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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: 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 postreproductive 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 will 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

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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; 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 Sibly, 1991).

Due to 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, instead it is 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. noted 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. were 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 relative 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 derived 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 "non-reproductive period in the life history" a U-shaped mortality schedule was optimal for some parameter settings. Within such a period, the 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.