma)c(x,\gamma)dx}{\int_0^\omega e^{-rx}l(x,\gamma)c(x,\gamma)dx}$$

The denominator in (4.3) is C, the discounted life time value of consumption, which must equal the similarly calculated πY.

Define F(a), remaining fertility above age a:

$$(4.4)\quad F(a) = \int_a^\omega e^{-rx}l(x,\gamma)m(x,\gamma)dx$$

And similarly T(a), cumulative survival weighted and discounted transfers received up to age a per new born:

$$(4.5)\quad T(a) = \int_0^a e^{-rx}l(x,\gamma)\left[\pi\left(A/N\right)y(x,\gamma) - c(x,\gamma)\right]dx$$

Fertility: differentiate R=1 and B=0, and simplify to find

(4.6) $$\frac{dr}{d\varepsilon(a)} = e^{-ra} l(a) / A_f + r_R' \frac{d\gamma}{d\varepsilon(a)}$$

(4.7) $$\frac{dr}{d\varepsilon(a)} = r_B' \frac{d\gamma}{d\varepsilon(a)}$$

Substitute out $d\gamma/d\varepsilon(a)$ and solve for $dr/d\varepsilon(a)$ to find (3.1).

Mortality: differentiate R=1 and B=0, and simplify to find

(4.8) $$\frac{dr}{d\delta(a)} = \frac{d\gamma}{d\delta(a)} r_R' + F(a) / A_f$$

(4.9) $$\frac{dr}{d\delta(a)} = T(a) / C(A_y - A_c) + \frac{d\gamma}{d\delta(a)} r_B'$$

Substitute out $d\gamma/d\delta(a)$ and solve for $dr/d\delta(a)$ to find (3.2).

Figure 1. Equilibrium of density and intergenerational transfers. Initial equilibrium is at X, which satisfies both renewal and balance equations at a positive r. Population density increases until it reduces productivity from π_1 to π_2, and then the balance curve intersects the renewal curve at Z, where r=0.

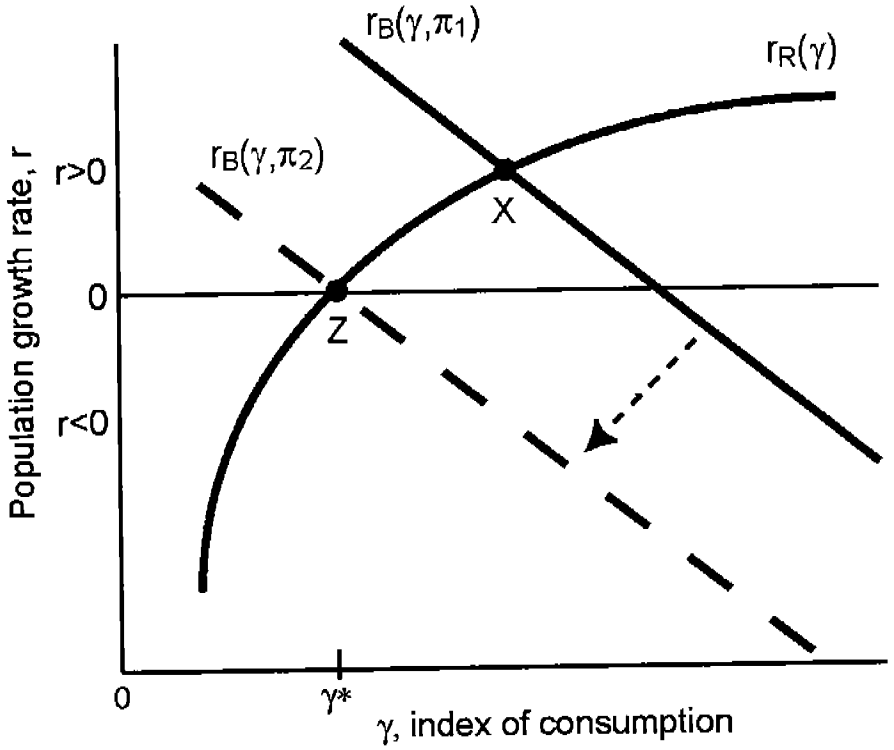

Figure 2. The effect on r and γ of a mortality-reducing mutation when the balance curve is downward sloping. The initial equilibrium is at Z. The mortality-reducing mutation gives the new intersection at X. The density adjustment of the balance curve restores a zero growth equilibrium at U. The vertical distance between Z and V is the classical effect. The need for transfer balance reduces this distance to that between Z and X.

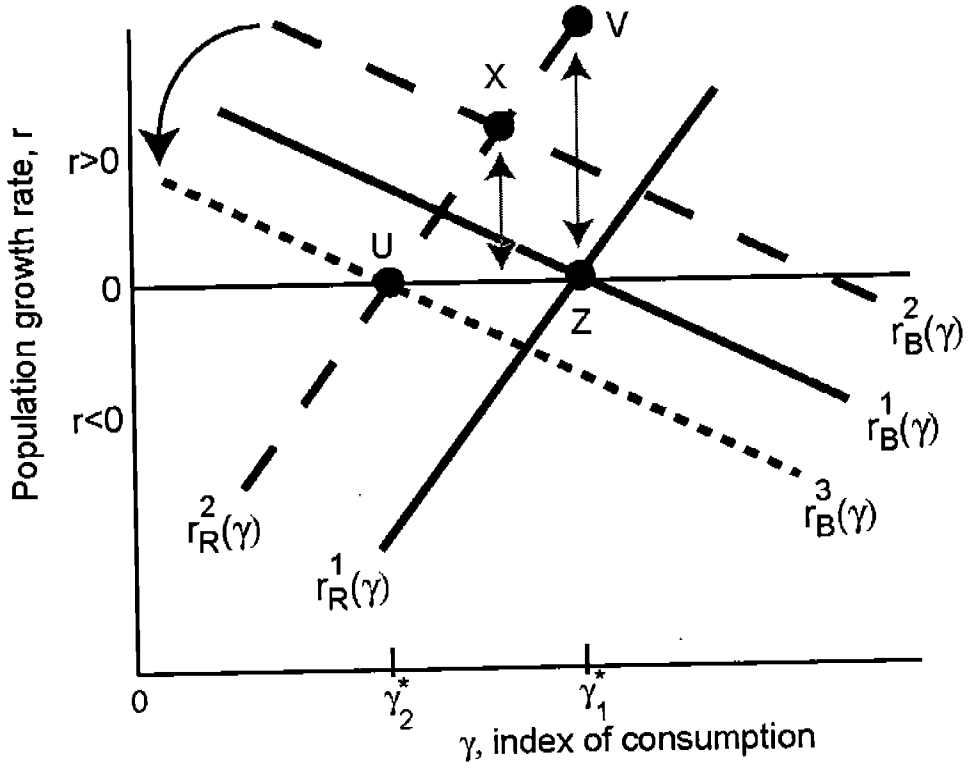

Figure 3. Selection of mutation on the left of a hump reverses the classical effects, moving toward the optimal equilibrium at the peak of the hump. Lower fertility or higher mortality shifts renewal down to $r_R^2(\gamma)$ to intersection at U at higher r, and is selected. Lower mortality shifting balance up to $r_B^2(\gamma)$ to intersect at V is also selected. Both simultaneously lead to X. The balance curve will then shift down for optimal equilibrium at r=0, not shown.

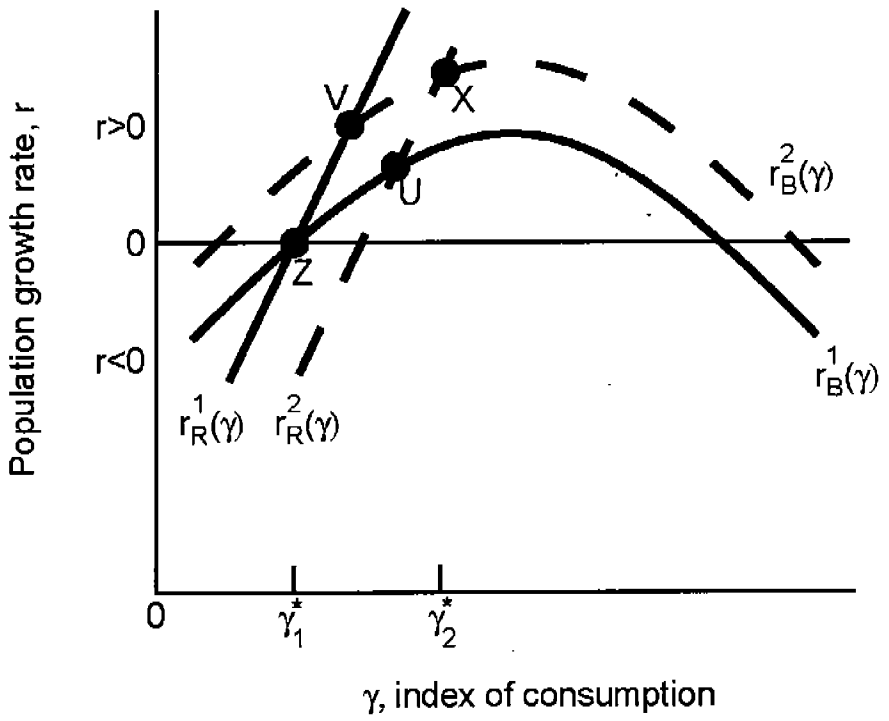

Figure 4. The force of selection on a mutation that reduces mortality at a specific age is a weighted average of the fertility effect and the transfer effect. Weights vary with slope of balance curve. The fertility effect equals $F(a)/A_f$. The transfer effect equals $T(a)/[C(A_y-A_c)]$. Based on data for Ache hunter-gatherer-horticulturalists (10, 11,16). When the balance curve is downward sloping, the weights on the fertility effect and the transfer effect are both positive, which is the case on the right side of a hump, and selection for juvenile survival is emphasized. When the balance curve is upward sloping, on the left side the weight on fertility is negative, and selection for adult survival is emphasized. At the peak, the fertility weight is zero, and only the transfer effect (bold line) matters.

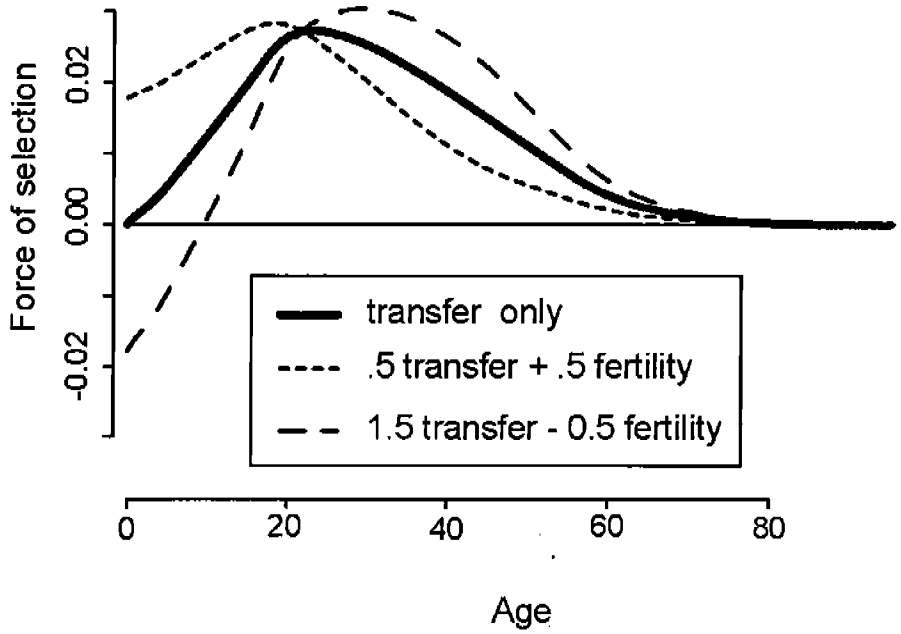

Figure 5. Comparison of actual inverted mortality schedules with the age-specific force of selection on mortality as given by the classical theory (remaining fertility, F(a)) and cumulated net investments or remaining transfers,T(a). Based on data for Ache hunter-gatherer-horticulturalists (10, 11,16). The chart plots F(a), T(a), and 1 divided by each age-specific death rate based on data for the forest-dwelling Ache, a well-studied contemporary hunter-gatherer-horticulturalist group (10,11,16). Also plotted is 1 divided by the age-specific death rates for 18th century Sweden. For theoretical force of selection, F(a) and T(a) are plotted. All curves are adjusted to have the same maximum at 1.0, since only shape is being compared.