Economics and the evolution of life histories

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Birth and death are subjects of perennial interest. They are shaped by both physiology and behavior, which are themselves shaped (presumably) by natural selection. Thus, it seems natural to suppose that evolutionary ideas would form the core of demography. This, however, is not the case. Students of birth and death are largely divided: social scientists call the subject demography, and evolutionists call it life history evolution. In this issue of PNAS, Ronald Lee (1) proposes a theory that may help heal this divide.

Lee's theory is about aging. Within evolutionary biology, discussion of this subject has been dominated for decades by two theories: mutation accumulation and antagonistic pleiotropy (2). The first of these holds that selection is less effective at removing harmful mutations that act in old age, so such mutations accumulate. The second holds that some mutations are beneficial in youth but harmful later on. Such mutations accumulate because selection is more responsive to early effects than to late ones. Kirkwood's (3) "disposable soma" theory describes a mechanism that can plausibly generate antagonistic pleiotropy.

Lee's focus is on parental care and other transfers of resources between individuals of different ages. When a woman dies at, say, age 30, the death deprives her children of the care they would have received from her. Thus, a mutation that increases the mortality of 30-year-olds has harmful effects at several ages. This has the flavor of antagonistic pleiotropy, but the theory is broader in that it encompasses cases in which the early and late effects are both harmful or both helpful. The real value of the new theory, however, is in its success in wedding an economic model of exchange between individuals to the evolutionary theory of aging.

Lee's article addresses several important questions. The first of these has to do with the age profile of mortality during childhood. Among humans and many other species, death rates decline dramatically during the initial part of life (4, 5). This pattern is pronounced in some species but apparently absent in others (6). Why should this be? The first attempt at an answer was made 70 years ago by Fisher (7), who observed that an average 15-year-old will contribute more to future generations than will a newborn because the 15-year-old is less likely to die before reproducing. Thus, he argued, selection affects genes more strongly if they are expressed in 15-year-olds than if they are expressed in newborns. From this perspective, high newborn mortality results from the relatively weak selection acting on them.

This argument seemed to fit the facts and held sway for 36 years, until Hamilton (8) found its flaw in 1966. Fisher had been right to ask what contribution each age class makes to future generations. But what matters is the contribution of the age class as a whole, not the contribution per individual. Correcting this mistake, Hamilton found that the force of selection on genes affecting mortality is constant during childhood. It is as strong for newborns as it is for 15-year-olds. Thus, the revised theory fails completely to account for the facts of juvenile mortality.

If selection favors production of children, how could it favor an early end to fertility?

There have been other suggestions (9, 10). Perhaps the most influential idea is what Hamilton (8) called the principle of sibling replacement. In species with intense parental care, offspring are costly. When one dies, the parental resources that are freed can be used to produce another. If this must happen, it is best for it to happen early, for this minimizes the parental resources squandered on reproductive failures. Thus, selection should shove vulnerabilities into early childhood. This idea was anticipated by Fisher (7) and has been discussed by many later authors. Until now, however, there has been no theoretical model capable of predicting the magnitude of the effect. Lee's article fills this hole and sheds light on a second issue: the early menopause of human females.

This is a puzzle that takes us to the opposite end of the female lifespan. If selection favors production of children, how could it ever favor an early end to fertility? Why do women not continue producing babies into old age? Or, to look at the problem from the other direction, how does selection weed out harmful mutations that increase mortality late in life? The force of selection affecting genes expressed in 50-year-old women should depend on the contribution that such women make to future generations. But if these women have stopped reproducing, this contribution would seem to be nil. Thus, harmful mutations acting late in life should accumulate and death should follow soon after reproduction stops (11).

In most species, this is exactly what does happen. But there are exceptions. Among humans and some whales, females may live long after reproduction has ceased (12–14). Williams (11) suggested that older females have more to gain by helping their existing children and grandchildren than by producing more of their own. Thus, selection opposes mutations that would increase either the mortality or the fertility of older females.

Although other explanations have been proposed (15–18), there is an abiding interest in variants of Williams's hypothesis. In 1993 I was able to reject the view that early menopause evolved to protect women from childbirth mortality. I was unable to reject the "opportunity cost" hypothesis, which holds that each new child reduces the woman's ability to enhance the survival and fertility of existing children and grandchildren (19). Since then, Shanley and Kirkwood (20) have found a reasonable fit to a model combining both of these hypotheses, and ethnographic research has generated clear evidence that older women have a beneficial effect on children and grandchildren in traditional societies (21, 22). Comparisons across primate taxa show that, after regressing out the lengths of childhood and lifespan, birth rates are higher in humans than in other apes (23, 24). This is all consistent with the "grandmother hypothesis," which holds that the labor of older women accelerates the rate of childbearing in humans. Yet there is still room for skepticism. We might expect human fertility to exceed that of apes simply because the human population has been growing and ape populations have been declining in recent decades. Other efforts to test variants of Wil-

See companion article on page 9637.

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www.pnas.org/cgi/doi/10.1073/pnas.1733942100

liams's idea have failed to yield significant results (25). Thus, this issue is still open after a decade of accelerating research interest.

Lee (1) brings a new style of mathematical model to these issues. He begins with the standard renewal equation of life history theory (10). Onto this he grafts a "balance equation," which stipulates that mean lifetime consumption must equal mean lifetime production. This latter condition carries a lot of freight: it allows an analysis of evolutionary equilibria in the context of parental care and other transfers of resources between individuals.

This model is very general. It assumes that fertility increases with consumption, that mortality declines with consumption, and so on, but does not make specific assumptions about the forms of any of these functions. It does, however, make one restrictive assumption: it assumes that production equals consumption within each genotypic class. This means that there are no transfers of resources between individuals of different genotypes. But in an outbred sexual population, a rare mutant is equally likely to have a normal or a mutant mother. If transfers are from mother to daughter, half of the transfers received by a rare mutant will be across genotypic classes. Thus, Lee's model implicitly assumes clonal reproduction in which each individual has a single parent of identical genotype.

There is a long tradition in ecology of building clonal models to study sexual organisms (26). Such models are useful because they simplify problems that are otherwise complex, yet often reveal essential features. They should, of course, be regarded cautiously, because one sometimes has to eat one's words when the sexual model fails to replicate the clonal one (27).

The model has one other limitation. It depends critically on the shape of what Lee calls the "balance curve." This is the relationship, as implied by the balance equation, between growth rate and γ, an index of consumption. The analysis depends on this curve having the shape of an inverted "U," but there is no clear delineation of the circumstances under which this is true. This is no fatal flaw, for it is easy to show that the inverted "U" is there in at least some cases, but it will be important to work out the conditions under which this assumption holds.

Lee shows that the force of selection on mutations affecting mortality has two components, one measuring expected future births (the Hamilton effect) and the other measuring expected future net production (the transfer effect). At evolutionary equilibrium, the Hamilton effect falls to zero so that selection is affected by the transfer effect alone.

The transfer effect increases throughout childhood because children consume more than they produce. Thus, mutations that increase mortality are removed more rapidly if they act late in childhood than if they act early. This may account for the decline in mortality during childhood. It formalizes the sibling replacement hypothesis of Hamilton (8). Net productivity is also positive in postmenopausal women, so the transfer effect remains positive after reproduction has ceased. This formalizes the hypothesis of Williams (11). Thus, both hypotheses receive support in the context of a single model.

Although the analysis is entirely qualitative, the equilibrium result is in terms of a quantity, the transfer effect, that can be estimated. Thus, Lee is able to show an excellent agreement between the entire age profile of human mortality rates and the predicted force of selection. This is the most comprehensive evolutionary theory of aging that we have seen to date.

I am grateful for comments from Elizabeth Cashdan, Kristen Hawkes, Henry Harpending, Jon Seger, James Wood, and Ronald Lee.

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