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Why do We Die? Economics, Biology and Aging*

Arthur J. Robson and *Hillard S. Kaplan*

Why do we die? Why, to be more precise, do we age, in the sense that our mortality rate rises rapidly in a terminal phase of life?

One way to illustrate the effect of aging on longevity is to calculate the life expectancy of nine year olds if they could sustain their current mortality rate. For the U.S. population in 2003, this life expectancy would be just over 7,000 years. (See United States Centers for Disease Control, 2006.) That is, we would not be immortal, because there is still a constant positive probability of dying; but our lives would be vastly longer if mortality risks did not increase with age. In fact, 2% of the population of nine year olds would live to almost 30,000 years of age.

Mortality has been modeled as endogenous within economics. Previous literature has taken mortality to be influenced by the stock of health capital, for example, where this is subject to discretionary investment. (See Michael Grossman (1972) and Isaac Ehrlich and Hiroyuki Chuma (1990), for example.) However, this literature makes aging inevitable because the depreciation rate for health capital is assumed to be an exogenously given increasing function of time. It is therefore increasingly expensive to maintain mortality, and aging results. Without this assumption, that is, optimal mortality would be constant.

Indeed, from the perspective of conventional autonomous optimal control theory, aging seems puzzling. If the body, its health, and other functional abilities represent a capital investment that grows in value during development, how can it be economically or evolutionarily optimal to allow that stock to depreciate? Why wouldn't the body evolve towards an optimal steady state, under the optimal investment strategy?

Why aging occurs is a biological question with profound importance generally and for economics in particular. Further, the insights derived from economics help to provide an answer. This paper has two principal goals. The first specific goal is to sketch such a theory of optimal aging, in which the increase in mortality rates with age is endogenous. The second general goal is to exemplify how biology and economics can advantageously be integrated.

I. The Biology of Aging

The classical biological theory of aging, due importantly to William D. Hamilton (1966), argues that natural selection on genes that act at various different ages is weaker at older ages. That is, since death always occurs with a positive probability, traits expressed at older ages have a smaller impact on fitness, other things equal. Since the frequency of deleterious mutations is a balance between the mutation rate and the force of natural selection against them, the frequency and overall severity of such mutations should increase with age.

There are problems with this mutation-selection balance account. For example, Hamilton's analysis implies that mortality should be constant across all ages up to sexual maturity. Thus, as he recognized, his theory fails to predict the actual decrease in mortality during the first pre-reproductive phase of life. Indeed, the most fundamental aspect of this classical theory---the claim that aging is an inevitable consequence of natural selection---has recently come under vigorous attack. From an empirical perspective, it is now known that some species exhibit negligible aging (Caleb E. Finch, (1998)), and mortality rates, in other cases, may even fall late in life (James W. Vaupel et al. (2004)).

Another problem with this classical model is that it considers only half the picture. It is as if analysis of a firm considered only revenue, but suppressed all discussion of cost. A mutation that lowered mortality at any age would certainly be beneficial, if this had no cost. But surely increased immune function, for example, has a cost and selection for such a mutation depends on the balance between cost and benefit. The crucial issue is: When are these costs incurred? Perhaps these costs are contemporaneous, so immune function, for example, is enhanced immediately with more metabolic resources, at the cost of reduced present fertility. Now an early mutation will be evolutionarily favored in exactly the same circumstances as would a late mutation. Although the benefits of the later mutation are reduced relative to the first by intervening mortality, the cost of this later mutation is reduced by exactly the same factor.

Thomas B.L. Kirkwood (1990) offered an alternative to this mutation accumulation argument with his "disposable soma" theory of aging. A key feature of this theory is the segregation of the “germ line”---the sex cells---from the “somatic line”---the body cells. This segregation implies that damage or mutations arising in somatic cells are not transmitted to offspring via the sex cells. Kirkwood argues that this segregation means that optimal repair of somatic tissue may be incomplete, so the soma (body) deteriorates with age, ultimately being replaced by descendants. However, in Kirkwood's model, it is assumed to be impossible to make the mortality rate fall over time and prohibitively expensive at the margin to keep it constant. This assures aging without really explaining it. His model also fails to account for an initial phase of life with decreasing mortality.

It is clearly desirable that a theory account both for decreasing mortality in a first phase of life, and for increasing mortality, or aging, in a second phase. At the same time, it should ideally also account for the significant post-reproductive longevity displayed by

humans. The model we sketch below builds on both the biological and economic models, while also remedying some of their salient weaknesses.

II. An Overview of Our Model

The basis of our model (Arthur J. Robson and Hillard S. Kaplan (2006)) is that organisms invest in somatic (bodily) capital, which is then used to produce energy to support continued life and further reproduction. Such somatic capital is characterized by both quantity and quality. The quantity of capital is the number of somatic cells, which is closely related to size. We are a species with determinate growth, so the model assumes that the number of somatic cells increases up to some optimally determined age and is constant thereafter. Cell quality, interpreted as functional efficiency in our model, is endogenous. Its deterioration can always be slowed or reversed, by investment in repair. Without such investment, cell quality depreciates over time due to the build-up of deleterious by-products of cell metabolism, for example.

Our theory is a version of the disposable soma theory, in which the cost of investment in quality of a line of cells depends positively on the number of cells. This is compelling since each cell must generate its own maintenance costs. It is then evolutionarily optimal to generate a high level of initial quality, but to let it fall with age. This is because the quality of the relatively small number of cells in the germ line can be independently maintained cheaply, while the quality of the large soma achieved after growth would be much more expensive to maintain.

III. An Example

A key aspect of the model is that growing large militates against the maintenance of quality. We illustrate this aspect of the general theory by means of an example. There are, however, a number of important aspects of the general theory that cannot be

illustrated by this example. For example, the general model involves an endogenous optimal mortality rate, whereas this is fixed and constant in the example.

The elements of the example are outlined in the following five paragraphs.

Each individual has gross energy output given by

(1) $F(K,Q) = aK - bK^2 + cQ - dQ^2, a,b,c,d > 0, K \in [0, a/2b], Q \in [0, c/2d]$

where *K* is the quantity of somatic capital and *Q* is its quality. At the beginning of life, each individual has initial quantity $K_0 > 0$ and initial quality $Q_0 > 0$. Additional investment in quantity takes place all at once at the beginning of life, where the cost of choosing $K \geq K_0$ is $\alpha(K - K_0)$. Investment in quantity is irreversible and not subject to depreciation. This is plausible since quantity is interpreted here as the number of somatic cells.

The quality of somatic capital evolves according to

(2) $$\frac{dQ}{dt} = w - \rho Q, \rho > 0,$$

where $w \geq 0$ is subject to choice. A key aspect of the model is that the cost of a given level of quality improvement is higher, the greater the quantity of somatic capital involved. Suppose, indeed, that choice of $w \geq 0$ entails a cost of *βKw.* This is intended to capture the biological economics of quality maintenance discussed above.

Fertility at age *t* is given by $s(t) \geq 0$ and that the cost of this is $\gamma s(t), \gamma > 0$. This cost includes the cost of K_0 and may include, in addition, a fixed cost that is independent of the level of capital.

Suppose that mortality in this example is fixed and constant at rate μ > 0. The rate of growth of population, which will be the target of natural selection, is *r.* It follows

then that density function of the steady state age distribution at age $t \geq 0$ is given by $e^{-(\mu+r)t}$.

Assuming that transfers of resources can be made freely within the social group yields the following social budget constraint—

(3) $$\int_0^\infty e^{-(\mu+r)t}\left(F - \beta K w - \gamma s\right)dt - \alpha(K - K_0) = 0$$

Under this condition, the adult surpluses cover the deficits of the young. In addition, standard demographic arguments imply that the Euler-Lotka equation

(4) $$\int_0^\infty e^{-(\mu+r)t} s dt = 1$$

must hold.

This completes the description of the set up for the example.

Consider first the evolution of the germ line. This is subject to the same technology for maintaining quality, but is assumed to involve a negligible number of cells. There is then no cost to quality maintenance of the germ line, and it can be maintained at the ideal quality level of $c/2d$. Every individual created from this germ line then has this as the initial level of quality so $Q_0 = c/2d$.

The evolutionary problem for the optimal design of an individual is then to choose $K \geq K_0$ and $w(.) \geq 0$ so as to maximize *r* subject to (2), (3) and (4).

If the parameter β is large enough, but α and K_0 are small enough, the optimal solution involves increasing the quantity of somatic capital, but also allowing the quality of this somatic capital to decline over all ages. (See supplementary online material.)

IV. The Results of the General Model

The model in Robson and Kaplan (2006) incorporates more general formulations of the cost of investment in the quantity of somatic capital, the cost of maintaining its quality, and the cost of fertility. Most crucially, perhaps, mortality is endogenously determined with a convex cost function. Altogether, the general model generates a gross energy flow that is hump-shaped. The phase where energy flow increases is driven by the process of investment in the quantity of somatic capital. This phase is extended relative to the above example. On the other hand, there is still a terminal phase where gross energy decreases and this is driven by decreasing quality of somatic capital. In the general model, fertility is zero at first, but then rises to a maximum at the same age where gross energy reaches a maximum. Fertility subsequently falls, and becomes zero again in the final phase of life, despite a continuing net energy contribution. This aspect of the model helps explain why natural selection extended human lives beyond menopause. Finally, the evolutionarily optimal trajectory of mortality is predicted to be U-shaped, where the age of minimum mortality is no greater than the age at which gross energy reaches a maximum.

More specifically, peak gross energy in the model occurs at the age when somatic growth ceases. Thus the model predicts that maximum fertility should occur at this age of physical maturity. This is consistent with the hunter-gatherer data. For, although women reach maximum height rather earlier, they continue to add body mass until their mid-twenties, and this is when fertility peaks. The model also predicts that the minimum of mortality should occur no later than this same mid-twenties age range. This is also consistent with the hunter-gatherer data, since minimum mortality arises at age 13 or so.

V. Future Research

Two human economic characteristics that seem especially amenable to evolutionary explanation are the rate of time preference and attitudes to risk. Indeed, time preference and attitudes to risk are aspects of general intertemporal preferences, and are best studied together. To illustrate this, note that, if all risk is idiosyncratic, then intertemporal preferences have a familiar representation as the discounted sum of expected utilities, where the constant discount factor is the rate of population growth. On the other hand, if some of the risk is aggregate, such simple results may be lost. (See Robson and Larry Samuelson, 2006).

The example we present here is asexual, so offspring are genetically identical to their parents. Since genetic identity eliminates conflicts of interests, the use of an overall social budget constraint is appropriate. In a sexual model, on the other hand, parents are only one half relatives of their offspring. Such a social budget constraint is then less compelling. It is more appropriate to suppose that individuals transfer resources only to their own descendants, on the basis of incentives weakened by degrees of relatedness that are now less than one. The implications for intertemporal preferences have yet to be fully developed (see, however, Robson and Balazs Szentes, 2006).

REFERENCES

Ehrlich, Isaac and Hiroyuki Chuma. 1990. "A Model of the Demand for Longevity and the Value of Life Extension." *Journal of Political Economy*, 98, 761-782.

Finch, Caleb E. 1998. "Variations in Senescence and Longevity Include the Possibility of Negligible Senescence." *Journal of Gerontology: Biological Sciences*, 53A, B235-B239.

Grossman, Michael, 1972. "On the Concept of Health Capital and the Demand for Health." *Journal of Political Economy*, 80, 223-255.

Hamilton, William D. 1966. "The Moulding of Senescence by Natural Selection." *Journal of Theoretical Biology*, 12, 12-45.

Kirkwood, Thomas B.L. 1990. "The Disposable Soma Theory of Aging." In *Genetic Effects on Aging II*. D.E. Harrison, Ed. Caldwell, N.J.: Telford Press, 9-19.

Robson, Arthur J. and Hillard S. Kaplan 2006. “Why We Grow Large and then Grow Old: Economics, Biology and Mortality.” http://www.sfu.ca/~robson/wwgo.pdf

Robson, Arthur J. and Larry Samuelson, 2006. “The Evolution of Impatience with Aggregate Uncertainty.” Unpublished.

Robson, Arthur J. and Balazs Szentes, 2006. “On the Evolution of Time Preference.” Unpublished.

United States Centers for Disease Control, 2006. “National Vital Statistics Reports: United States Life Tables, 2003.” Vol. 54, No. 14, Washington, DC. http://www.cdc.gov/nchs/data/nvsr/nvsr54/nvsr54_14.pdf

Vaupel, James W., Annette Baudisch, Martin Drolling, Deborah A. Roach and Jutta Gampe. 2004. "The Case for Negative Senescence." *Theoretical Population Biology*, 65, 339-351.

FOOTNOTE

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