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Explaining the Optimality of U-Shaped Age-Specific Mortality

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Abstract

Mortality is U-shaped with age for many species, declining from birth to sexual maturity, then rising in adulthood, sometimes with postreproductive survival. We show analytically why the optimal life history of a species with determinate growth is likely to have this shape. An organism allocates energy among somatic growth, fertility and maintenance/survival at each age. Adults may transfer energy to juveniles, who can then use more energy than they produce. Optimal juvenile mortality declines from birth to maturity, either to protect the increasingly valuable cumulative investments by adults in juveniles or to exploit the compounding effects of early investment in somatic growth, since early growth raises subsequent energy production, which in turn supports further growth. Optimal adult mortality rises after maturity as expected future reproduction declines as in Hamilton, but intergenerational transfers lead to postreproductive survival as in Lee. Here the Hamilton and transfer effects are divided by probabilities of survival in contrast to the fitness impact measures, which are relevant for mutation selection balance. If energetic efficiency rises strongly with adult experience, then adult mortality could initially be flat or declining.

Keywords and phrases

optimal life history; evolution of mortality; intergenerational transfers; parental care; parental investment; postreproductive survival; infant mortality; senescence

1 Introduction

Age-specific mortality is U-shaped for many species, declining from birth to sexual maturity, then rising in adulthood, possibly after some delay and sometimes with post-reproductive survival (Finch, 1990; Caughley, 1966; Promislow, 1991; Sibly et al., 1997; Gage, 1998). Although there are exceptions, this 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), and including the possibility that there are intergenerational transfers.

A seminal paper by Hamilton (1966), formalizing Williams (1957), argued that mortality must inevitably rise with age after sexual maturity, because mortality at older ages has a diminishing effect on reproductive fitness, and therefore deleterious mutations which raise mortality at these ages will be selected out of the population less rapidly (this application of his argument to

deleterious rather than beneficial mutations was a later development). Therefore mutations affecting older ages will be present at a higher frequency in the population in mutation-selection balance than mutations which affect younger ages (Charlesworth, 1994, 2001). Hamilton recognized two problems with this theory. First, it predicted constant rather than declining mortality from birth until sexual maturity; and second, it predicted a rapid increase in mortality 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) extended and formalized an idea discussed by Hamilton, Williams and others that parental care or, more generally, intergenerational transfers could explain both these features of mortality. Following birth, older relatives transfer resources to the juvenile, and survival will be selected to conserve the quantity of cumulated transfers already made to a juvenile (which grows with age); or, equivalently, to conserve the expected net transfers to be made by this individual to others in the future. Thus, mortality declines from birth until sexual or economic maturity. At the same time, mortality in adult years affects fitness not only directly through lost future reproduction, as Hamilton emphasized, but also indirectly through lost future parental care or intergenerational transfers, an effect which diminishes with age but can continue for many years after fertility ceases and may include investment in grandoffspring. Both Hamilton's and Lee's analyses were based on fitness impacts and the accumulation of deleterious mutations rather than on optimality, tradeoffs and selection of beneficial mutations.

The mutation accumulation approach describes certain relationships that should hold among life history traits in mutation-selection balance, but it cannot by itself tell us much about the age schedules of the traits themselves. There is an inherent circularity: if we know the age schedule of fertility under Hamilton's theory, then we can infer some features of the shape of the age schedule of mortality; and if we know the age schedule of mortality, then we can infer something about the age schedule of fertility; but none of this enables us to construct or explain actual life histories unconditionally. Furthermore, this approach assumes that mutations are purely deleterious, ignoring tradeoffs and pleiotropic effects.

The optimality approach, by contrast, starts with assumptions about physiological and energetic constraints and from these seeks to construct an optimal life history (Taylor et al., 1974; Goodman, 1982; Schaffer, 1983; Stearns, 1992; Abrams and Ludwig, 1995; Cichon and Kozlowski, 2000, Clark and Mangel, 2000, Robson and Kaplan, 2003, and Houston and McNamara, 1999). It is constructive and intuitive. At the same time, it is not concerned with genetic mechanisms, implicitly assuming that beneficial mutations will occur and be positively selected whether their fitness impact is large or small. For the most part it ignores deleterious mutations. (See Partridge and Barton, 1993, for the relation between the two approaches). While pleiotropic effects of genes may reflect physiological tradeoffs, they may alternatively reflect accidental linkages of different traits on the same gene. When effects occur through genetic linkage, we would expect other mutations to occur and be selected so as to counteract these effects and move the organism toward the efficient physiological tradeoff frontier (Partridge and Sibley, 1991).

Because of these differences in assumed genetic mechanism (removal of deleterious mutations versus positive selection of beneficial ones), mathematical results from the optimal life history approach and from that of fitness impacts can be different, and their qualitative conclusions can be different as well. Hamilton, and many after him, believed he had proven that senescence was universal and inevitable. With some qualification (Baudisch, 2005; Lee, 2003), his conclusion would be broadly correct if the only genetic mechanism driving life history evolution were mutation accumulation. But his theory is not about the optimal life history, it

is instead about the force of selection by age, which is a different matter. Vaupel et al. (2004), following Williams (1957) and others (reviewed by Heino and Kaitala, 1999), show that for a species with indeterminate growth (continuing to grow significantly after sexual maturity), an optimal life cycle can exhibit flat or even declining adult mortality ("negative senescence"). Vaupel et al. note that real world organisms exhibiting indeterminate growth may have this mortality pattern, including some plants and fish. For species with determinate growth (that is, reaching a mature size before reproducing and then switching to reproduction with little or no further growth) Vaupel et al. are unable to derive negative senescence. The Vaupel et al. result on optimal negative senescence need not conflict with the positive senescence result of Hamilton under mutation accumulation. A comprehensive theory would include both effects, and either could dominate in theory or in nature, depending on the frequency and impact of beneficial and deleterious mutations and other details.

Here, taking the optimality approach, we find anew the central features of the Hamilton and Lee results, but now they are conditional on survival to each age (i.e., divided by l_x) in contrast to mutation accumulation results. Hamilton criticized Fisher (1930) on this point, since terms in Fisher's Reproductive Value are divided by l_x at each age, unlike Hamilton's fitness impact. Both can be right, depending on the genetic mechanism under consideration. We also find other forces at work shaping the age schedule of mortality, including the greater advantage of early investment in somatic growth within the juvenile stage versus less or no advantage to early scheduling of investment in survival.

In this paper, we will concentrate specifically on the case of determinate growth, which, for example, characterizes mammals and birds. We set up a model of optimal life history combining the effects of fertility, growth, survival and transfers and analyze the optimal pattern of age-specific mortality 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 juvenile mortality will optimally decline and adult mortality will optimally rise, yielding a U-shape, and how for species that make transfers, the accumulated value of transfers affects optimal mortality in youth and old age. We compare our results with Robson and Kaplan (2003), who also derive a U-shaped mortality schedule.

Mangel and Munch (2005) developed a model of compensatory growth which included a tradeoff between the advantages of larger size for survival and reproduction and the costs of more rapid growth due to damage at the cellular level. They found that within a "nonreproductive period in the life history" a U-shaped mortality schedule was optimal for some parameter settings. Within such a period, remaining reproduction does not change, so Hamilton's theory cannot explain mortality increase, and, because transfers are not included in the model, the transfer theory does not explain either initial mortality decline or subsequent increase. The forces at work here are not incorporated in our theory, and conversely.

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 mortality schedules for juvenile and adult ages respectively and compare our results with the literature. The final section concludes.

2 A Model of Optimal Life History

2.1 The Autarchic Case: No Intergenerational Transfers

To avoid the complications of mating and sexual reproduction, we consider a population reproducing asexually. Let μ_a be the instantaneous mortality rate at age a. The probability that

an individual survives from birth to age a is denoted ℓ_a. By definition,ℓ_a is given by $\exp\left(-\int_0^a \mu_x dx\right)$. The fertility rate of an individual at age a is denoted m_a.

At age a, a typical individual expects to have energy or resources which, following much of the literature (Partridge and Sibly, 1991; Abrams and Ludwig, 1995; Cichon, 1997; and Vaupel et al., 2004) she allocates to fertility (m_a), maintenance (μ_a) and growth (z_a). The disposable food or energy acquired by an individual aged a depends on her body size, denoted w_a.

Most animals neither store food outside their bodies nor invest in external productive capital, although there are exceptions.[2] Some degree of intertemporal reallocation can be accomplished by storing energy somatically as body fat, but our model will ignore this possibility for simplicity. Credit markets do not exist in nature, although in some social species reciprocal altruism may permit some temporal exchange.[3] Intergenerational transfers, and particularly parental care, are sometimes very important, and we will explicitly model them in the next section. Here, we will begin with the case in which there are no transfers and therefore assume that an animal's energy budget at a given time is constrained by the energy it acquires at that time. Specifically, the energy constraint at age a is written as

$$f_a(\mu_a, m_a, z_a) \leq \zeta_a(w_a), \quad \forall a. \tag{1}$$

Let $f_{a,\mu}$, $f_{a,m}$ and $f_{a,z}$ denote the partial derivatives of f_a w.r.t. μ_a, m_a and z_a, respectively. These coefficients express the rate at which energy can be used to effect marginal changes in the levels of mortality, fertility or growth. The derivative $\zeta_{a,w} \equiv \zeta'_a$ is a production coefficient linking the marginal effect of body size to the net acquisition of disposable energy through foraging. The body size of an individual grows according to the rule: $dw_a/da = z_a$.

Mortality μ_a is the instantaneous hazard of death (the force of mortality), defined as $d \ln l_a = da$ where l_a is the probability of surviving from birth to age a, bounded between *0* and *1.0*. μ_a is bounded between 0 and infinity. Evidently $f_{a,\mu} < 0$; that is, it is costly to reduce the risk of death. As μ_a approaches 0 (or perhaps some finite small level) we expect $f_{a,\mu}$ to increase rapidly in absolute value, since eliminating all risk of death is impossible. Note that in this specification, current expenditures to reduce mortality have no lasting effect, they affect only current mortality. Some of our results depend on this feature, as we will explain when discussing them. We expect that $f_{a,m}$ eventually increases with m, and similarly for $f_{a,z}$.

We expect that natural selection will maximize reproductive fitness, measured as the future representation of an individual's genes at some distant time point, subject to the constraints in (1). This optimal life history problem can be solved by dynamic programming (McNamara, 1991 and Chu and Lee, 2006), and the solution is characterized by

$$1 = \max_{\theta_x} \int_0^\infty e^{-\rho x} \ell_x m_x dx \tag{2}$$

subject to the constraints in (1), where ρ is the intrinsic growth rate of the population, and $\theta_x \equiv (\mu_x; m_x; z_x)$. Chu and Lee (2006) show that the optimal solution corresponding to a discrete version of (2) does characterize the trajectory of an optimal life history.

[2]Many rodents store seeds and nuts, for example, and breeding groups of acorn woodpeckers store thousands of acorns. Nests, burrows, termite mounds and hives, as well as the tools and weapons of human hunter gatherers are all examples of important external capital goods.

[3]Reciprocal altruism refers to apparently altruistic acts of giving which in fact are conditional on having received a gift from the recipient in the past. This conditionality can be interpreted either as a kind of risk sharing or as a kind of borrowing and lending, or both. Such patterns have been studied extensively in humans (Gurven and Kaplan, 2006) and other primates. At best they appear to facilitate a temporally local reallocation which would not affect the broad shape of the life history, as it spans a much greater temporal range.

Many species exhibit "determinate growth," that is, they first grow without reproducing and then largely cease growth and become fertile once they have reached their adult size. Our main interest is in species that have intergenerational interactions after birth, such as mammals or birds, and these exhibit determinate growth. Various authors have shown that in models with linear constraints, m_a and z_a cannot be interior solutions at the same time, so the determinate growth pattern is optimal (Taylor et al., 1974; Vaupel et al., 2004; Chu and Lee, 2006). If the switch occurs at age r, then in our notation the organism would have $m_a = 0$ (no fertility) for $a \leq r$, and would have $z_a = 0$ (no somatic growth) when $a \geq r$.

2.2 Optimal Life History With Intergenerational Transfers

Many animals, including nearly all mammals and birds, make extensive intergenerational transfers of energy and various kinds of care. Examples are lactation and the feeding of nestlings, as well as the more extended provision of food for human children continuing until around age 20 among hunter gatherers (Kaplan, 1994). In cooperatively breeding species, these transfers are made by other members of the group in addition to the parents. Intergenerational transfers alter the budget constraint in ways we now consider.

For most relevant cases, transfers are from a mature adult to an immature juvenile. We consider a transfer from a particular adult age $t > r$ to juvenile individuals age $s \leq r$. Later we will consider transfers made and received at more than one age. An intergenerational transfer necessarily involves more than one generation, so our model of optimal life history explicitly characterizes the lineage of a species. Details can be found in Chu and Lee (2006); here we only provide a sketch.

2.2.1 The Demographic Constraint on Intergenerational Transfers—Suppose the average adult at age t gives away T_t units of energy and the average juvenile at age s receives R_s units of usable energy, which may be 0 for some s. Since no growth occurs after age r (as per earlier discussion) the energy of an age t adult net of transfers is all allocated between p_t and m_t. Thus her budget constraint, building on (1), can be written as

$$f_t(\mu_t, m_t, 0) = \zeta_t(w_r) - T_t. \tag{3}$$

At a juvenile age s, all energy is allocated between μ_s and z_s, so the budget constraint can be written as

$$f_s(\mu_s, 0, z_s) = \zeta_s(w_s) + R_s. \tag{4}$$

Within each cooperatively breeding group, the transfers made by adults at age t must equal the sum of transfers received by juveniles of all ages from all adults at age t. Thus the sum over s of the transfer received at s from an adult age t, R_{st}, weighted by the number of age-s juveniles in the group must be equal to the transfer made by an age-t adult, T_t, times the number of age t adults in the group, as in the "balance equation" of Lee (2003). But to formulate the balance equation, we must know the relative numbers of individuals at different ages.

While the lineage descended from the original optimizer can properly be viewed as reaching a stable population age distribution, the individual members of this lineage live and share resources in smaller groups ranging from parent-offspring sets to larger cooperative breeding groups for species such as humans (Mace and Sear, 2005), pilot whales (Carey and Gruenfelder, 1997; Mann et al, 2000), African wild dogs (Creel and Creel, 2002) or acorn woodpeckers (Koenig and Mumme, 1987). It is within these groups that the transfer identity is expected to hold, rather than in the lineage as a whole. These individual groups may not have stable age distributions, but their average age distributions across groups or across time will be approximately stable. For this reason, we use stable population weights ($e^{-\rho s}\ell_s$) as a useful

approximation for the balance equation, which can be viewed as holding for this average age distribution, as in Lee (2003).

$$\int_0^r e^{-\rho s}\ell_s R_{st} ds = e^{-\rho t}\ell_t T_t. \tag{6}$$

When more than one age makes transfers, the balance equation should hold for each t, and (4) should be rewritten as

$$f_s(\mu_s, 0, z_s) = \zeta_s(w_s) + \int_r^\infty R_{st} dt. \tag{4′}$$

3 Juvenile Mortality Trajectories

Recall that at each juvenile age s, body weight accumulates according to $dw_s/ds = z_s$. If an age-s juvenile receives a transfer of an additional unit of energy, then using (4) iteratively we see that the *compound* effect on her mature body size (w_r) would be

$$\frac{\partial w_r}{\partial R_s} \equiv K_s = \frac{1}{f_{s,z}} \exp\left(\int_s^r \frac{\zeta_{x,w}}{f_{x,z}} dx\right). \tag{5}$$

The first term $1/f_{s,z}$ tells us the amount of the increase in body size that can be achieved with one unit of energy received at age s. Inspection of the exponentiated factor shows us that K_s is decreasing in s, since the integrand $\zeta_{x,w}/f_{x,z}$ is positive, and hence the integral decreases as s rises. This means that it is more efficient to invest in somatic capital (body size) early than late, for the increased capital raises the output from foraging, which in turn leads to faster future growth. Here $\zeta_{x,w}/f_{x,z}$ is the instantaneous growth rate in body size at age x. Alternatively, if K_s refers to neural capital or brain size, the compound factor K_s can be understood as an effect of learning by doing (see Robson and Kaplan, 2003).

3.1 The First Order Condition for μ_a

Now we solve the problem in (2). Let $(\mu_a^*, m_a^*, z_a^*)\ \forall a$ denote the optimal life history that solves (2). The optimal survival probability ℓ_a^* is given by $\exp\left(-\int_0^a \mu_x^* dx\right)$. Let $V_a(\ell_a, w_a) \equiv (1/\ell_a)\int_a^\infty e^{-\rho(x-a)}\ell_x^* m_x^* dx$ be the value function for the dynamic problem. We obtain the Bellman equation as follows:

$$0 = \max_{\theta_a}\left\{\ell_a m_a - \frac{\partial(\ell_a V_a)}{\partial \ell_a}\ell_a \mu_a + \ell_a \frac{\partial V_a}{\partial w_a} z_a + \ell_a \frac{\partial V_a}{\partial a} - \rho \ell_a V_a\right\},$$

where $\partial V_a/\partial a$ is defined as $\lim_{h\to 0}[(V_{a+h} - V_a)/h]$. The state variables for the dynamic problem are ℓ_a and w_a. Let $\eta_\ell(a)$ and $\eta_w(a)$ be the corresponding adjoint variables. By solving the adjoint equations,[4] we obtain

[4]The adjoint equation for $\eta_\ell(a)$ is $\dot{\eta}_\ell(a) = -(m_a^* + \ell_a^* \frac{\partial m_a}{\partial T_a}\frac{\partial T_a}{\partial \ell_a}) + (\rho + \mu_a^*)\cdot \eta_\ell(a)$, in which $\partial m_a/\partial T_a = -1/f_{a,m}\ \forall a$ and $\partial T_a/\partial \ell_a = -(1/\ell_a)\int_0^a e^{-\rho(x-a)}(\ell_x/\ell_a)R_{xa}dx$ if $a \le r$ or $-T_a/\ell_a$ if $a > r$. The initial condition for the differential equation is $V_0(\ell_0, w_0) = \int_0^\infty e^{-\rho x}\ell_x m_x dx$. It is then easy to verify that $\eta_\ell(a)$ specified here is indeed the solution to the adjoint equation. For $\eta_w(a)$, specified here is indeed the solution to the adjoint equation For $\eta_w(a)$, the adjoint equation is $\dot{\eta}_w(a) = (\rho - \zeta_{a,w}^*/f_{a,z}^*)\cdot \eta_w(a)$, for which the solution is $\eta_w(a) = \eta_w(r)e^{-\rho(r-a)}\exp(\int_a^r \zeta_{x,w}^*/f_{x,z}^* dx)$ for $a \le r$. Further, $\eta_w(r)$ can be derived by noting that $\eta_w(r) = \partial(\ell V_r)/\partial w_r$ and that $\ell_r V_r = \int_r^\infty e^{-\rho(x-r)}\ell_x^* m_x^* dx$. The solution results by applying $\partial m_x^*/\partial w_r = \zeta_{x,w}^*/f_{x,m}^*$.

$$\eta_\ell(a) \equiv \frac{\partial(\ell_a V_a)}{\partial \ell_a}$$
$$= \begin{cases} \frac{1}{\ell_a^*}\left(\int_r^\infty e^{-\rho(x-a)}\ell_x^* m_x^* dx + \int_r^\infty \frac{1}{f_{x,m}^*}\int_0^a e^{-\rho(s-a)}\ell_s^* R_{sx} ds dx\right) & \text{if } a \le r, \\ \frac{1}{\ell_a^*}\int_a^\infty e^{-\rho(x-a)}\ell_x^*\left(m_x^* + \frac{T_x}{f_{x,m}^*}\right)dx & \text{if } a > r; \end{cases}$$

and

$$\eta_w(a) \equiv \ell_a \frac{\partial V_a}{\partial w_a} = e^{\rho a}\exp\left(\int_a^r \frac{\zeta_{x,w}^*}{f_{x,z}^*}dx\right)\int_r^\infty e^{-\rho x}\ell_x \frac{\zeta_{x,w}^*}{f_{x,m}^*}dx \quad \text{for } a \le r.$$

The superscript (*) for the partial derivatives ($\zeta_{x,w}$, $f_{x,z}$, and $f_{x,m}$) represents the fact that they are evaluated at the optimal life history.

Substituting m_t in (3) for $t > r$ and z_s in (4′) for $s \le r$, we can derive the first order condition for age-specific mortality μ_a. We shall first present our discussion for the case of immature ages in this section.

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 $a \le r$ to maximize fitness, the first order condition for the optimal μ_a, after factoring out a constant, is the following:

$$\Delta_{\mu_a} = M + \int_r^\infty \frac{1}{f_{x,m}}\int_0^a e^{-\rho s}\ell_s R_{sx} ds dx + f_{a,\mu} K_a N = 0, \tag{7}$$

where Δ is the partial derivative of the right hand side of the Bellman equation with respect to μ_a, K_a is given in (5),

$$M \equiv \int_r^\infty e^{-\rho x}\ell_x m_x dx$$

and

$$N \equiv \int_r^\infty e^{-\rho x}\ell_x \frac{\zeta_{x,w}}{f_{x,m}}dx.$$

The interpretation of M and N will be given as we proceed.[5]

When we totally differentiate the growth rate ρ with respect to μ_a, we are considering a movement of μ_a along the efficient (maximal) trait surface, possibly away from the optimum position on this surface where it is tangent to a fitness contour (Partridge and Barton, 1993; Partridge and Sibly, 1991). Because the movement is along the surface, the increase or decrease in μ_a absorbs or releases energy and thereby affects growth, fertility or transfers, depending on whether a is before or after sexual maturity. If this movement is evaluated at the optimum, as in (7), these tradeoff effects must exactly counterbalance each other, so that the derivative is zero.

This is different from the situation considered by Hamilton (1966) or Lee (2003), in which a mortality perturbation is purely inefficient and deleterious, raising mortality with no offsetting release of energy. Such a movement is toward the origin from the efficient trait surface, not along it, so the cost (last) term in (7) is not considered. Alternatively, Hamilton and Lee consider

[5]If we totally differentiate (2) and arrange terms, we find $\Delta_\rho d\rho + \Delta_{\mu_a} d\mu_a = 0$. This equation gives us $d\rho/d\mu_a$, the marginal impact of change in age-specific mortality μ_a on the fitness parameter ρ Note that Δ_ρ is a normalization term that is independent of a, involving some average ages, and therefore does not concern us here.

a costless improvement in survival, opposite to the deleterious case just described. That is why the derivative in (7) equals zero, while the derivative in the Hamilton or Lee analysis indicates a non-zero fitness impact. Real mutations could be of either sort or of intermediate kinds, and their consequences would vary accordingly.

In (7), M is the weighted expected lifetime fertility (NRR or R_0). This is closely related to Hamilton's fitness impact for a mortality perturbation at some juvenile age $a < r$. 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 juvenile age a 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, his theory cannot explain the high infant mortality and declining juvenile mortality of many species.

The second term in (7) gives the cumulated transfers received per individual that attains age a, including the transfers wasted on other juveniles who died before reaching age a, in the inner integral, which is expressed by the outer integral in units of births foregone by the adults making the transfers. This amount, which increases in age a, captures Lee's (2003) accumulated transfer effect. In particular, when a is smaller, the fitness cost of "sunk" transfers up to age a is smaller, hence an increase in mortality for age a is less costly. Alternatively, because weighted transfers made and received must integrate to zero over the life cycle, the sunk costs up to some age equal the net transfers to be made in the future by an individual this age. Yet 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.[6] 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.

The first two terms in (7) capture the two benefits of a mutation that exogenously lowers μ_a. In the context of beneficial mutations and optimal life history, as opposed to deleterious mutations and mutation accumulation theory, the decrease in μ_a has an opportunity cost, as described by (4′). This tradeoff cost is ignored under the mutation accumulation approach, because deleterious mutations are assumed to be inefficient, yielding no benefits elsewhere, as discussed earlier. But varying μ_a along the efficient trait surface entails offsetting costs or benefits. For a juvenile to increase her survival probability slightly at age a by $-d\mu_a$, she must decrease the energy allocated to the accumulation of her body weight by $-f_{a,\mu} \cdot d\mu_a$. This reduction in energy for growth will in turn shrink her mature size (w_r) by K_a according to (5), which we discussed earlier. For each unit of reduction in mature size w_r, the impact on fitness (force of selection) is the expected reduction in weighted lifetime fertility, and that is in fact the N term in (7):

$$N \equiv \frac{\partial \int_r^\infty e^{-\rho x} \ell_x m_x dx}{\partial w_r} = \int_r^\infty e^{-\rho x} \ell_x \frac{\partial m_x}{\partial w_r} dx = \int_r^\infty e^{-\rho x} \ell_x \frac{\zeta_{x,w}}{f_{x,m}} dx.$$

3.2 The Optimal Juvenile Mortality Trajectory

Equating Δ_{μ_a} to zero, we get the first order condition for μ_a, as in (7). It is easily shown that the second order condition for maximization is satisfied. Simultaneously solving for μ_a explicitly would be tedious, but for the purpose of finding the shape of the μ_a across different ages a, we can change the variable by setting $\xi_a \equiv \exp(\mu_a)$ and rewriting $\Delta_{\xi_a} = 0$ as follows:

[6]This kind of effect on sibling survival has been studied experimentally for birds, by adding or removing eggs in a brood, and later observing the numbers of hatchlings and fledglings. In humans, the lactation interruption effect, in which the death of a lactating infant shortens the interval to the next birth, illustrates the same effect.

$$\xi_a = \frac{M + \int_r^\infty \frac{1}{f_{x,m}} \int_0^a e^{-\rho s} \ell_s R_{sx} ds dx}{-f_{a,\xi} K_a N}, \tag{8}$$

where $f_{a,\xi} \equiv \partial f_a(\text{In } \xi_a, m_a, z_a)/\partial \xi_a$. Note that (8) is not an explicit reduced-form solution for ξ_a, for the right-hand side of (8) still has ξ_a in it. However, it helps us understand how ξ_a varies with age a. We know from the above discussion that the numerator of (8) is increasing in a due to the increasing transfer effect, but what about the denominator?

In view of (5) the denominator of (8) can be written as

$$-f_{a,\xi} K_a N = -\frac{f_{a,\xi} N}{f_{a,z}} \exp\left(\int_a^r \frac{\zeta_{x,w}}{f_{x,z}} dx \right)$$

Evidently, the integral, and hence its exponential in the above expression, is non-increasing in a, for the compounded effect of accumulating size becomes smaller as age increases, since compounding operates over a shorter period.[7] 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 larger size permits more production of energy, leading 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 doing so has a lower opportunity cost. 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 $\zeta_{a,w} = 0 \ \forall a \leq r$), 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.

Note that this argument depends on our assumption that energy expenditures on survival only reduce current mortality and have no lasting beneficial effect, a common assumption in the literature, implicitly or explicitly (Williams, 1966; Schaffer, 1974; Taylor et al., 1974). Examples of this kind of expenditure include food consumption to avoid starvation and maintain adequate function and energetic costs of evading predators or operating the immune system. Some other expenditures on survival are long term investments, such as building an immune system, as some analysts emphasize (Abrams and Ludwig, 1995). So long as there is an important component of survival expenditure that yields only transitory survival benefits, our argument will hold.

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

[7] The exponential term can be approximated in discrete form, which illuminates the underlying effect of compounding. Let J be the number of periods before maturity, and $\delta \equiv r/J$ be the length of a period. For convenience of exposition, suppose $k \equiv a/\delta$ is an integer. When δ is small, $\exp\left(\int_a^r \zeta_{x,w}/f_{x,z} dx \right) \approx \prod_{i=k+1}^{J} (1 + \delta \zeta_{i,w}/f_{i,z})$.

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 non-monotonic age variations in any of the parameters. Likewise, it is difficult to draw any conclusions about the age pattern of the efficiency of allocations to maintenance and survival, $f_{a,\xi}$. 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. For this reason, for the time being we will not consider variation in ($f_{a,\xi}$, $f_{a,z}$) as an influence on the age pattern of juvenile mortality.

3.3 The Forces Shaping Juvenile Mortality

Now let us pause to take stock and interpret what we have learned. From (7) or (8) we have identified four forces that influence the shape of ξ_a for immature ages.

1. The Hamilton Effect: the term M representing expected future reproduction which is independent of a and so does not affect the shape of juvenile mortality.
2. The Transfer Effect: $\int_r^\infty \frac{1}{f_{x,m}} \int_0^a e^{-\rho s} \ell_s R_{sx} ds dx$, which is increasing in a 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: $\exp\left(\int_a^r \frac{\zeta_{x,w}}{f_{x,z}} dx\right)$, which is increasing in a 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). 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. Because energy production is usually proportional to body size, earlier investments in body growth expand the feasible range of energy allocations at these older ages. The effects considered fourth are unknown, but unless powerful, would be overwhelmed by the others.

In a different setting, Robson and Kaplan (2003) also derived a declining mortality schedule at 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 leads to U-shaped mortality, for an optimal life history must secure high reproductive value through low mortality. The model they presented, however, does not explicitly have a biological immature period prior to reproduction and hence cannot be compared with the Hamilton result directly. For the case of primates and human beings in particular, Robson and Kaplan (2003, 160) did assume a pattern of neural capital accumulation similar to the scenario of determinate growth. 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 at immature ages, so that $\zeta_{a,w} = 0 \ \forall a$

$\leq r$. Even in this case without any hump, we are able to generate a declining age-specific mortality result as long as the juveniles receive a sufficient amount of transfers.

4 Mature Mortality Trajectories

Now we consider the μ_a trajectory for mature (adult) ages $a > r$. Using identity (6), we see that the first order condition for maximizing (2), after factoring out a constant, is the following:

$$\begin{aligned}\Delta_{\mu_a} &= M_a + \int_a^\infty e^{-\rho x}\ell_x \tfrac{T_x}{f_{x,m}}dx + \tfrac{f_{a,\mu}}{f_{a,m}}e^{-\rho a}\ell_a \\ &= \int_a^\infty e^{-\rho x}\ell_x\left(m_x + \tfrac{T_x}{f_{x,m}}\right)dx + \tfrac{f_{a,\mu}}{f_{a,m}}e^{-\rho a}\ell_a = 0,\end{aligned}$$

where

$$M_a \equiv \int_a^\infty e^{-\rho x}\ell_x m_x dx.$$

Term M_a is the weighted fertility effect, similar to the Hamilton effect M in (7) but varying with a, and T_x is the transfer made at age x. Once having reached age a, only later ages (x greater than a) can possibly contribute to the benefit of surviving past age a, hence the integrations in (9) start at $x = a$. All these future fertility and transfer effects have to be added up as we consider the benefit of a change in μ_a.

The integral in (9) gives the total benefit which an individual protects by sustaining survival. The last term is the opportunity cost of μ_a. As one can see from (3), reducing μ_a can be achieved only at the cost of reducing m_a. The ratio $|f_{a,\mu}/f_{a,m}|$ characterizes the tradeoff costs of μ_a relative to m_a. The cost term is multiplied by $e^{-\rho a}\ell_a$ because we have done so for terms associated with the benefit. Note that the cost term is also decreasing in a, partly offsetting the declining pattern on the benefit side.

4.1 Optimal Adult Mortality

Changing the variable to $\xi_a \equiv \exp(\mu_a)$ and equating Δ_{ξ_a} to zero, we derive the following characterization of ξ_a, which is not a reduced form but is nonetheless helpful:

$$\xi_a = \int_a^\infty e^{-\rho x}\frac{\ell_x}{\ell_a}\left(m_x + \frac{T_x}{f_{x,m}}\right)dx \Big/ \left(-e^{-\rho a}\frac{f_{a,\mu}}{f_{a,m}}\right). \tag{10}$$

A principle difficulty in drawing a firm conclusion about whether survival falls with age and mortality rises is that ℓ_a is decreasing in a. Optimization at each age is forward looking and conditional on survival to that age, which is why ℓ_a appears in (10). We will now consider whether the Hamilton and transfer effects are declining faster or slower with age than ℓ_a.

Recall that the dynamic maximization problem in (2) for mature ages is to find $\theta_x\ \forall x > r$ that solves

$$\max_{\theta_x \in \Omega_x, x>r} \frac{1}{\ell_r}\int_r^\infty e^{-\rho x}\ell_x m_x dx, \tag{11}$$

where the set Ω_x is the feasible set for age x, as described in (3): $\Omega_x \equiv \{\theta_x : f_x(\mu_x; m_x; 0) \leq \zeta_x(w_r) - T_x\}$. The maximal value obtained from the optimization problem in (11) starting from any age a is denoted $B(a)$:

$$B(a) \equiv \max_{\theta_x \in \Omega_x, x>a} \frac{1}{\ell_a}\int_a^\infty e^{-\rho x}\ell_x m_x dx.$$

In what follows, we shall study the pattern of $B(a)$.

We note from (11) that the a-forward problem is symmetric for each a except: 1) the terms to be integrated in the maximand decrease when a increases, and 2) the feasible set Ω_x may be different across x. The first factor certainly causes the maximum value of the a-forward problem to decline as a increases. Thus, we can conclude that $B(a)$ is decreasing in a, provided that the feasible set Ω_a does not increase as a increases. The constraint in (3) can be approximated using a Taylor expansion: $f_{a,\mu} \cdot (\mu_a - \mu_a^*) + f_{a,m} \cdot (m_a - m_a^*) \leq \zeta_a(w_r) - T_a$. Therefore, we know that the condition of a contracting Ω_a is met if either the cost of maintaining survival and reproducing $(-f_{a,\mu}, f_{a,m})$ is not decreasing, or $\zeta_a(w_r) - T_a$ is not increasing, or both. In Vaupel et al. (2004), there is indeterminate growth, so that w_a may increase even after maturity. This would expand the feasible set Ω_a, and hence "negative senescence" could occur. A similar expansion in Ω_a with age could occur if productivity rises with experience. Male hunting productivity rises in young adulthood for humans and peaks in the early 40s, for example (Gurven and Kaplan, 2006).

There are various reasons to expect $-f_{a,\mu}$ and $f_{a,m}$ to increase in a: 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, that is mutation accumulation. Can we say something about the pattern of $\zeta_a(w_r) - T_a$?

There are two interesting cases we can consider for the age shape of this disposable energy. First, suppose there are no transfers or transfers are small in the relevant range of a. Then $\zeta_a(w_r) - T_a \approx \zeta_a(w_r)$. In this case, $B(a)$ is indeed decreasing in a. It is easy to see that when $T_x = 0\ \forall x$, the integral in the numerator of (10) is exactly $B(a)$. Thus, as long as $\zeta_{a,w}$ is not increasing in a, the survival probability would decrease with age in the case without transfers, a conclusion the same as in Hamilton.

Second, if the transfer at age a is close to a fixed proportion of the energy produced at this age, $T_a \approx \gamma\zeta_a(w_r)$, where γ is a constant, then $\zeta_a(w_r) - T_a \approx (1 - \gamma)\ \zeta_a(w_r)$. Then we can simply reparameterize w_r, and the conclusion would be the same as that of the first case.

In the most general case, when T_a may vary in other ways, one does not know the exact pattern of Ω_a, and hence the shape of ξ_a, for adult ages. However, as $a \to \infty$, terms to be summed in (11) go to zero. Thus, unless the ratio $-f_{a,\mu}/f_{a,m}$ in the denominator decreases more slowly than the numerator due to a decline in the energy efficiency of fertility that is more rapid than that of survival, we can conclude that ξ_a should increase with age and therefore that mortality *eventually* rises with age. We have no reason to expect any particular trend in the ratio $-f_{a,\mu}/f_{a,m}$, let alone a strong one. Note, however, that the pace of the increase of optimal mortality with age is slower than the pace under mutation accumulation, due to the factor $1/\ell_a$ which can account for a period of slow senescence in adults.

Some researchers have noted a delay between maturity and the onset of rising adult mortality, and sometimes even a period of declining adult mortality, although this is uncertain due to data issues (Promislow, 1991; Sibly et al., 1997). Promislow offers several possible explanations. One is heterogeneity and selection, so that the robustness of surviving adults rises with age. Heterogeneity and selection would be entirely consistent with our analysis here, since each genotype could experience rising mortality risks from maturity, even if the aggregate population showed initially stable or declining mortality following maturity. Promislow also suggests that, following maturity, adults may continue to learn to forage and evade predators more efficiently, as we mentioned earlier. In our theory, this would lead to increasing foraging efficiency $\zeta_{a,w}$ and decreasing energetic costs of survival $|f_{a,\mu}|$, either of which could lead to stable or declining mortality as discussed in the preceding paragraph.

A recent study (Baudisch, 2008) develops a model of an organism which allocates resources among maintenance, vitality, and fertility, and finds that one possible optimal outcome is that the organism grows to maturity and then allocates energy so as to achieve sustenance, a steady state without senescence. This appears to be inconsistent with our conclusion of U-shaped mortality with determinate growth, but in fact comparison is not straightforward.

As with most treatments in the literature, we do not include vitality as a variable, but our weight variable has some of vitality's features. In the Baudisch model, there is a single choice: how to allocate energy between growth/maintenance on the one hand and fertility on the other, with vitality increasing only if more than enough energy is allocated for maintenance. In our model, total energy can be allocated for four purposes (fertility, maintenance, growth and transfers) so there are three independent choices rather than one. Another key difference is that in our model, production is always positively related to weight, but in the Baudisch model energy production is first increasing and then decreasing in vitality (her equation 5.2). The models are not nested, so we cannot say that one is a special case of the other. Getting different results does not imply that one of the models is wrong.

4.2 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. Surviving to adult age a has the following effects:

1. The Hamilton Effect: the term M_a evaluates expected net fertility after age a. But, unlike for juveniles, here M_a necessarily declines monotonically toward 0 as age increases, indicating that it is progressively less beneficial to allocate resources to survival. The original Hamilton fitness impact effect is not divided by ℓ_a, but in this optimization context it is, as discussed earlier.
2. The Transfer Effect: the total expected transfers that remain after age a (survival weighted and discounted by population growth). This necessarily declines monotonically toward 0 as age increases, provided that adults are not net receivers of transfers at any age (see Lee 2003). Unlike the Hamilton Effect, 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 ($f_{x,m}$) to convert the energy involved in the transfers into units of fertility. Conditioning on survival to a (division by ℓ_a) is due to the optimization context.
3. The Fertility Cost of Reducing Mortality: The energy tradeoff is $|f_{a,\mu}/f_{a,m}|$, which is survival-weighted and discounted. The energetic cost of reducing mortality expressed in units of foregone fertility is $|f_{a,\mu}/f_{a,m}|$. There is no clear reason to expect this ratio to vary in one way or another. Unless very large, such variations will be overwhelmed by the other factors.

5 Conclusions

We have considered the characteristic U-shape of mortality. 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 genetic mechanism of mutation accumulation. However, his approach failed to explain either declining juvenile mortality or postreproductive survival, it did not apply to species that make intergenerational transfers and it did not take into account tradeoffs among life history traits. Even his apparently definitive answer to the second question has been shown to fail when the theoretical approach is optimization of the life history and when 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. We have also focused on the shape of the optimal life history, achieved through selection of beneficial mutations, while ignoring the influence of accumulated deleterious mutations. The accumulation and selection of deleterious mutations could be readily included, leading to systematic departures of the evolved life history from its optimal shape, causing adult mortality to rise more rapidly. At the same time, we have broadened our analysis to include physiological tradeoffs, a necessity for the optimality approach, and to allow intergenerational transfers.

Given this setup, we have derived a number of important new results for the optimal shape of mortality for species with determinate growth and with or without intergenerational transfers. We have shown that juvenile mortality must decline from birth to maturity, either to protect the increasing cumulative investments in juveniles found in the mutation accumulation context) or to exploit the advantages of early investment in somatic growth at the expense of survival, arising from compounding effects, or to do both. At least one of the two effects must be operative at any age, so juvenile mortality decline is assured. We have shown that adult mortality must increase following maturity in species without transfers, due to a version of the Hamilton effect that is weakened through division by the probability of survival up to each age in question, which declines with age. If a species makes intergenerational transfers, these provide an additional reason for mortality to rise with adult age but also lead to postreproductive survival as in Lee (2003).

These results hold as long as the constraint parameters do not imply energetic efficiency gains with experience in foraging, survival or fertility, or transfers do not decline relative to production in the optimal life history. Adult mortality must rise eventually in deterministic species, but an earlier phase of stability or decline in mortality could possibly occur.

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 causing juvenile mortality to decline–the compounding power of somatic growth. These forces combine to produce a U-shaped mortality pattern in species with determinate growth, including postreproductive survival for species that make transfers.

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