Explaining the U-Shape of Age-specific Mortality

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Abstract

The age-shape of mortality is U-shaped for many species, declining from birth to sexual maturity, then rising in adulthood (Finch, 1990; for mammals, see Caughley, 1966). Although there are exceptions, the U-shape is sufficiently common to invite explanation. Here we show why the optimal life history of a species with determinate growth is likely to have this shape, building on a literature which showed these optimal patterns through numerical simulation (Cichon, 1997; Cichon and Kozlowski, 2000). Our approach assumes a physiological technology characterized by a linear budget constraint for energy at each age. We also incorporate intergenerational transfers, so that a young organism can allocate more energy than it produces. Using dynamic programming to solve the optimization problem, we find the forces shaping the optimal age-shape of mortality, and show the conditions under which it will be U-shaped.

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1 Introduction

A seminal paper by Hamilton (1966) argued that mortality must inevitably rise with age after sexual maturity, because mortality at older ages has an increasingly small effect on reproductive fitness, and therefore deleterious mutations which raise mortality at these ages will be selected out of the population less rapidly. Deleterious mutations with effects at older ages will therefore be present at a higher frequency in the population (mutation selection balance, see Charlesworth, 1994 and 2001) than those with effects at younger ages. Hamilton recognized two problems with this theory: first, it predicted constant mortality from birth until sexual maturity, rather than declining; and second, it predicted that mortality would rise rapidly following cessation of reproduction, whereas some species, including humans, have substantial postreproductive survival. The left arm of the U is therefore missing, and the right arm rises too early and too fast.

Building on Hamilton's approach, Lee (2003) formalized the idea, discussed by Hamilton and others, that parental care or more generally intergenerational transfers could deal with both these problems. Following birth, mortality at earlier ages would be offset to some degree by the resources thereby saved from future transfers, which could be used for fitness enhancing investment in siblings or in adults. Put differently, mortality would be selected to conserve the cumulative transfers already made to a juvenile, or equivalently, to conserve the expected net transfers to be made to others in the future. Thus mortality declines until sexual or economic maturity. At the same time, mortality in adult years affects fitness not only through lost future reproduction, as Hamilton emphasized, but also through lost future parental care or intergenerational transfers, an effect which diminishes with age but can continue for many years post reproduction, and may even include investment in grandoffspring.

The optimality literature mentioned earlier does not concern itself with genetic mechanisms, implicitly assuming that beneficial mutations will be positively selected whether their fitness impact is large or small, and for the most part ignoring the accumulation of deleterious mutations (see Barton and Partridge, 1993 for a clear discussion of the relation of the two approaches). Because optimality theory ignores the size (as opposed to the sign) of the fitness impact of a mutation, the mathematical results are different, and the qualitative conclusions can be different. Hamilton, and many after him, believed he had proven that senescence, marked by rising adult mortality, was universal and inevitable. With some qualification (Bauditsch, 2005; Lee, 2003) his conclusion appears broadly correct if the only genetic mechanism driving evolution is mutation accumulation. But his theory is not about the optimal life history, it is about the force of selection by age. Vaupel et al (2004) show that an optimal life cycle can exhibit flat or even declining adult mortality, or "negative senescence". This pattern can be optimal for species that exhibit indeterminate growth, that is do not have a specific mature body size, and instead continue to grow and reproduce concurrently as adults. They note that real world organisms exhibiting indeterminate growth may have this mortality pattern, including some plants and fish. For species with determinate growth, that grow to a mature size before reproducing and then switch to reproduction without further growth, they are unable to derive negative senescence. The Vaupel et al negative senescence result does not disprove the positive senescence result of Hamilton. A comprehensive theory would include both effects, and either could dominate depending on details of the assumptions.

In this paper, we concentrate specifically on the case of determinate growth, which characterizes mammals and birds, for example. We set up a model of optimal life history combining the effects of both growth and transfers, and analyze the optimal pattern of age-specific mortality rates that

maximizes fitness. We explicitly derive the benefits and costs of a change in age-specific mortality, identifying the conventional Hamilton effect, the compounded growth effect, the intergenerational transfer effect, and the cost-benefit tradeoff. We show why the optimal age-specific mortality will have declining juvenile mortality and is likely to have rising adult mortality, yielding a U-shape, and how, for species with transfers, the accumulated transfers affect the mortality schedule in youth and in old ages. We also compare our results with Robson and Kaplan (2003), who also derive a U-shape mortality schedule.

The remainder of this paper is arranged as follows. Section 2 presents the model of optimal life history, which we use to study the determination and change of age-specific vital rates. The third and fourth sections analyze the survival probability schedules for immature and mature ages respectively, and compare our results with the literature. The final section concludes.

2 A Model of Optimal Life History

An intergenerational transfer necessarily involves more than one generation, so our model of optimal life history explicitly characterizes a species' lineage. Details can be found in Chu and Lee (2005); here we only provide a sketch. Consider an individual who is not fertile past some age y.[2] To avoid the complications of mating and sexual reproduction, we consider a population of females reproducing asexually. To unify the terminology and notation, we call the age interval $[a, a+1)$ age $a+1$, and assume that all decisions affecting age $a+1$ are made at time a. The probability that an individual survives from point a toward point $a+1$ (that is, toward the end of the open age interval) is denoted p_{a+1}. Her fertility decision at age a produces m_a children

[2] y could be a very large number, and could also coincide with the upper bound of the species' life.

towards the end of her age a, conditional on her survival.

At age a, a typical individual expects to have energy or resources which, following Abram and Ludwig (1995), Cichon (1997) and Vaupel et al. (2004), she allocates to fertility (m_a), maintenance (p_a) and growth (z_a). The disposable food or energy acquired by an individual aged a depends on her body size, denoted w_a. Specifically, her age-a energy constraint is written as

$$b_a p_a + c_a m_a + d_a z_a \leq \zeta_a w_a, \quad \forall a \tag{1}$$

where b_a, c_a, d_a are constant coefficients, which express the rate at which energy can be used to achieve various levels of survival, fertility or growth. Term ζ_a is a production coefficient linking body-size with the net production, or acquisition through foraging, of disposable energy. The body size of an individual grows according to the rule: $w_{a+1} \equiv w_a + z_a$. The linear form of the budget constraint is also used in Vaupel et al. (2004).[3]

We expect that natural selection will maximize reproductive fitness, measured as the future representation of an individual's genes. Consider an individual age a at time t. Let $V_{a,t}(.)$ be her direct and indirect contribution to the number of descendants at some future date τ. Here t measures the remaining length of time until τ, when fitness is assessed, so for individuals closer to τ, t is smaller.

2.1 Solution and Interpretation

We begin by taking age 1 as a benchmark and trying to solve the dynamics in terms of its value function, $V_{1,.}(w_1)$. For any $a \in \{1, 2, ..., y\}$, let the age-a

[3]Appropriate nonlinear effects would include an upper bound of unity for p_a, with increasing costs as this limit is approached; and a direct dependence of both survival and fertility on on body weight w_a as in Vaupel et al (2004). Other nonlinearities might occur depending on whether optimization starts with a blank slate, or from a preexisting physiological structure.

strategy be $\theta_a \equiv (p_a, m_a, z_a)$ and its feasible set be $\Omega_a(w_a)$. For any t, the Bellman (1957) equations can be written as (2), for which the interpretation is provided in the Appendix.

$$\begin{aligned} V_{1,t}(w_1) &= \max_{\theta_1 \in \Omega_1(w_1)} [p_1 m_1 V_{1,t-1}(w_1) + p_1 V_{2,t-1}(w_1 + z_1)] \qquad (2) \\ &\vdots \\ V_{y-1,t}(w_{y-1}) &= \max_{\theta_{y-1} \in \Omega_{y-1}(w_{y-1})} [p_{y-1} m_{y-1} V_{1,t-1}(w_1) + p_{y-1} V_{y,t-1}(w_{y-1} + z_{y-1})] \\ V_{y,t}(w_y) &= \max_{\theta_y \in \Omega_y(w_y)} [p_y m_y V_{1,t-1}(w_1)]. \end{aligned}$$

We denote the optimum in (2) by $\theta_a^* = (p_a^*, m_a^*, z_a^*)$. Let $l_a^* \equiv p_1^* \cdots p_a^*$ and $\phi_a \equiv l_a^* m_a^*$. Starting from the equation for age-y, lagging each equation by one period, and substituting it into the equation in (2) one line above, we obtain the desired formulation in terms of $V_{1,\cdot}$:

$$V_{1,t} = \phi_1 V_{1,t-1} + \phi_2 V_{1,t-2} + \cdots + \phi_y V_{1,t-y}. \qquad (3)$$

In the steady state, expression (3) is a simple difference equation for $V_{1,t}$, of which the solution is $V_{1,t} = A_1 \lambda_1^t + \cdots + A_y \lambda_y^t$, where the A_i's are constants, and λ's are solved from the characteristic equation of (3): $\lambda^t = \phi_1 \lambda^{t-1} + \phi_2 \lambda^{t-2} + \cdots + \phi_y \lambda^{t-y}$. Since the individual is maximizing clonal reproduction, for large τ only the largest root of the characteristic equation is relevant, call it λ^* and normalize the associated A_i to be 1. Then we have: $V_{1,t} = (\lambda^*)^t \quad \forall t$ in the steady state.[4] In view of the definition of ϕ_a, we can rewrite (3) as the Euler-Lotka equation:

$$1 = \sum_{a=1}^{y} l_a^* m_a^* \lambda^{-a}. \qquad (4)$$

[4]The equation system in (2) can be rewritten in dynamic form as in McNamara (1991) and Houston and McNamara (1999), where it is shown that under some technical conditions the dynamics will converge to the stable-population λ^*.

The largest root is the steady state population growth rate. In summary, we know that the solution to the value function in (2) has the form $V_{1,t} = (\lambda^*)^t$, where λ^* is the Euler-Lotka parameter solved from (4).

There are two reasons why we propose the primal problem in (2) to study our problem. First, some researchers start by assuming that maximizing fitness is equivalent to maximizing expected life time reproduction, $R_0 = \sum_a l_a m_a$. However, $\sum_a l_a m_a$ is specific to the generation in question, and is inappropriate for an analysis of intergenerational interactions, which is part of our focus. Second, most previous literature on population evolution, to our knowledge, *assumes* that the Euler-Lotka parameter is the target of maximization. In the analysis above, we *derive* what a selfish individuals that maximizes its own clonal replication would do, and show that the objective to be optimized turns out to be the Euler-Lotka parameter. As we shall see below, the optimization conditions corresponding to (2) are natural and straightforward, and help us understand the benefit-cost tradeoffs in life history analysis.

2.2 The Determinate Growth Pattern

Many species exhibit "determinate growth", which is to say that they first grow without reproducing and then cease growth and become fertile once they have reached their adult size. Our main interest is in species that have intergenerational interactions, such as mammals or birds, and these exhibit determinate growth. It has been proved in Chu and Lee (2005) that the determinate growth pattern is optimal in our model: m_a and z_a cannot be interior solutions at the same time. This intuitive pattern was also found in Taylor et al. (1974) and Vaupel et al. (2004). If the switch occurs after r periods, then in our notation the organism would have $m_a = 0$ in the first $a \leq r$ periods, and would have $z_a = 0$ when $a \geq r + 1$.

3 Immature Mortality Trajectories

For most relevant cases, transfers are from a mature adult to an immature juvenile. We shall consider a transfer from a particular mature age $j \geq r+1$ to individuals at an immature age $i \leq r$. Later we expand the discussion to consider more than one age of making and receiving a transfer.

3.1 Specifying the Constraints

Suppose age j gives away T_j units of energy and age i receives R_i units of energy. Since we allow R_i to be zero, there is no loss of generality to assume that all immature ages ($i = 1, \cdots, r$) receive some R_i. With determinate growth, the energy of an age j adult that is not transferred is all allocated between p_j and m_j, so her budget constraint, according to (1), can be written as

$$m_j = \frac{\zeta_j w_j - b_j p_j - T_j}{c_j}. \tag{5}$$

At an immature age i, energy is all allocated between p_i and z_i, so the budget constraint can be written as

$$z_i = \frac{\zeta_i w_i - b_i p_i + R_i}{d_i}. \tag{6}$$

Recall that at each immature age i, body weight accumulates according to $w_{i+1} = w_i + z_i$. If an age-i receives an additional unit of energy transfer, using (6) iteratively we see that the *compound* effect on her mature body size (w_{r+1}) would be

$$\frac{\partial w_{r+1}}{\partial R_i} \equiv K_i = \frac{1}{d_i}\Big(1 + \frac{\zeta_{i+1}}{d_{i+1}}\Big) \cdots \Big(1 + \frac{\zeta_r}{d_r}\Big), \tag{7}$$

Leaving aside the first term $1/d_i$ in (7), we see that K_i is decreasing in i since each pair of parentheses contains a term greater than or equal to 1, and the number of such factors decreases as i rises. This means that

it is more efficient to accumulate somatic capital early than late, for the increased capital raises the output from foraging, which in turn leads to faster future growth. Alternatively, if K_i refers to neural capital or brain size, the compound factor K_i can be understood as an effect of learning by doing (see Robson and Kaplan 2003).

Let $g_i(.)$ be the technology for converting transfers into energy for a child at age-i . Specifically, in order for the age-i to receive R_i energy, the energy transferred by the senior should be $g_i(R_i)$. Here we assume that the converting technology is different for each *receiving* age so that we attach subscript i's to $g(.)$; the more general case would be that the technology differs for each receiving age *and* each transferring age, in which case $g(.)$ will have subscripts ij : $g_{ij}(.)$. We do not see how this complication would lead to new insights, so we ignore it.

The g_i function should satisfy $g_i(0) = 0$, and $g_i'(.) > 0$.[5] If the age-j adult transfers T_j of resources to juveniles of all ages, and juveniles at each age i receive R_{ij}, then in a stable population the following transfer identity must hold:

$$\frac{g_1(R_{1j})\lambda^{j-1}}{p_1 \cdots p_{j-1}} + \frac{g_2(R_{2j})p_1\lambda^{j-2}}{p_1 \cdots p_{j-1}} + \cdots + \frac{g_r(R_{rj})p_1 \cdots p_{r-1}\lambda^{j-r}}{p_1 \cdots p_{j-1}} = T_j. \qquad (8)$$

In the above expression, R_{ij} is allowed to be zero for each i. When more than one age makes transfers, the above formula should hold for each j, and (6) should be rewritten as

$$z_i = \frac{\zeta_i w_i - b_i p_i + \sum_j R_{ij}}{d_i}. \qquad (6')$$

The maximization in (2) can be written more briefly as:

$$\lambda^t = \max_{\theta_s} p_1 \cdots p_r [p_{r+1} m_{r+1} \lambda^{t-r-1} + \cdots + p_{r+1} \cdots p_y m_y \lambda^{t-y}], \qquad (9)$$

[5]If we hope to have an interior solution for R_i, then $g''(.)$ should be positive.

where λ is the Euler-Lotka parameter, and is also the age-1 *indirect utility* $V_{1,t}$ solved from (5) for sufficiently long time perspective. Substituting in m_j in (5) for $j \geq r+1$ and z_i in (6′) for $i \leq r$, we can derive the first order condition for age specific survival probability p_k's. We shall first present our discussion for the case of immature ages in this section.

3.2 The First Order Condition for p_k

Consider the most general case in which adults of all ages provide some non-negative transfers to juveniles of all ages. For a juvenile aged $k \leq r$ to maximize fitness, the first order condition for the optimal p_k, after factoring out a constant, is the following:

$$\Delta_{p_k} = M + \sum_{j=r+1}^{y} \frac{p_j}{c_j}\Big[\frac{\lambda^y g_1(R_{1j})}{p_1 \cdots p_r} + \cdots + \frac{\lambda^{y-k+1} g_k(R_{kj})}{p_k \cdots p_r}\Big] - p_k b_k K_k N = 0, \quad (10)$$

where Δ_{p_k} is the partial differentiation of the right hand side of (9) with respect to p_k,

$$M \equiv p_{r+1} m_{r+1} \lambda^{y-r} + \cdots + p_{r+1} \cdots p_y m_y \lambda,$$

and

$$N \equiv \Big[\frac{\zeta_{r+1} p_{r+1} \lambda^{y-r}}{c_{r+1}} + \cdots + \frac{\zeta_y p_{r+1} \cdots p_y \lambda}{c_y}\Big].$$

The interpretation of M, N, and K_k will be given as we proceed.

When we differentiate fitness λ with respect to p_k, as in (10) above, we are considering a movement of p_k along the efficient (maximal) trait surface, away from the optimum position on this surface where it is tangent to a fitness contour (Partridge and Barton, 1993). Because the movement is along the surface, the increase or decrease in p_k absorbs or releases energy and thereby affects growth, fertility or transfers, depending on whether k is pre or post sexual maturity. Because this movement is evaluated at the optimum, these

effects must exactly counterbalance the opportunity cost of the change in p_k, so that the derivative is zero. This is different from the deleterious mutational perturbations considered by Hamilton (1966) or Lee (2003), which resulted from an inefficient increase in mortality with no offsetting release of resources, and therefore no offset. This is a movement to the inside of the efficient trait surface, not along it. That is why the derivative equals zero in the present analysis, while the derivative would indicate a non-zero fitness impact in the Hamilton or Lee analysis. Real mutations could be of either sort, or of other kinds, and their consequences would vary accordingly.

If we totally differentiate (9) and arrange terms, we find $\Delta_\lambda d\lambda + \Delta_{p_k} dp_k = 0$. This equation gives us $d\lambda/dp_k$, the marginal impact of a change in age-specific mortality p_k on the fitness parameter λ. Note that Δ_λ is just a normalization term that is independent of k, involving some average ages, and therefore does not concern us here.

In (10), M is the weighted expected lifetime fertility, conditional upon survival to age $r+1$. This is closely related to Hamilton's fitness impact for a mortality perturbation at age k. It expresses the direct effect on fitness of the mortality change, but does not include the offsetting changes in other variables. Since M is independent of the immature age k in question, Hamilton concluded that the force of selection against a mortality increase is the same for all immature ages. For this reason, as he noted (p.12), his theory cannot explain the high infant mortality and declining juvenile mortality of many species.[6]

The second term in (10) gives the cumulated transfers expended per individual that attains age k, including the transfers wasted on other juveniles

[6]Note also that M is calculated conditional on survival to sexual maturity and the start of reproduction at age $r+1$. In Hamilton's result, the lost reproduction is weighted by survival from birth to each age, not from the age of maturity. This is an important difference between the fitness impact calculation and the optimality condition.

who died before reaching age k. This amount, which of course increases in age k, captures Lee's (2003) accumulated transfer effect. In particular, when k is smaller (larger), the fitness cost of "sunk" transfers up to age k is smaller (larger), hence an increase in mortality for age k is less (more) costly. Another way to think of this is that the death of a young individual releases the future resources that would have been transferred to it, thereby allowing for other uses such as investment in siblings or better nutrition for the adults, offsetting the loss. This offset is smaller at older juvenile ages, and therefore mortality at these ages is more costly. Selection therefore will be stronger against the mortality of the elder juveniles relative to that of infants, and optimal mortality will be lower at these older juvenile ages.[7]

The first two terms in (10) capture the two benefits of a mutation that exogenously raises p_k. In the context of beneficial mutations and optimal life history, as opposed to deleterious mutations and mutation accumulation theory, the increase in p_k has an opportunity cost, as described by (6′). This tradeoff cost is ignored under the mutation accumulation approach, because deleterious mutations are assumed to be inefficient, yielding no benefits elsewhere, as discuseed earlier. But varying p_k along the efficient surface entails offsetting costs or benefits. For an age-k juvenile to slightly increase her survival probability by dp_k, she must decrease the energy allocated to the accumulation of her body weight by $b_k(dp_k)$. This reduction in energy from growth will in turn shrink her mature size (w_{r+1}) by K_k according to (7), which we discussed earlier. For each unit reduction in mature size w_{r+1}, the impact on fitness (force of selection) is the expected reduction in weighted

[7] Alternatively, to avoid "sunk cost" fallacies, terms in the square brackets of (10) can be thought of as the expected future net life time transfers to be made by an incividual age k, suitably survival weighted and discounted by the population growth rate. The bigger is k, the bigger will be these expected net transfers, and the bigger the fitness gain from raising p_k.

lifetime fertility, and that is in fact the N term:

$$N \equiv \frac{\partial \sum_{j=r+1}^{y} p_j \lambda^{y-j+1} m_j}{\partial w_{r+1}} = \frac{\partial \sum_{j=r+1}^{y} p_j \lambda^{y-j+1} \frac{\zeta_j w_{r+1} - b_j p_j - T_j}{c_j}}{\partial w_{r+1}}$$

Finally, the accumulation of weight at age k will be realized only with probability p_k, and this explains the third term in (10).

3.3 Searching for the Optimal Juvenile p_k

Equating Δ_{p_k} to zero, we get the first order condition of p_k. It can be easily seen that $\partial \Delta_{p_k} / \partial p_k < 0$, hence the second order condition for maximization is satisfied. Simultaneously solving for $(p_1, \cdots, p_r)$ explicitly would be tedious, but for the purpose of comparing the shape of p_k's for different k's, we can rewrite $\Delta_{p_k} = 0$ as follows:

$$p_k = \frac{M + \sum_{j=r+1}^{y} \frac{p_j}{c_j} \left[\frac{\lambda^y g_1(R_{1j})}{p_1 \cdots p_r} + \cdots + \frac{\lambda^{y-k+1} g_k(R_{kj})}{p_k \cdots p_r} \right]}{b_k K_k N}, \tag{11}$$

Note that (11) is not an explicit reduced-form solution for p_k, for the right hand side of (11) still has p_k in it. However, it helps us understand how p_k varies with age, k. We know from the above discussion that the numerator of (11) is increasing in k due to the transfer effect, but what about the denominator?

In view of (7) the denomenator of (11) can be written as

$$K_k b_k N = \frac{N b_k}{d_k} \left[\left(1 + \frac{\zeta_{k+1}}{d_{k+1}} \right) \cdots \left(1 + \frac{\zeta_r}{d_r} \right) \right].$$

Evidently, terms in the square bracket of this expression are non-increasing in k, for the compounded effect of accumulating size becomes smaller as age increases, since then compounding operates over fewer periods. Note that this important effect, which we have not found in the existing literature, does not depend on transfers, and therefore helps to explain why mortality declines

following birth in species that do not have parental care. Here is a different way to think about it. Reproductive success depends on both mature size and survival to maturity. Survival from birth to maturity is the product of the survival probabilities at each age, and the order of multiplication is irrelevant. But in the case of body size, early growth is more beneficial because the larger size that results permits more production of energy which leads to more growth or higher survival, and so on. This is the compounding effect. The optimal strategy, therefore, will involve sacrificing some early survival for more rapid growth, and then improving survival later when resources are more abundant. Gaining weight, like receiving transfers, has a cumulative effect, but compounding of growth applies to the cost side whereas the compounding of transfers applies to the benefit side. Of course, if the juvenile simply cannot forage or hunt (with $\zeta_a = 0\ \forall a \leq r$), then the compounding effect of growth disappears.for the ages in question. In this case, however, the juvenile must be receiving transfers of energy from adults, so that declining mortality is assured through the Transfer effect. One or the other or both must be present.

We can view the process of gaining weight as a production process, transforming energy into tissue and flesh, with an efficiency that may vary with age. The energy cost of achieving a given weight gain per unit time might at first be high, since the organism is initially small, so the proportional increase would be great. When it becomes larger, the proportional increase is smaller, and therefore might be achieved more efficiently. But as the individual approaches its mature size, adding weight might again become more costly. Such considerations suggest an S-shaped pattern for weight-gaining efficiency. However, since the correspondence between age, size and maturity is not known until the optimization problem is solved, it is not appropriate to posit any nonmonotonic age variations in any of the parameters. For this reason we will not consider variation in d_k as an influence on the age pattern

of juvenile mortality.

Likewise, it is difficult to draw any conclusions about the age pattern of the efficiency of allocations to maintenance and survival, b_k. Body size will grow with age until age r, but r is not known until the optimization is solved. Larger body sizes may have lower costs of avoiding predation, but larger maintenance costs due to the increased number of cells and cell replications.

3.4 The Forces Shaping Juvenile Mortality

Now let us pause to take stock and interpret what we have learned. We have identified four forces that influence the shape of p_k for immature ages.

1. The Hamilton Effect: the term M representing expected future reproduction conditional on reaching maturity, which is independent of k and so does not affect the shape of juvenile mortality.

2. The Transfer Effect: $\sum_{j=r+1}^{y} \frac{p_j}{c_j}\left[\frac{\lambda^y g_1(R_{1j})}{p_1 \cdots p_r} + \cdots + \frac{\lambda^{y-k+1} g_k(R_{kj})}{p_k \cdots p_r}\right]$, which is increasing in k, and therefore provides a reason to expect that juvenile mortality will decline with age, in species with parental care or other intergenerational transfers.

3. The Compounded Effect of Growth: $1/\left(1+\frac{\zeta_{k+1}}{d_{k+1}}\right)\cdots\left(1+\frac{\zeta_r}{d_r}\right) \equiv 1/K_k$, which is increasing in k, and leads us to expect declining juvenile mortality whether or not species have parental care and intergenerational transfers.

4. Unknown variations with age of the energetic costs of growth and/or survival. However, there seems no clear reason to expect these to vary in one way or another, so we set them aside.

Now we will consider these results in relation to the literature. The first effect was emphasized by Hamilton (1966) as discussed earlier. The second

effect was emphasized by Lee (2003), but it also corresponds partially to the "sibling replacement effect" discussed informally by Hamilton (p.40). The third effect we believe is new, and important. Others have linked mortality risks to size, and therefore found that mortality declines with growth, but our argument is more subtle. The effects considered fourth are unknown, but unless powerful, would be overwhelmed by the others. Under a different setting, Robson and Kaplan (2003) also derived a declining mortality schedule in young ages for species having a "learning-by-doing" property in their age-specific production profile. Specifically, they assumed that the age-output profile is hump-shaped (assumption 5), which in turn implies an energy deficit during childhood and old age, and an energy surplus during middle ages. The corresponding Fisher's reproductive value is therefore hump-shaped. This facilitates the U-shape mortality, for an optimal life history must secure high reproductive value through low mortality. The model they presented, however, does not have an explicit biological immature period prior to reproduction, and hence cannot be compared with the Hamilton result directly.[8] Our results, evidently, do not depend on any assumptions about a hump-shape for age-specific output ($\zeta_a w_a$). For instance, it may be the case that the species in question is not productive at all in immature ages so that $\zeta_a = 0 \ \forall a \leq r$. Even in this case, we are able to generate a declining age-specific mortality result as long as the juveniles receive a sufficient amount of transfers.

[8]For the case of primates and human beings in particular, Robson and Kaplan (p. 160) did assume a pattern of neural capital accumulation similar to the scenario of determinate growth.

4 Mature Mortality Trajectories

Now we consider the p_k trajectory for mature (adult) ages $k \geq r+1$. Using identity (8), we see that the first order condition for maximizing (9), after factoring out a constant, is the following:

$$\Delta_{p_k} = M_k + \sum_{j=k+1}^{y} l_j \lambda^{y-j+1} T_j / c_j - \frac{b_k}{c_k} \lambda^{y-k+1} l_k p_k = 0, \tag{12}$$

where

$$M_k \equiv \sum_{j=k}^{y} l_j \lambda^{y-j+1} m_j$$

is the weighted future fertility effect after age k, similar to the Hamilton effect M in (10) but varying with k, and T_j is the transfer made at age j. The two summation terms start at different ages ($j = k$ or $j = k+1$) because we assume fertility is realized toward the end of each period, while transfers are realized at the beginning of each period. Once having reached age k, only later ages (j greater than k) can possibly contribute to the benefit of surviving past age k, hence the summations in (12) start at $j = k$. All these future fertility and transfer effects have to be added up as we consider the benefit of a change in p_k.

4.1 The Forces Shaping Adult Mortality

Again we will pause to consider the forces at work, which are closely related to those for juvenile mortality, but with quite different implications. The first two are fitness benefits of increased survival, whereas the third is the cost of increasing survival.

1. The Hamilton Effect: the term M evaluates expected future net fertility conditional on reaching maturity. But unlike for juveniles, here M necessarily declines monitonically toward 0 as age increases, indicating that it is progressively less beneficial to allocate resources to survival.

2. The Transfer Effect: the total expected transfers (survival weighted and discounted by population growth) that remain after age k, which necessarily declines monotonically toward 0 as age increases, provided that adults are not net receivers of transfers at any age. Note that this may remain positive after cessation of reproduction, providing continuing benefits to postreproductive survival. It is divided by the cost of fertility at each age to convert the energy involved in the transfers into units of fertility.

3. The Fertility Cost of Increasing Survival: The energy tradeoff is b_k/c_k, which is survival weighted and discounted.

4. Unknown variations with age of the energetic costs of survival relative to fertility, b_k/c_k. There is no clear reason to expect this ratio to vary in one way or another. Unless very large, such variations would be overwhelmed by the other factors.

The sum of the first and second terms is the total future benefit which an individual protects by sustaining survival. The third term is the opportunity cost of p_k. As one can see from (5), increasing p_k can be achieved only by reducing m_k. The term $b_k\lambda^{y-k+1}/c_k$ characterizes the costs of p_k relative to m_k, which is multilied by p_k because fertility is realized toward the end of the age interval k after survival. The cost term is multiplied by $p_1 \cdots p_k \equiv l_k$ because we have done so for terms associated with the benefit part. Note that the cost term is also decreasing in k, partly offsetting the declining pattern of the benefit side.

4.2 Optimal Adult Mortality

Equating Δ_{p_k} in (12) to zero, we derive the following characterization of $p_k$which is not a reduced form, but which is nonetheless helpful.

$$p_k^* = \frac{\left[\sum_{j=k}^{y} l_j m_j \lambda^{y-j+1} + \sum_{j=k}^{y} l_j T_j \lambda^{y-j+1}/c_j\right]/l_k}{b_k/c_k}, \tag{13}$$

As explained, the first term in the square parentheses is the Hamilton effect, and the second term the transfer effect, and both decrease with age, k. The difficulty in drawing a firm conclusion about whether survival falls with age and mortality rises, is that l_k divides the square brackets, and it is decreasing in k. Optimization at each age is forward looking, and conditional on survival to that age, which is why l_k appears in (13). We will now consider whether the Hamilton and transfer effects are declining faster or slower with age than l_k.

We know from (5) that $m_j + T_j/c_j = (\zeta_j w_{r+1} - b_j p_j)/c_j$, so (13) can be written as

$$p_k^* = \frac{\left[\sum_{j=k}^{y} l_{kj} \lambda^{y-j+1} (\zeta_j w_{r+1} - b_j p_j)/c_j\right]}{b_k/c_k}, \tag{14}$$

where $l_{kj} = p_{k+1} \cdots p_j$. Note that the dynamic programming problem in (2) for mature ages can be written explicitly as

$$\max_{\Omega_{r+1}}\Big[p_{r+1} m_{r+1} \lambda^{t-r-1} + \max_{\Omega_{r+2}}\Big(p_{r+2} m_{r+2} \lambda^{t-r-2} + \cdots + \max_{\Omega_y}(p_y m_y \lambda^{t-y})\Big)\Big]. \tag{15}$$

The set Ω_k for mature ages in the above problem is characterized by

$$b_k p_k + c_k m_k \leq \zeta_k w_{r+1} - T_k. \tag{16}$$

As one can see, the numerator on the right hand side of (14) is in fact the maximum value obtained from the remaining optimization problem in (15) starting from any age k, which we denote $B(k)$. We shall argue below that $B(k)$ is decreasing in k.

We note from (15) that the k-forward problem is symmetric for each k except: 1) the terms to be added in the maximand decrease when k increases, and 2) the feasible set Ω_k may be different across k. The first factor certainly causes the maximum value of the k-forward problem to decline as k increases. Thus, we can conclude that the numerator of (14), $B(k)$, is decreasing in k, provided that the feasible set Ω_k does not increase as k increases. This condition is met if either the $\zeta_k w_{r+1} - T_k$ is not increasing,[9] or the cost of maintaining survival and reproducing (b_k, c_k) is not decreasing, or both. There are various reasons to expect b_k, c_k to increase in k: wear and tear on the organism as it ages, the accumulation of somatic mutations with age, and the accumulation of mutations in the germ line that lead to less efficient physiology in old age. What is not known is the pattern of $\zeta_k w_{r+1} - T_k$.

There are two interesting cases we can consider for the shape of $\zeta_k w_{r+1} - T_k$. First, suppose there are no transfers, or transfers are small in the relevant range of k. Then $\zeta_k w_{r+1} - T_k \approx \zeta_k w_{r+1}$. Second, since we do not have any a priori information concerning T_k, we may assume that the age-k transfer is a fixed proportion of age-k energy: $T_k = \gamma \zeta_k w_{r+1}$, where γ is a constant. In this scenario, $\zeta_k w_{r+1} - T_k = (1 - \gamma)\zeta_k w_{r+1}$. In either case, a sufficient condition for the feasible set to be non-expanding is that ζ_k is not-increasing in k, which also seems reasonable for old ages for the same reasons given for non-decreasing b_k, c_k. In sum, in either of the above two cases, as long as the efficiency of energy production through foraging ζ_k does not increase with age, and neither does the efficiency of achieving survival or fertility through expenditure of energy (which is inversely related to b_k, c_k), we know that $B(k)$, and hence the numerator of (14), should be decreasing in k. Thus, unless the ratio b_k/c_k in the denominator decreases more rapidly than the numerator, due to a more rapid decline in the energy efficiency of fertility

[9] In Vaupel et al. (2004), they have indeterminate growth, so that w_k may increase even after maturity. This will expand the feasible set Ω_k, and hence "negative senescence".

than of survival, we can conclude that p_k.decreases with age, and therefore that mortality is rising with age We have no reason to expect any particular trend in the ratio b_k/c_k, let alone a strong one.

For general transfer patterns, the budget constraint in (16) is $b_k p_k + c_k m_k \leq \zeta_k w_{r+1} - T_k$. A sufficient condition to tell the shape of the numerator of (14) is a non-increasing pattern for $(\zeta_k w_{r+1} - T_k)/c_k$. However, one should note that this condition is far stronger than what is needed to have the numerator of (14) decline as k increases, even if $(\zeta_k w_{r+1} - T_k)/c_k$ increases, the effect of decreasing number of terms has to be offset for the maximum value to be increasing.

4.3 The Transition from Juvenile to Mature

Now we briefly discuss the intersection age where maturity and immaturity meet. We argued in section 2 that the species in question will never choose $m_a > 0$ and $z_a > 0$ at the same time. This can be seen as follows. Applying the envelope theorem to (2), we see that in a steady state

$$V'_{a,t} = p^*_a\{\zeta_a \lambda^{t-1}/c_a + p^*_{a+1}[\zeta_{a+1}\lambda^{t-2}/c_{a+1} + p^*_{a+2}(\zeta_{a+2}\lambda^{t-3}/c_{a+2} + ...]\}.$$

For the age-a problem, concerning the trade-off between m_a and z_a, we have the following first order condition (in terms of economics, MRS equals price ratio) for an interior solution:

$$\frac{\lambda^{t-1}}{p^*_{a+1}\{\zeta_{a+1}\lambda^{t-2}/c_{a+1} + p^*_{a+2}[\zeta_{a+2}\lambda^{t-3}/c_{a+2} + p^*_{a+3}(\zeta_{a+3}\lambda^{t-4}/c_{a+3} + ...)]\}}$$
$$\equiv \text{MRS}_a = \frac{c_a}{d_a}. \tag{17}$$

We see that both sides of (17) are not dependent on any age-a choice variable. Thus, expression (17) could hold only by accident in a steady state, which in turn implies that m_a and z_a cannot be interior solutions at the same time.

If ages are refined to very small intervals, then around the borderline where the immature and mature is divided, the parameters c_a, d_a and ζ_a do not differ much for close enough a's. Applying (17), one can see that the survival probabilities for age r and $r+1$ are connected. The shape of p_a for mature and immature ages are respectively dominated by the formula in (11) and (13).

5 Conclusions

The characteristic U-shape of mortality for many species invites explanation. First, why does mortality decline at the start of life? Second, why does mortality rise in adulthood? Third, why is there postreproductive survival? Hamilton (1966) provided an answer to the second question, now understood to be based on the evolutionary genetic mechanism of mutation accumulation. However, his approach failed to explain either declining juvenile mortality or postreproductive survival, and it did not apply to species that make intergenerational transfers (Lee, 2003). Even his apparently definitive answer to the second question has been shown to fail to hold when the theoretical approach is optimization of the life history, and the dominant genetic process is assumed to be positive selection of beneficial mutations (Vaupel et al, 2004). The optimal life history for species with indeterminate growth can include declining adult mortality.

We have limited our analysis to the case of determinate growth, which arises naturally from our assumed linear energy constraint. We have also focused on the shape of the optimal life history, while ignoring the influence of accumulated deleterious mutations, although this could be readily included, leading to systematic departures of the evolved life history from its optimal shape, causing the *optimal* adult mortality to rise more rapidly. At the same time we have broadened our analysis to include physiological tradeoffs, a ne-

cessity for the optimality approach, and to allow intergenerational transfers, which has not previously done in the optimality literature except in Chu and Lee (2005). Our dynamic optimization approach also differs from the previous literature by starting from the primal objective of maximizing the genetic representation at a future date, from which we derive the intrinsic growth rate (Euler-Lotka parameter) as the appropriate meaure of fitness. Elsewhere we have shown that the common procedure of taking expected births (the Net Reproduction Ratio) as the fitness goal yields incorrect comparative static results (Chien et al, 2005).

Given this setup, we have derived a number of important new results.for the optimal shape of mortality for species with deterministic growth, and with or without intergenerational transfers. We have shown that juvenile mortality must decline from birth to maturity, due either to protection of increasing cumulative investments in juveniles (as Lee, 2003, found in the mutation accumulation context) or to the advantages of early investment in growth at the expense of survival, due to compounding effects, or to both. At least one of the two effects must be operative at any age, so juvenile mortality decline is assured. For adult mortality, we have shown that it must increase steadily following maturity in species without transfers or with small transfers, due to a weakened version of the Hamilton effect–weakened in the sense that in the optimal history, the decline in remaining expected fertility after a given age is partially offset by the decline in proportions surviving to that age, which is not the case for the accumulation of deleterious mutations. When a species makes intergenerational transfers, then there is postreproductive survival. Here also declining future transfers tends to make adult mortality rise, but again this effect is weakened by the decline in proportions surviving to any given age. Although we think declining adult mortality in the case with large transfers is unlikely, a definite conclusion of such cannot be reached due to the decreasing probability of surviving to older ages.

In sum, we have shown how changes in remaining fertility and cumulated transfers operate differently in the optimization context to shape mortality patterns, we have seen how physiological tradeoff costs qualify these effects, and we have identified a new force, the compounding power of growth. We conclude that these forces combine to produce a U-shaped mortality pattern, although the possibility of declining adult mortality in some circumstances cannot be ruled out due to the declining probability of surviving to any age.

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Appendix: Interpreting Equation (2)

The interpretation of equation (2) is as follows: $p_1 m_1$ in the first term on the right hand side of (2) characterizes the event that an age-1 individual survives (with probability p_1) and bears m_1 offspring. Since each of these offspring is valued $V_{1,t-1}$ in period $t-1$ (because the offspring is one period closer to τ), $V_{1,t-1}$ should be multiplied by $p_1 m_1$ to obtain the expected value. The $V_{2,t-1}$ in the second term of (2) is the value function of this individual at age-2. With probability p_1 the individual will survive to face this state, and so $V_{2,t-1}$ should be multiplied by probability p_1. The age-2 body size should be $w_2 = w_1 + z_1$ instead of w_1. The interpretations of other expressions are similar, so we move forward to the last equation. For an individual aged y in period t, p_y and m_y are chosen to maximize the expected value of the last birth. At age y, there is no gain from further growth.[10] This generates the age-y expected value $p_y m_y V_{1,t-1}$. Since y is the last fertile age by assumption, there is no second term for the last equation.

[10] The growth of a post-reproductive individual might still be valuable if she could transfer some wealth to her young offspring, but for the time being we are considering the case in which this is not possible, reflected in the budget constraint in equation (1). See also Lee (2003).