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Why do We Die? Economics, Biology and Aging

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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 expected age at death if people could sustain the mortality rate of ten year olds. In the case of the U.S. population in 2002, the expected age at death would be just over 6,600 years. That is, people would not be immortal, because there is always a positive probability of dying; but our lives would be more than 80 times longer if mortality risks did not increase with age. In fact, the oldest 1% of the population would live past 30,000 years of age, instead of just past 100 as they currently do.

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---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 evolutionarily or economically 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 essay 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? Suppose 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. He argues that repair of somatic tissue must be optimized by natural selection. At some point, greater returns will be obtained through reproduction than through repair. Perhaps, then, optimal repair is less than complete and the soma (body) deteriorates with age, ultimately being replaced by descendants. The existence of a line of sex cells---the “germ line”---that is segregated from the line of body cells---the “somatic line”---permits the degradation of the soma. 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 explaining it. This theory also fails to account for an initial phase of life with decreasing mortality.

It is clearly important 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 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

A key feature of the disposable soma theory is the segregation of the germ line and the somatic line. This segregation implies that damage or mutations arising in somatic cells cannot be transmitted to offspring via the sex cells. In our version of this theory (Arthur J. Robson and Hillard S. Kaplan (2006)), the cost of investment in quality depends positively on the quantity of cells, because each cell is subject to deterioration and so generates 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.

The basis of our theory is that organisms invest in somatic (bodily) capital, which is then used to produce energy to support continued life and 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 mass. We are a species with determinate growth, so our somatic cell number increases up to some particular age and then stops. Cell quality, interpreted as functional efficiency in our model, is endogenous and 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.

III. An Example

A key aspect of the model is that growing large militates against the maintenance of quality, despite the optimality of growing large in the first place. We illustrate this aspect of the general theory by means of an example. There are a number of aspects of the general theory that cannot be illustrated by this example, however. For one thing, in the general model, there is an extended phase of investment in the quantity of somatic capital, in addition to the phase of quality deterioration illustrated by the example.

The elements of the example are outlined in the next few 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 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, it follows 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 $Q_0 = c/2d$.

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 is as follows. (See supplementary online material.) Although it is optimal to increase the quantity of somatic capital, this entails that it is now optimal to allow the quality of somatic capital to decline over all ages. Although it is possible to maintain the quality of somatic capital at the original ideal level, or at any intermediate positive level, for that matter, it is simply not optimal to do so. A type that did this would not grow as fast as a type that followed the optimal trajectory, and so the former would lose the evolutionary contest.

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, then rises to a maximum at the same age where gross energy reaches a maximum. It then 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, maximum fertility should occur at the age when somatic growth ceases, or about age 18 for human females. Hunter-gatherer data suggest that maximum realized fertility might be a little later than age 18. Since social pressures might well slightly delay fertility, 18 is still a reasonable estimate of the age at which *potential* fertility is maximal. The model also predicts that the minimum of mortality should occur no later than age 18. This is also consistent with the data, since minimum mortality may arise at age 13 or so.

V. Future Research

Two human economic characteristics that seem especially amenable to evolutionary explanation are rates 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. For example, if the only risk that arises 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 are 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 inter-generational conflicts of interests, the use of a social budget constraint, under which people run deficits at early ages and produce surpluses at older ages, is appropriate. In a sexual model, on the other hand, parents share only half their genes with their offspring, roughly speaking. This provides a quantitative basis for understanding both familial altruism and inter-generational conflicts of interest. The implications for intertemporal preferences have yet to be fully studied (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.

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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TECHNICAL NOTE (NOT FOR PUBLICATION)

It follows immediately that any solution to the problem described in Section III must also solve the following problem

(5) $$\max_{K\geq K_0, w(.)\geq 0} \int_0^{\infty} e^{-(\mu+r)t}\left(F-\beta K w\right)dt-\alpha(K-K_0)=\gamma \text{ subject to } \frac{dQ}{dt}=w-\rho Q.$$

Simplification is possible here since the optimal fertility profile is indeterminate in this example, subject only to satisfying the Euler-Lotka equation, (4).

Given that $Q_0 = c/2d$ and that the cost of quality maintenance is linear in w, the optimal trajectory in (5) for w involves allowing quality to run down until it reaches its optimal steady state level, and then maintaining it at that level. That is, for some $Q^* \geq 0$, $w=0$ until $t=t^*$ but $w=\rho Q^*$ thereafter, where $Q_0 e^{-\rho t^*} = Q^*$. Let $Q(t)$ be the overall time path of quality this implies. Thus, the problem (5) reduces to

$$\max_{K^*,Q^*} V(K^*,Q^*)=\gamma \quad \text{where}$$

$$V(K^*,Q^*)=\int_0^{\infty} F(K^*,Q(t))e^{-(\mu+r)t}dt-\frac{\beta\rho K^*Q^*}{\mu+r}\left(\frac{Q^*}{Q_0}\right)^{\frac{\mu+r}{\rho}}-\alpha(K^*-K_0)$$

Dropping the asterisks on K and Q, and since the upper bound is never binding, the first-order Kuhn-Tucker condition for choice of *K* for this problem is then

$V_K \le 0$ and $V_K(K-K_0)=0$ so $a-2bK-\left(\frac{Q}{Q_0}\right)^{\frac{\mu+r}{\rho}}\beta\rho Q-\alpha(\mu+r)\le 0$ and

$$\left(a-2bK-\left(\frac{Q}{Q_0}\right)^{\frac{\mu+r}{\rho}}\beta\rho Q-\alpha(\mu+r)\right)(K-K_0)=0$$

The first-order condition for choice of Q is similarly

$V_Q \le 0$ and $V_Q Q=0$ so that $c-2dQ-(\mu+r+\rho)\beta K\le 0$ and

$\left(c-2dQ-(\mu+r+\rho)\beta K\right)Q=0$.

There cannot be an interior solution of (5) for Q if $\beta > \frac{c}{(\mu+r+\rho)K_0}$; rather $Q=0$ in this case. There must, on the other hand, be an interior solution of (5) for K as long as α and K_0 are small enough to satisfy $a>\alpha(\mu+r)+2bK_0$.

Finally, note that

$$\frac{d}{dr}\left(\max_{K\ge K_0, w(.)\ge 0}\left[\int_0^\infty e^{-(\mu+r)t}\left(F-\beta K w\right)dt-\alpha(K-K_0)\right]\right)=-\int_0^\infty te^{-(\mu+r)t}\left(F-\beta K w\right)dt$$

Since, furthermore

$$\frac{d}{dt}\left(t\int_t^\infty e^{-(\mu+r)\tau}\left(F-\beta K w\right)d\tau\right)=\int_t^\infty e^{-(\mu+r)\tau}\left(F-\beta K w\right)d\tau-te^{-(\mu+r)t}\left(F-\beta K w\right)$$

then

$$\int_0^\infty te^{-(\mu+r)t}\left(F-\beta K w\right)dt=\int_0^\infty dt\int_t^\infty e^{-(\mu+r)\tau}\left(F-\beta K w\right)d\tau>0$$

because choosing $w=0$ ensures that $\int_t^\infty e^{-(\mu+r)\tau}F d\tau>0$. Hence

$$\frac{d}{dr}\left(\max_{K\geq K_0, w(.)\geq 0}\left[\int_0^{\infty} e^{-(\mu+r)t}\left(F-\beta Kw\right)dt-\alpha(K-K_0)\right]\right)<0.$$

Consider now any solution of the problem in (5). Since increasing *r* must be infeasible, it follows that this solution of (5) is also a solution to the problem posed in Section III.