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The Evolution of Human Life Expectancy and Intelligence in Hunter-Gatherer Economies

By ARTHUR J. ROBSON AND HILLARD S. KAPLAN*

The economics of hunting and gathering must have driven the biological evolution of human characteristics, since hunter-gatherer societies prevailed for the two million years of human history. These societies feature huge intergenerational resource flows, suggesting that these resource flows should replace fertility as the key demographic consideration. It is then theoretically expected that life expectancy and brain size would increase simultaneously, as apparently occurred during our evolutionary history. The brain here is considered as a direct form of bodily investment, but also crucially as facilitating further indirect investment by means of learning-by-doing. (*JEL* A12, J10, P00)

This paper adopts an alternative view of the relationship between economics and biology. Rather than viewing human biological characteristics as effectively exogenous determinants of economic phenomena, we consider how the economics of hunter-gatherer societies shaped economically relevant human biological characteristics by means of natural selection.[1]

In particular, we examine why intelligence and life expectancy were simultaneously exaggerated in human evolutionary history. Indeed these variables are robustly associated across living species, and across primates, in particular. For example, a human brain is at least three times bigger than that of a chimpanzee and humans live about two times longer. To explain this close connection between intelligence and life expectancy, this paper models the brain as a form of capital. It then derives the dynamic paths of investment and of expenditure to reduce mortality, so to better reap the returns on such investment. In addition to new insights into human origins and characteristics, this integration of biology, anthropology, and economics suggests a new approach to economic questions, such as those related to altruism in families, and to age effects on time preference and risk aversion.

Section I of the paper provides the relevant biological and anthropological background. It discusses natural selection of somatic (bodily) tissue, focusing on the brain, mortality rates, and life expectancy. It presents empirical evidence that human evolution has exaggerated both brain size and life expectancy. Furthermore, it shows that the economies of hunter-gatherers rely on skill-intensive food production strategies that would not be viable without massive intergenerational resource flows and exceptional adult life expectancy.

To address this simultaneous exaggeration of brain size and life expectancy, we consider two models. Model 1, in Section II, considers how any somatic investment is related to optimal mortality reduction. Energy production is a function of a somatic capital stock, where this stock can be increased by reinvestment of energy; mortality depends on the energy allocated to mortality reduction. We show that an optimal

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[1] To some extent, the present approach is then consistent with that of Gary S. Becker (1991, for example), who applied economic principles to explain biological phenomena. See also Haim Ofek (2001). Robson (2001) advocates consideration of the connection between biology and economics.

life history has an initial investment phase during which mortality falls, followed by a stationary state with constant mortality. Greater productivity of the somatic capital stock, or a decrease in extrinsic mortality, leads to longer life expectancy and a higher long-run stock.

The brain has distinctive properties as a somatic stock that are implied by the observations of hunter-gatherer societies but not fully captured by Model 1. That is, the overall lifetime investment associated with the brain seems to have two interrelated stages. In the first of these stages, metabolic resources build brain tissue itself, as with any somatic tissue. What distinguishes investment in the brain qualitatively from other forms of somatic investment, however, is that a larger brain does not immediately imply higher output. Rather, in the second stage of overall investment, a larger brain makes learning more effective. This induces a steeper rise in productivity over time since cognitively demanding activities such as hunting and gathering now become more productive with practice.

Model 2, in Section III, is tailored to focus on this second stage of investment. Somatic investment in the brain is confined to an initial period. Production is a function of time as well as of the brain as a neural capital stock. For any given size of this stock, production initially rises over time, thus capturing learning-by-doing. In a terminal stage, production falls, thus also allowing for aging. We show that these assumptions imply U-shaped mortality, the typical biological pattern. The advent of a more learning-intensive environment leads to the simultaneous exaggeration of longevity and intelligence, as does a reduction in extrinsic mortality. It is finally shown how profiles of net output that stem from greater somatic investment in the brain may imply mortality profiles that are "rectangularized"—U-shaped rather than V-shaped.

Section IV concludes with a discussion of other applications of this synthesis of biology, anthropology, and economics. For example, Models 1 and 2 are also relevant to voluntary choice as well as long-term evolution and so apply to the interrelationship between health and educational investments, for example. Perhaps the most promising further applications, however, consider the biological shaping of intergenerational altruism, time preference, and risk aversion as functions of age. Finally, it is argued that these models suggest why new research is desirable on the theory of aging, either for organisms or machines.

I. Biological and Anthropological Background

A. *Life History Theory and the Economics of Energy*

Life history theory is the branch of biology that considers how evolution shapes the timing of life events. Central here is explaining how the optimal timing of growth and reproduction depends on energy production and mortality rates. (See Stephen C. Stearns, 1992, for example.) The fundamental trade-off is between current and future reproduction. By growing, an organism can increase future energy intake and thus future fertility. Similarly, a repeatedly reproducing organism should reserve some energy during reproduction for health maintenance so that it can live to reproduce again. Over the entire lifetime, natural selection would, in some circumstances, maximize total expected energy surplus available for reproduction.

Growth of any type of body tissue can be seen as investment in somatic capital. Since such stocks tend to depreciate over time, maintenance can also be seen as investment. In a physical sense, somatic capital includes organized tissue such as muscles or the brain. In a functional sense, somatic capital includes strength, coordination, skill, and knowledge. Thus, the present-future reproductive trade-off can be viewed as current reproduction versus investment in somatic capital.

Although life history models often have an underlying logic of investment, economic theory has not been fully exploited here. A contribution of the present paper is then to apply the economic theory of capital to somatic investment in the brain. Moreover, despite the centrality of mortality profiles to life history, these have usually been treated as exogenous. Another contribution of the present paper is then to consider how natural selection shaped the profile of mortality rates and hence affected life expectancy. The basis of this explanation is the variation of economic value of life over the life span. Most significantly, perhaps, the current approach can then explain why intelligence and life expectancy would "coevolve," or be

simultaneously exaggerated. These two characteristics of humans are now considered further.

B. *Natural Selection on the Brain and Life Expectancy*

The brain is a special form of somatic capital. On the one hand, neural tissue monitors the organism's internal and external environment, and induces immediate physiological and behavioral responses. Presumably for this reason, there is a strong tendency for brain size to increase with body size. More relevantly here, the brain also transforms present experiences into future performance by means of learning. To assess the relationship of this function with brain size, it is necessary to control for body weight, and a typical means of doing this is as follows. Regression of the logarithm of brain weight on the logarithm of body weight across all mammals yields a highly significant "allometric" coefficient of 0.76. Robert D. Martin's (1981) "encephalization quotient," EQ, is then the actual brain weight relative to the brain weight predicted from body weight, or $EQ = (\text{Brain weight})/11.22 \times (\text{Body weight})^{0.76}$. Primates, especially monkeys and apes, have higher EQs than other mammals. More specifically, it is the neocortex that is exaggerated in primates. Comparisons across primate species show that the relative size of the brain and, especially, of the neocortex, are associated with the duration of cognitive development and the highest levels of cognition attained. (See Harry J. Jerison, 1973; Sue T. Parker and Michael L. McKinney, 1999.)

The action of natural selection on the neural capital involved in memory and learning depends on the benefits and costs arising over the organism's lifetime. The benefits of learning arise from increased productivity that occurs mainly when the organism is relatively old, perhaps well beyond the age at which the benefits of overall body size peak. On the other hand, although there are substantial energetic costs of maintaining the brain throughout life, brain growth itself is heavily concentrated in the earliest years. Altogether, in the first year of human life, about 65 percent of all resting energetic expenditure is devoted to the growth and maintenance of the brain (Malcolm A. Holliday, 1978). Thus there is a huge time lag between the incurring of the direct metabolic cost of a large brain and the ultimate payback in the form of higher output. Investment in the brain is, in this sense, a more "roundabout" biological strategy than is investment in the body in general.

Since the brain increases future productivity, the expected return from the initial investment depends crucially on the probabilities of being alive in the future. Such survival probabilities are also subject to natural selection. The effectiveness of the immune system, for example, depends on the metabolic energy devoted to building and maintaining the antibodies for defense against pathogens.

There is, indeed, a strong empirical relationship between brain size and life expectancy. (John Allman et al., 1993, show that brain size significantly influences life span, controlling for body size, and Robert A. Barton, 1999, summarizes the literature.) Such a relationship for primates can be shown independently as follows.[2] There are 234 species of primates, altogether, but data on life span, body weight, and brain weight could be obtained for only 101 of these, apart from humans.[3] We found that the partial correlation between the logarithm of brain weight (LBRWT) and the logarithm of maximum life (LNMAX), while controlling for the logarithm of body weight (LNBW), is 0.4655, with a significance level of zero to three decimal places. Moreover, brain weight seems to have a more substantial effect in promoting longevity than does body weight, while allowing for the strong influence of body weight on brain weight. An instrumental variables approach, where the exogenous instruments are the logarithms of body weight, range size, and the percentage of fruit in the diet, as well as the evolutionary category or grade, yields the following result: $LNMAX = \underset{(8.584)}{-3.084} + \underset{(3.696)}{0.516} LNBRWT \underset{(-2.075)}{-0.222} LNBW$. Indeed, body size has a negative estimated effect on maximum life in this structural equation. The t-statistics are given in parentheses; adjusted R^2

[2] These effects are not at all limited to primates, but also arise in birds. Parrots, for example, engage in a great deal of extractive foraging. They are also extreme among birds in terms of both relative brain size and longevity. Terrestrial galliforms, such as quail, that simply peck at foods on the ground are much shorter-lived and smaller-brained. (See Kaplan and Robson, 2002.)

[3] The figure of 234, which includes humans, is from Noel Rowe (1996). Humans were omitted here to allay any suspicion that the results relied on this single observation of both high brain weight and great life expectancy.

is 0.446. The coefficient of LNBRWT, in particular, has a significance level of zero to three decimal places.

C. *The Human Case*

Interest in the interaction between intelligence and life expectancy is enhanced by what is known about human evolutionary history. Within primates in general, there have been three shifts to longer lives and larger brains—from prosimians to monkeys, from monkeys to apes and, of particular interest, from apes to humans.

Our genus *Homo* has indeed only existed for about two million years. Figure 1 shows the dramatic shift from an EQ of just over two for earlier hominids, represented by *Australopithecus boisei,* to 3.5 for the earliest *Homo.* It also exhibits the subsequent rapid increase of EQ in *Homo,* especially during the second half of this period. Much of this hominid brain expansion occurred in the neocortex, the part of the brain most implicated in cognitive ability. It is likely that part of the resulting increase in human intelligence is due to reorganization of brain tissue rather than weight per se. Possibly for this reason, the small differences in brain weight within modern humans may be only weakly correlated with intelligence. Nevertheless, these large increases in brain size on the species level are almost certainly associated with increased learning and information-processing abilities. (See Simon M. Reader and Kevin N. Laland, 2002.)

Life spans of extinct species are not directly observable, of course. Nevertheless, indirect evidence suggests that the life span of *Australopithecus* was much less than that of modern humans, being comparable instead to that of modern chimpanzees, with early species in the genus *Homo* having intermediate life spans. (See B. Holly Smith, 1991, for example.)

What environmental shift might have induced these increases in encephalization and life span? Why would this shift have affected humans rather than chimpanzees, for example? In the first place, the shift to bipedalism already evident in *Australopithecus* almost certainly reflected greater time in more open, less-forested habitats. Bipedal walking is more energetically efficient than knuckle walking, but is disadvantageous in the trees. At the beginning of the

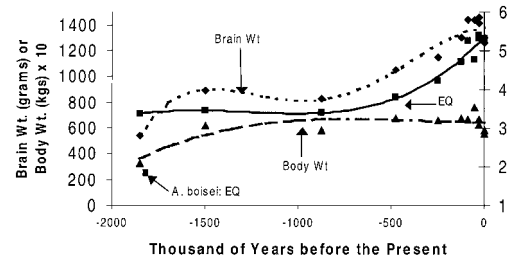

FIGURE 1. HOMINID BRAIN SIZE AND BODY WEIGHT

Pleistocene about two million years ago, lower rainfall led to the emergence of large savannahs, where the animal biomass increased dramatically and many plants evolved to store energy in the form of tubers and nuts. This opened up a new niche in which a bipedal ape had a comparative advantage. With free hands and an already considerable intelligence, early hominids could extract these tubers and nuts. Hunting and scavenging opportunities also increased with animal density, and hominids began to specialize in a niche emphasizing large, calorie-rich plants and animals. (See Robert Jurmain et al., 2000, for example.) Such filling of a new evolutionary niche may be analogous to how firms with existing relevant physical and human capital assets might have a comparative advantage in the production of a new product.

The main point is that, compared to other primates and mammals, human foragers use the most skillful and learning-intensive techniques to get the most nutrient-dense food from the environment. When they gather, humans use digging sticks to obtain underground tubers, stones and other tools to extract protein-rich nuts, and use complex methods to leach poisons out of energy-rich foodstuffs.

Human hunting is particularly cognitively demanding, applying techniques that are varied and creative. Kaplan et al. (2000, p. 171) contains the following examples of these techniques among the Ache, Hiwi, Machiguenga, and Yora:

> Arboreal animals are shot with arrows from the ground or in a tree, driven by climbing, shaken down from branches, frightened into jumping to the ground, brought down by felling the tree with an axe, lured by imitating calls, lured by making captured infants emit distress calls, captured by spreading sticky resin on branches to trap

them, and captured by constructing scaffolding from tree branches and vines. Ground dwelling prey are shot with arrows, driven to other hunters or capture devices, run down upon encounter, slammed to death against the ground, strangled around the neck, or suffocated by stepping on them while trapping them in a tight spot. Burrowing prey are dug out, chopped out of tree trunks, stabbed through the ground with spears, frightened to the point they bolt from the burrow, smoked out, and captured by introducing a lasso through a small hole. Aquatic prey are shot on the surface, driven into traps, poisoned, shot below the surface, discovered on muddy bottoms by systematically poking the bottom of a pond, and speared underwater by random thrusts in drying lakes.

The wide diversity of behavior here would be prohibitively expensive for evolution to program directly; rather it reflects the evolution of a large brain with the ability to learn general rules and to solve particular problems.

These hunting and gathering strategies produce a life history with dramatic dynamic trade-offs. Figures 2A and 2B (adapted from Kaplan et al., 2000) provide evidence of these trade-offs for the Ache, Hiwi, and Hadza, also providing a comparison to chimpanzees in the wild.

An adolescent human male is a substantial drain on society, but a mature hunter provides sufficient calories to support several individuals. Since the lower female food productivity curve can be attributed to raising children and to other tasks such as food preparation, the male curve is taken to represent total production of either sex. This steeply rising male curve indirectly reflects the biological formation of the brain early in life, while directly reflecting the learning-by-doing when hunting that this brain allows. (There is a contribution here from increasing physical strength, but this peaks more than a decade before maximum productivity is reached. Note also that the growth in male output is not due to an increase in the number of hours working, but to an increase in productivity per hour. See Kaplan et al., 2000.)

What is the evidence on the life expectancy of our species? Figure 3 shows that hunter-gatherers (the Ache) are subject to significantly lower mortality than are chimpanzees in the

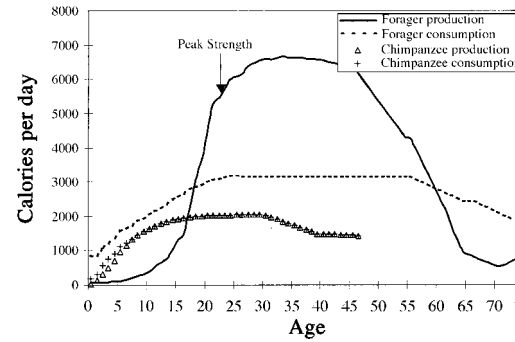

FIGURE 2A. MALE PRODUCTION AND CONSUMPTION

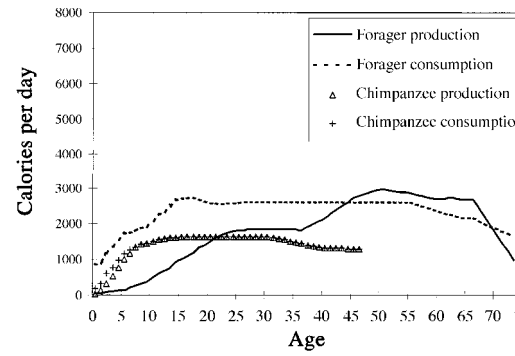

FIGURE 2B. FEMALE PRODUCTION AND CONSUMPTION

wild.[4] It also shows that, although the mortality curve for chimpanzees is V-shaped, the curve for humans is more U-shaped. Human mortality is relatively high during infancy, but falls sharply until age 5 or so. There is then a long period during which mortality at first slowly falls and then slowly rises, followed finally by a sharp rise in old age after age 65 or so.[5]

Such patterns of mortality are often taken as exogenous in biology.[6] However, the present paper can help answer two additional questions: *Why* does mortality fall and then rise? *Why* is human mortality *more* U-shaped than is chimpanzee mortality?

[4] This is adapted from Kaplan et al. (2000). Hill and Hurtado (1996) analyze detailed data on the life course of the Ache.

[5] The transformation of the chimpanzee mortality profile into that for humans is then an example of "rectangularization," as is discussed further in Section IV.

[6] An exception is Mariusz Cichon (1997), who takes the *increase* in the mortality rate to be determined by the resources dedicated to this.

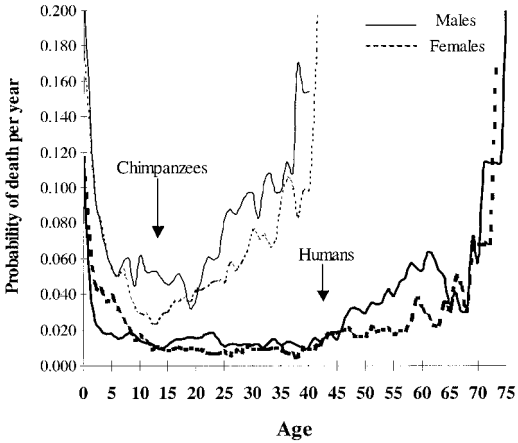

FIGURE 3. YEARLY MORTALITY

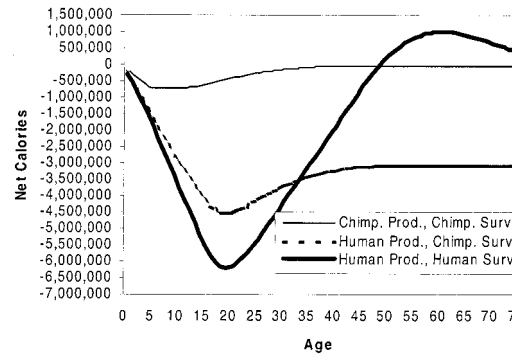

FIGURE 4. CUMULATIVE EXPECTED ENERGY SURPLUS BY AGE: HUMANS AND CHIMPANZEES

Using both productivity and mortality data, Figure 4 (adapted from Kaplan et al., 2001) is an explicit representation of the huge intertemporal trade-off in hunter-gatherer life history. This figure plots cumulative net food transfers weighted by the probability of survival as a function of age, for both sexes in all hunter-gatherer societies for which data exist. Inclusion of the intergenerational transfer represented by the female provision of child care would reinforce the huge trade-off already evident here. It takes until about age 50 to break even and the ultimate excess is relatively small.

Figure 4 also vividly illustrates the essential way in which intelligence and mortality are intertwined. If this calculation is performed using human net transfers but chimpanzee mortality, the curve remains in substantial deficit, which would imply a rapidly decreasing population. Assuming that chimpanzee mortality approximates the mortality of human evolutionary ancestors, this ancestral mortality could therefore not have supported the emergence of human intelligence and the associated economic life history. The evolution of these human characteristics then necessitated a substantial concurrent reduction in mortality.

Consider now two formal models of the coevolution of intelligence and life expectancy.

II. Model 1—General Somatic Capital

At each point in time, an individual has a somatic capital stock, $K > 0$, producing a stream of energy output, $F(K)$. The initial capital stock is $K(0) = K_0 > 0$. The production function F satisfies standard restrictions as follows.

ASSUMPTION 1: *F is such that $F(0) = 0$; $F'(K) > 0$ and $F''(K) < 0$, for all $K > 0$; $F'(K) \uparrow \infty$, as $K \uparrow 0$ and $F'(K) \uparrow 0$, as $K \uparrow \infty$.*

The energy output can be used as investment, v, then augmenting the capital stock according to $\frac{dK}{dt} = v$, where $v \in [0, \bar{v}]$.[7] The upper bound on the investment rate, $\bar{v}$, represents a simple type of increasing cost of investment, as is compelling here.

Some of the energy output, $s \geq 0$, can instead be used to reduce the instantaneous mortality rate, $\mu(s)$, which is a decreasing convex function of s, reaches a minimum level at some $\bar{s}$, and is such that only interior solutions for s typically arise.

ASSUMPTION 2: *μ is such that $\mu'(s) < 0$, $\mu''(s) > 0$, for all $s \in (0, \bar{s})$; $\mu(s) \uparrow \mu(0) < \infty$, $\mu'(s) \uparrow -\infty$, as $s \uparrow 0$; $\mu'(\bar{s}) = 0$ and $\mu(s) = r > 0$, for all $s \geq \bar{s}$.*[8]

[7] Despite the empirical relevance of brain maintenance, capital does not depreciate here, for simplicity. However, the model can be easily modified to allow capital to depreciate at a constant rate with little qualitative change in the results.

[8] If K represents brain mass, it seems plausible that μ should be independent of K. If K represents body size, μ should perhaps depend on K as well as s. In this case, it is

This mortality rate has a mathematical effect similar to that of a time-varying interest rate. In the same way that an overall discount factor for time-varying interest is calculated, the overall probability of survival to age t, $p(t)$, say, satisfies $\frac{dp(t)}{dt} = -p(t)\mu(s)$ where $p(0) = 1$. The energy surplus flow at age t is then $y(t) = F(K(t)) - v(t) - s(t)$. Finally, it is assumed that the initial production of each individual costs $C_0 \geq K_0 > 0$. The biologically appropriate objective criterion is the topic of the next subsection.

Consider first how the theory here might be reconciled with the observations in Section I, subsection C. For this purpose, $F(K)$ is identified with observed production, $v + s$ with observed consumption, so that y is the observed net transfer, as in Figures 2A, 2B, and 5.[9]

A. *The Objective Criterion*

By way of introducing the appropriate criterion here, consider a species in which individuals have probability $p(t)$ of reaching age t, when they produce expected offspring $m(t) \geq 0$. What steady-state rate of population growth, ρ, say, does this allow? If the total birth rate is $Q(\tau)$ at time τ, say, the birth rate at a time $\tau - t$ must have been $Q(\tau)e^{-\rho t}$, given steady-state growth. Hence the density of individuals of age t at time τ is $p(t)Q(\tau)e^{-\rho t}$ and the contribution of these individuals to the birth rate at time τ is $m(t)p(t)Q(\tau)e^{-\rho t}$. The steady-state growth rate, ρ, is then the unique solution of the "Euler-Lotka equation" $\int_0^\infty e^{-\rho t}p(t)m(t)\,dt = 1$. (See Stearns, 1992, for example. It also can be shown that the age distribution generally converges to the steady-state distribution from an arbitrary initial configuration.) If there are a number of types, any type with the maximal growth rate will ultimately dominate other types with lower rates, so the steady-state growth rate is the appropriate criterion for biological success.[10] The Euler-Lotka equation focuses on the biological reproduction of individuals as the crucial characteristic that limits and determines the growth rate.

However, this conventional biological approach ignores the crucial energetic constraints on population growth. This seems especially an issue for human beings, who invest heavily in their young. The following economic approach, where the resource flows between generations are the crucial limiting characteristic, is then more appealing. Suppose individuals have a probability of survival profile p and a surplus energy profile y, where initial production of each individual costs C_0. What steady-state growth rates, ρ, are then feasible? Reasoning along similar lines as before, if the birth rate at time τ is $Q(\tau)$, say, the net economic surplus derived from y at time τ is $\int_0^\infty Q(\tau)e^{-\rho t}p(t)y(t)\,dt$. Since energy expenditure on newborns is $Q(\tau)C_0$, a growth rate ρ is economically feasible if and only if $\int_0^\infty e^{-\rho t}p(t)y(t)\,dt \geq C_0$. The problem then becomes that of finding the maximum growth rate that is economically feasible over choice of p and y that satisfy the constraints of the model.

Implicit in this approach is that individuals who have energy deficits, typically the young, have these deficits made up by the energy surpluses of other, typically older, individuals. The food-sharing in hunter-gatherer societies performs such redistribution on a large scale, as Figures 2A and 2B imply. In a snapshot of such a society, there is an age distribution of individuals, each with a characteristic energy surplus or deficit. Since there is essentially no storage of food, instantaneous feasibility dictates that these surpluses must be nonnegative in the aggregate.

From the lifetime point of view of an individual in such a society, this redistribution functions as a loan. That is, in exchange for a subsidy while young, the individual makes repayments while older. What compels older individuals to repay the loan they were given while young? The answer is that individuals have a biological motive to foster their own offspring. Indeed, natural selection would act

clear that mortality will tend to fall during the initial growth phase. (Richard Sibly et al., 1985, present such a model.) In any case, the present model shows that the initially increasing *value* of an individual suffices for this result.

[9] The cost C_0 is interpreted as the metabolic transfer from mother to the unborn infant, which is small compared to the transfers from parents to children.

[10] Such a growth rate criterion would determine biological success in the absence of overall resource constraints. If these were modeled explicitly, greater attention would need to be paid to the interactions within and across species.

on genes influencing resource transfers between generations. Genes maximizing their representation in future generations would be those generating such parental concern. The social food-sharing arrangements of hunter-gatherer societies smooth out this intergenerational transfer and are presumably Pareto improving, but they do not change its basically selfish nature.[11]

To illustrate the distinctness of the present approach, note that it recognizes that humans have economic value, and *therefore* biological value, after the age when their own reproduction ceases. From the point of view of the Euler-Lotka equation, it is mysterious why individuals should even live beyond this age; from the present economic point of view, the biological purpose of this is clear: It is to provide resources to offspring.

Although the growth rate criterion is unusual in economics, it is straightforward and convenient to transform it into a typical economic one, as follows:

LEMMA 1: *If p and y generate the maximum growth rate $\bar{\rho} \in (-\infty, \infty)$, then p and y maximize $\int_0^\infty e^{-\bar{\rho}t} p(t) y(t)\, dt$ at the value C_0.*[12]

PROOF:

For example, if p and y generate $\bar{\rho}$, but $\int_0^\infty e^{-\bar{\rho}t} p(t) y(t)\, dt > C_0$, then $\int_0^\infty e^{-\rho t} p(t) y(t)\, dt > C_0$ for some $\rho > \bar{\rho}$, and a rate greater than $\bar{\rho}$ is possible.

Further, since the actual growth rate of hominids over the last two million years must have been essentially zero, it is assumed that $\bar{\rho} = 0$. Lemma 1 then motivates considering the maximization of total expected energy surplus $\int_0^\infty p(t) y(t)\, dt$ subject to the above model.[13] Theorem 1 below ultimately rigorously justifies this approach by proving that this transformed problem has a unique solution, and, as long as $\int_0^\infty p(t) y(t)\, dt = C_0$, that this solution also generates the maximum growth rate of zero.[14]

B. *A Key Steady State*

As the first step to solving the problem of maximizing total expected energy surplus, consider states in which capital is fixed at K, so investment v is zero, and where expenditure on mortality reduction, s, is also constant. Take then the optimal such s and so define

$$(1) \quad W(K) = \max_{s \geq 0} Z(K, s),$$

$$\text{where } Z(K, s) \equiv \frac{F(K) - s}{\mu(s)}.$$

That is, since the constant flow of energy surplus in such a steady state is $y = F(K) - s$ and the constant mortality rate $\mu(s)$ has the same mathematical effect as a constant interest rate, $Z(K, s)$ is total expected energy surplus in such a steady state.

LEMMA 2: *Given Assumptions* 1 *and* 2, *a unique optimal s in* (1) *exists for all $K > 0$, given by $s(K) \in (0, F(K))$, say, the unique solution of $Z_s(K, s) = 0$. Furthermore,* $\dfrac{ds(K)}{dK} > 0$. *Hence*

[11] Another major function of food sharing in hunter-gatherer societies is to spread risk, since, for example, the return from hunting large game is highly variable. (See Kaplan and Hill, 1985, for example.)

[12] Although $\bar{\rho}$ is an *unknown* parameter, it turns out to be sufficient in general to solve this transformed problem. Observe also that $\bar{\rho}$ functions as a source of impatience. An additional source is that, whereas energy in youth favors one's own reproduction, that in old age benefits the reproduction only of one's offspring, who are only ½ relatives under sexual reproduction. Alan Rogers (1994) derives the rate of time preference from such considerations, which are neglected here, for simplicity.

[13] Although the average growth rate must have been close to zero, there might still have been periods of positive growth interrupted by occasional crashes. As long as the crashes were selectively neutral, the appropriate growth rate might then be positive. This makes little qualitative difference to the results, however, since a positive growth rate could be incorporated into the definition of the mortality rate.

In terms of Oded Galor and David N. Weil (2000), the regime here is "Malthusian." The effect here that an environment promoting learning induces a larger brain is formally reminiscent of their effect that technological change spurs education, in the "Modern Growth Regime." Galor and Weil, however, do not allow endogenous mortality. See also Galor and Omer Moav (2002).

[14] Total offspring is also maximized, at the value 1, by choice of the optimal profiles p and y. Such biological maximization problems, that also specify the maximized value, are reminiscent of perfect competition, where firms maximize profits, and profits are zero.

higher gross output, $F(K)$, implies higher optimal expenditure on mortality reduction.

PROOF:

The proofs of this and all the subsequent Lemmas and Theorems are in the Appendix.

It follows from Assumptions 1 and 2 that $\lim_{K\uparrow 0} W'(K) = \infty$ and that $\lim_{K\uparrow \infty} W'(K) = 0$. For technical convenience, it is further assumed that:

ASSUMPTION 3: *There exists a unique $K^* > 0$ such that $W'(K^*) = 1$ and $W''(K^*) < 0$.*[15]

The condition $W'(K^*) = 1$ characterizes an optimal steady state. [The marginal cost of increasing the capital stock is 1, since this must be obtained by sacrificing current energy output, whereas the marginal gain is $W'(K)$.] That is, it will be shown that the steady state with capital K^* and the associated $s(K^*) = s^*$, say, describes long-run behavior in any optimal life history.

C. *The Optimal Life History*

Suppose first that $K_0 \geq K^*$, where K^* satisfies $W'(K^*) = 1$. This implies that $W'(K_0) \leq 1$, so the return from an increment in capital is less than its cost in terms of forgone output now. Set then $v(t) \equiv 0$, and $s(t) \equiv s(K_0)$. If $K_0 < K^*$, instead, invest so as to converge as fast as possible to the steady state with capital K^* and expenditure on mortality reduction s^*. Define then $t^* = (K^* - K_0)/\bar{v}$, and take the investment path as

$$v(t) = \begin{cases} \bar{v}, & \text{for } t < t^* \\ 0, & \text{for } t \geq t^* \end{cases}.$$

The candidate path of mortality reduction expenditure for $K_0 < K^*$ can be motivated as follows. Suppose that the total expected future energy surplus, or the "value of a life," at age t, is $J(t) = (p(t))^{-1} \int_t^\infty p(\tau) y(\tau)\, d\tau$. Hence the value of a life evolves as $\dfrac{dJ}{dt} = -y + J\mu(s)$. Given J, the optimal expenditure on mortality reduction, s, is determined by $-J\mu'(s) = 1$. That is, the flow of gain from an increment in s should equal its cost flow, 1. Altogether, then:

$$(2) \quad \frac{dJ}{dt} = -F(K) + v + s + J\mu(s)$$

$$\text{where } -J\mu'(s) = 1.$$

This problem is now shown to have a unique solution. It is convenient to establish two of its properties at the same time.

LEMMA 3: *The problem (2) has a unique solution for s, where $\dfrac{ds}{dt} > 0$, and $s > s(K)$, for all $t \in [0, t^*)$.*

Thus the candidate path for expenditure on mortality reduction, s, over the initial phase is strictly increasing and s is always greater than the level that would be optimal if investment were to cease immediately.

These observations illuminate typical biological patterns of initial mortality. That is, expenditure on mortality reduction is governed by the value of life, J. This is initially increasing, and mortality is falling, because somatic investment is occurring. Similarly, it is plausible that mortality expenditure should be increased by the anticipation of the higher somatic capital stocks that will be accumulated.

Equation (2) is Bellman's equation. Although this is a necessary condition for optimality only under undesirably strong assumptions, it is sufficient in a satisfactory sense:

THEOREM 1: (a) *The functions $v(t)$ and $s(t)$ described above imply time paths for $K(t)$ and $p(t)$ which maximize $\int_0^\infty p(t) y(t)\, dt$ over the set of feasible absolutely continuous paths. This solution is essentially unique.* (b) *If $C_0 = \int_0^\infty p(t) y(t)\, dt$ for this solution, then this solu-*

[15] Note that $W''(K) = \dfrac{F'(K)^2}{\mu(s(K))}\left[\dfrac{F''(K)}{F'(K)^2} - \dfrac{(\mu'(s(K)))^3}{\mu''(s(K))}\right]$. A condition on the mortality function that is sufficient for the *global* concavity of W, for a given concave F, is that $-\dfrac{\mu'(s)^3}{\mu''(s)} \leq U$, for all $s \in [0, \bar{s}]$, where $U > 0$ is small enough. There are functions satisfying Assumption 2 and this requirement.

tion also maximizes the associated growth rate, as in Section II, *subsection* A, *and this maximum growth rate is zero.*

This first result extends standard economic growth theory by allowing an endogenously chosen mortality (or interest) rate. Despite this, the optimal path converges to a steady state in a familiar way. The second result is that this solution to the problem of maximizing total expected energy surplus also provides the solution to the basic biological problem of maximizing the growth rate.

D. *More Productive Capital; Lower Extrinsic Mortality*

How does a parametric decrease in mortality or a parametric increase in production affect the choices of s and K as functions of time? Introduce a parameter α into the production function such that an increase in α means that output rises and the marginal product of capital does not fall. Suppose also that mortality has an "extrinsic" component given by β, where μ is now considered "intrinsic mortality."[16]

ASSUMPTION 4: (a) *The production function is now* $F(K, \alpha)$, *for* $\alpha \in \mathcal{A}$, *where* $\mathcal{A}$ *is a compact interval, such that* (i) $F(\cdot, \alpha)$ *satisfies Assumption* 1 *for each* $\alpha \in \mathcal{A}$, (ii) $F_{\alpha}(K, \alpha) > 0$ *and* $F_{K\alpha}(K, \alpha) \geq 0$, *for all* $\alpha \in \mathcal{A}$ *and* $K > 0$. (b) *The mortality function is now* $\mu(s) + \beta$, *where* $\beta \in \mathcal{B}$, *for* $\mathcal{B}$ *a compact interval, and* μ *satisfies Assumption* 2.

Consider first a change in the parameter α to reflect greater productivity. Denote the entire time path of s as $s(t, \alpha)$.

THEOREM 2(a): *In Model* 1, *with these parameters added,* $\dfrac{\partial s(t, \alpha)}{\partial \alpha} > 0$, *for all* $t \geq 0$, *and* $\dfrac{dK^*}{d\alpha} > 0$.

In particular, even if productivity increases across the board, so that the marginal product of capital is not affected, long-run mortality falls. The reason is that such a productivity increase raises the value of life. This decrease in long-run mortality then causes the long-run level of capital to rise. These effects are only strengthened if the marginal product of capital is increased by the productivity increase. Indeed, such an increase in productivity reduces mortality at *every* age.

Consider now changing the parameter β that represents extrinsic mortality.[17] Denote the entire time path of s as $s(t, \beta)$.

THEOREM 2(b): *In Model* 1, $\dfrac{\partial s(t, \beta)}{\partial \beta} < 0$, *for all* $t \geq 0$, *and* $\dfrac{dK^*}{d\beta} < 0$.

That is: A decrease in extrinsic mortality, β, leads to a further *amplifying* decrease in intrinsic long-run mortality, $\mu(s^*)$. Although such a result might seem at first contrary to economic intuition, it follows here just because such a fall in β must increase the value of life at each age. The reduction in overall mortality leads to an increase in long-run capital, K^*. Indeed, a reduction in extrinsic mortality causes a reinforcing reduction in intrinsic mortality at *every* age.

These comparative static results for Model 1 are further discussed in Section III, subsection B, together with the related results for Model 2.

III. Model 2—Specific Neural Capital

Especially when the somatic capital in Model 1 is interpreted as the brain, this model does not account for key features of the lifetime production profiles in Figures 2A and 2B and the mortality profiles in Figure 3. First, it does not generate the steep rise in output evident during adulthood among men. Growth in physical strength is initially a factor here, but, since this peaks at about age 22, the subsequent rise in output seems to be due to learning-by-doing.

[16] The additively separable form for mortality can be relaxed to allow a function of the form $\mu(s, \beta)$, where $\mu(\cdot, \beta)$ satisfies Assumption 2, $\mu_{\beta}(s, \beta) > 0$ and $\mu_{s\beta}(s, \beta) \geq 0$.

[17] Either an increase in α or a decrease in β by itself implies that positive population growth would occur. Zero growth should be reestablished. Although this could be done in general via density dependence of the production or mortality functions, it is assumed here that this is done by increasing C_0.

This delayed growth in productivity implies that investment in neural capital induces a significant dynamic trade-off after physical growth of the brain is complete. Indeed, this consideration distinguishes the brain from most other forms of somatic capital.[18] Secondly, Model 1 does not account for the declining output and rising mortality most evident beyond age 65 or so.

Model 2, in which output varies over time with a constant stock, addresses these issues and permits the focus to be specifically on neural capital. If now the brain has fixed size K, production at date t is $F(K, t)$, say. There is an initial range over which $F(K, t)$ rises. One factor that would tend to induce this rise is continuing overall bodily growth, where $F(K, t)$ is interpreted as output less investment in other nonneural somatic stocks.[19] However, the key factor leading to rising productivity, even after all somatic investment is complete, is learning-by-doing. Thus a large brain, K, induces high levels of $F(K, t)$, which only become evident at ages, t, long after brain growth itself has ended. Finally, senescence is captured in a terminal range of ages over which $F(K, t)$ declines.

ASSUMPTION 5: (a) (i) *There is some* $T > 0$ *and, for each* $K > 0$, *some* $\hat{t}(K)$, *where* $T > \hat{t}(K) > 0$, *such that* $F(K, 0) = 0$; $F_t(K, t) > 0$, *for all* $t \in [0, \hat{t}(K))$; $F_t(K, \hat{t}(K)) = 0$ *and* $F_{tt}(K, \hat{t}(K)) < 0$; $F_t(K, t) < 0$, *for all* $t \in (\hat{t}(K), T]$; *and* $F(K, t) = 0$, *for all* $t \geq T$.[20] (ii) $F(\cdot, t)$ *satisfies Assumption* 1, *for each* $t \in (0, T)$. (b) *Mortality* μ *satisfies Assumption* 2.[21]

For analytic simplicity, neural investment $\frac{dK}{dt} = v$, is now constrained to occur in a block of time at the beginning of life.[22] That is, for some $t \geq 0$,

$$v(\tau) = \begin{cases} \bar{v} > 0 & \tau \in [0, t) \\ 0 & \tau \geq t \end{cases}.$$

There is again an initial cost $C_0 \geq K_0 > 0$ for each individual.

Lemma 1 remains valid, so that any end date for investment, t, and any mortality reduction expenditure profile, s, that maximize the steady-state growth rate, also maximize total expected energy surplus, discounted using this growth rate. Assuming again that this maximum growth rate is zero, Model 2 then suggests considering the following "overall problem:"

$$(3) \quad \max_{t \in [0,T]} M(t),$$

$$\text{for } M(t) = \max_{s} \left[\int_0^t p(\tau)(F(K_0 + \bar{v}\tau, \tau) - s(\tau) - \bar{v})\, d\tau + p(t) W(K_0 + \bar{v}t, t) \right],$$

$$(4) \quad \text{where } W(K, t) \equiv \max_{s} (p(t))^{-1} \times \left(\int_t^T p(\tau)(F(K, \tau) - s(\tau))\, d\tau \right),$$

for all $K > 0$, and $p(t) = \exp - \int_0^t \mu(s(\tau))\, d\tau$, for all $t \in [0, T]$. Theorem 3 rigorously justifies this approach by showing that this overall problem has a unique solution and, given the maximum total expected energy surplus equals C_0, that this solution attains the maximum growth rate of zero.

[18] Some such lag between the end of direct investment and the peak of output might arise with other kinds of somatic capital, if investment in one kind should precede investment in another. For example, perhaps bones should be fully formed before muscular growth and development can be completed. The magnitude of this lag might only be several years, however, contrasting with the 25–30 years between the end of direct neural investment and the peak output it permits.

[19] In general, $F(K, t)$ could be negative initially, as long as $M(0)$ remains nonnegative, where $M(t)$ is introduced below.

[20] For simplicity, T is independent of K.

[21] It might be realistic for μ to be time dependent, tending to rise at the end of life. It is not *necessary* to assume this, however, to generate eventually rising mortality.

[22] This assumption seems biologically realistic. However, if the production function yields low enough output for a long enough time initially, but then rises rapidly, unconstrained optimal investment involves delay. On the other hand, there are production functions for which the optimal investment path involves a block of maximal investment initially, as assumed here.

A. *The Optimal Life History—U-Shaped Mortality*

Address first the subproblem of maximizing total expected energy surplus $W(K, t)$ conditional on an arbitrary starting date, t, and an arbitrary constant capital stock, $K > 0$. This subproblem generalizes that of the optimal steady state in Model 1 and the assumptions made here generalize those made there. Consider the following equations for s and J:

$$(5)\quad J\mu'(s) = -1, \frac{dJ}{dt} = -y + J\mu(s),$$

$$\text{for all } t \in [0, T], \text{ and } J(T) = 0,$$

where $y(t) = F(K, t) - s(t)$. It follows that:

LEMMA 4: *There is a unique solution for s of* (5), *and this is the essentially unique optimal path. It follows from* (4) *that* $W_K(K, t) = (p(t))^{-1}(\int_t^T p(\tau) F_K(K, \tau)\, d\tau) > 0$, *for all* $t \in [0, T)$ *and from* (3) *that* $\frac{dM(t)}{dt} = p(t)(W_K(K_0 + \bar{v}t, t) - 1)\bar{v}$, *for all* $t \in [0, T]$.

Assumption 5 and equation (4) imply that $\lim_{K \downarrow 0} W_K(K, t) = \infty$, for all $t \in [0, T)$, and that $W_K(K, T) = 0$, for all $K > 0$. Hence $\frac{dM(T)}{dt} < 0$; whereas, if $K(0)$ is small enough, $\frac{dM(0)}{dt} > 0$. Hence $M(t)$ has a maximum at some $t^* \in (0, T)$ where $\frac{dM(t^*)}{dt} = 0$ and $\frac{d^2M(t^*)}{dt^2} \leq 0$. This is sharpened slightly, in the usual way, as follows:

ASSUMPTION 6: *There is a unique* $t^* \in (0, T)$ *maximizing* $M(t)$ *over* $[0, T]$, *such that* $\frac{dM(t^*)}{dt} = 0$ *and* $\frac{d^2M(t^*)}{dt^2} = p(t^*)(W_{KK}(K_0 + \bar{v}t^*, t^*)\bar{v} + W_{Kt}(K_0 + \bar{v}t^*, t^*)) < 0$.

Model 2 is now shown to provide an economic explanation for the typical U-shape of mortality rates. This follows because the present assumptions imply that the value of life is hump-shaped, and the value of life and mortality are closely linked. To be precise: As long as output continues to rise after t^*, the associated optimal mortality profile must be U-shaped. In addition, this solution of the overall problem, as in (3), maximizes the growth rate at zero:

THEOREM 3: (a) *There is an essentially unique optimal path s yielding* $M(t^*) > 0$. *Given that optimal neural investment ends before output peaks, so that* $0 < t^* < \hat{t}(K^*) = \hat{t}$, *say, there is some* $\bar{t} \in (0, \hat{t})$ *such that the optimal* μ *is continuous, strictly decreasing for* $t \in [0, \bar{t})$, *and strictly increasing for* $t \in (\bar{t}, T)$. (b) *If* $C_0 = M(t^*)$, *this solution for s generates the maximum growth rate, as in Section* II, *subsection* A, *and this maximum growth rate is zero.*

A nice empirical test of the present approach is then provided by the predicted inverse relationship between $J(t)$ and $\mu(s)$, which stems from how mortality is determined here—from the relation $J\mu'(s) = -1$. Consider Figure 5, using data for both sexes on production, consumption, and mortality for the Ache, Hiwi, and Hadza.[23]

Although the present specification of the mortality function is mainly for simplicity, and Figure 5 suggests it is not exactly valid, it also suggests it is a good approximation.

B. *An Environment Favoring Learning; Lower Extrinsic Mortality*

Consider now an economic environment in which learning is more effective, as modeled by a shift in a parameter α in the production function. With an increase in α, output after brain growth is complete may at first decline, upon the advent of a cognitively demanding foraging activity. For example, a teenage male who hunts may accept lower current output than that from alternative productive activities. At older ages, however, output rises substantially.[24] It is

[23] Although the present model treats other nonneural somatic investment as a reduction in output rather than as a component of consumption, energy flow surplus is not affected by this transfer.

[24] It is enough that output may at first be lower and then must be higher, regardless of how this pattern relates to t^*. The present assumption is realistic, however, because there

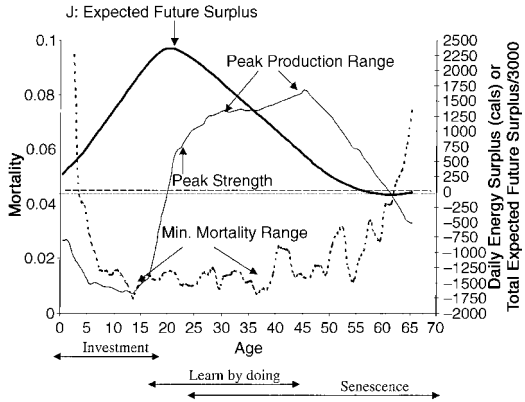

FIGURE 5. FORAGER MORTALITY AND ENERGY SURPLUS

indeed only the ultimate rise in the productivity of hunting resulting from learning-by-doing that makes such early specialization in hunting optimal. It is also then assumed that the overall effect of the increase in α is either to raise or hold constant total expected energy surplus.[25] As an alternative to such a shift in productivity, we will consider also the effect of a parametric reduction in extrinsic mortality.[26]

ASSUMPTION 7: (a) *The production function* $F(K, t, \alpha)$ *satisfies Assumption* 5, *for each* $\alpha \in \mathcal{A}$, *where* $\mathcal{A}$ *is a compact interval. It also satisfies* $F_{K\alpha}(K, t, \alpha) \geq 0$, *for all* $K \geq 0$, $t \in [0, T]$ *and* $\alpha \in \mathcal{A}$. *In addition, there exists* $\check{t} \in [t^*, T)$ *such that* (i) $F_\alpha(K^*, t, \alpha) = 0$, *for all* $t \in [0, t^*]$; (ii) $F_\alpha(K^*, t, \alpha) < 0$, *for all* $t \in (t^*, \check{t})$; (iii) $F_\alpha(K^*, t, \alpha) > 0$, *for* $t \in (\check{t}, T]$; *and* (iv) $\int_{t^*}^{T} p(t) F_\alpha(K^*, t, \alpha)\, dt \geq 0$. (b) *Mortality is* $\mu(\cdot) + \beta$, *where* $\mu(\cdot)$ *satisfies Assumption* 2, *and* $\beta \in \mathcal{B}$, *where* $\mathcal{B}$ *is a compact interval.*

An environment that favors learning in this sense induces simultaneous increases in life expectancy and brain size, despite the substantial lag that may now exist between the time that brain growth is complete and the peak favorable productivity shift. Lower extrinsic mortality has similar effects:

THEOREM 4: *The following comparative static results now hold.* (a) $\dfrac{\partial s(t, \alpha)}{\partial \alpha} \geq 0$, *for all* $t \in [0, t^*]$, $\dfrac{\partial s(t, \alpha)}{\partial \alpha} > 0$, *for all* $t \in (t^*, T)$, *and* $\dfrac{dK^*}{d\alpha} > 0$. *Thus, a shift to an environment favoring learning leads to greater life expectancy and to increased neural capital.* (b) $\dfrac{\partial s(t, \beta)}{\partial \beta} < 0$, *for all* $t \in [0, T)$, *and* $\dfrac{dK^*}{d\beta} < 0$, *so that lower extrinsic mortality also induces greater life expectancy and increased neural capital.*

The comparative static results for Model 1 and, especially, Model 2 imply that human intelligence and life expectancy might have coevolved as follows. A drier climate several million years ago resulted in the expansion of the African savanna at the expense of the rainforest. The resulting ecology sustained more grasses and hence more herbivores, as well as plants with edible roots and nut-bearing trees. The availability of these skill-intensive, high-energy food resources raised the productivity of the brain. This might have been true to a small extent in the immediate sense of Model 1, but seems especially plausible in the delayed sense of Model 2. In either case, this shift induced greater expenditure on mortality reduction, so that both life expectancy and intelligence rose. In addition, the skill, weapons, and social organization required for hunting seems bound to have simultaneously reduced extrinsic mortality in the form of predation on humans.[27] As in either model, this led to an amplifying decrease

is little output before brain growth is complete, and it slightly simplifies matters.

[25] The effect of an increase in α by itself is then to increase the maximal growth rate above zero or to hold it constant at zero. See Kaplan and Robson (2002).

Note also that such a shift might have a roughly neutral effect on overall body size, helping explain why this went up only slightly over the last two million years.

[26] Again, the effect of a decrease in β or an increase in α is offset here by a change in C_0 that restores zero growth.

[27] These same qualities might also have led to an increase in mortality due to warfare. However, the reduction in mortality due to predation might well have been important at low population densities, whereas the increase in mortality due to warfare might only have become important at higher densities, where it served to limit ultimate growth.

in intrinsic mortality, and the reduced overall mortality induced an increase in intelligence. These two effects—from increasing productivity and from reducing extrinsic mortality—therefore reinforced one another.

C. *"Rectangularization" of Mortality*

The specification of mortality here predicts a distinction between human and chimpanzee mortality patterns that stems from the *economics* of the two species. In either model, mortality is determined by the relation $J\mu'(s) = -1$, but we focus here on Model 2, in which J is more flexibly determined. Suppose humans and chimpanzees have the same mortality function, μ, and experience the same mortality rate, $\mu(s)$, perhaps at two different ages during childhood.[28] The values of J and s then also coincide. However, since $\frac{dJ}{dt} = -y + \mu(s)J > 0$, where $y(t) = F(K(t), t, \alpha) - s(t)$, it follows that mortality $\mu(s)$ will fall faster in human beings, given they have larger energy deficit, $-y$, in this comparison. In addition, since the maximum value of a human life is higher, minimum mortality will be lower. Figure 3 bears out these predictions.[29]

Model 2 similarly predicts that mortality should rise faster for humans than for chimpanzees if human energy surplus flow is the higher of the two when older individuals of the two species with the same mortality rate are compared. Although Figure 3 seems to suggest that mortality rises faster for humans in this sense, it should be noted that there are a limited number of individuals of such ages in the data set.

More generally, μ might also depend on overall body size and so be a time dependent function of s. Nevertheless, *one* factor tending to make human mortality rates fall rapidly in youth, attain a low minimum, and rise rapidly in old age may be the economic one described here.

[28] The underlying reason for the difference between the two species is then a shift in productivity, not a shift in exogenous mortality.

[29] The model also predicts that the initial infant mortality level for humans should be lower than for chimpanzees, if the overall value of a human life is larger, as is consistent with Figures 3 and 4, respectively.

IV. Discussion

Although the two models here investigate how natural selection modified genes influencing intelligence and life expectancy, they can be reinterpreted as more conventional economic decision-making. For example, consider how individuals simultaneously decide to reduce mortality rates via medical treatment, for example, and to invest in human capital. Model 1 might then imply, for example, that modernization of health care or greater productivity of education would result in both greater investment in human capital and greater expenditures on health.[30] Further, Model 2 provides an alternative explanation for "rectangularization" of mortality rates, as is sometimes claimed to be an ongoing trend in modern human demography. The usual explanation offered is that a reduction in mortality from infectious disease exposes a process of senescence that sets a biological maximum life span. (See, for example, Kenneth G. Manton et al., 1991.) However, modernization presumably also involves greater investment in youth and a greater flow of returns from mature adults. This increases the rate at which the value of life increases during youth and the maximum value that it reaches. It might also increase the rate at which it eventually decreases. Reinterpreting the second model as one of endogenous health care, mortality rates would then fall faster during youth, and reach lower values during adolescence and early adulthood, but possibly rise faster during old age, as required.

There are less immediate but perhaps more interesting implications of the present approach for the genetic shaping of preferences in hunter-gatherer societies. Hunter-gatherer age profiles of production could not have evolved without intergenerational resource flows. Without credit markets, such downward flows would not arise in a standard economic setting with selfishly rational borrowing and lending. An evolutionary model like those here implies that the flow of resources from old to young should be interpreted instead as maximizing a dynastic

[30] This is then an alternative approach to that of Isaac Ehrlich and Hiroyuki Chuma (1990), for example.

utility function that measures the growth rate of a genetic lineage.

These huge intergenerational flows mean standard biological approaches may not be appropriate either. One such standard biological approach involves "reproductive value," for example. This is the sum of the probabilities of being alive at each age multiplied by the expected number of offspring produced at that age, all discounted at the rate of population growth. Humans in their fifties and older then have negligible reproductive value. It is then unclear why they would even survive to such ages, if there were any possibility of raising fertility by even a small amount at younger ages, while decreasing survival later. Reproductive value cannot then readily account for the evolutionary formation of preferences across the entire life span.[31]

The present approach views children as investments within the parents' genetic lineage. During childhood and adolescence, children need parental subsidy. As parents approach middle age, the total burden on them increases, since there are more dependent children. A change in wealth should then be considered to affect the survival and reproduction of this increasing number of children, and it is not puzzling why humans survive into their fifties and beyond. This yields a new framework for examining how time preference and risk aversion vary with age, which might be especially illuminating for middle and old age. It has the potential to explain patterns of age variation of these economic attributes that may seem anomalous for an individual concerned only with herself.

In particular, such an evolutionary approach could provide a solid underpinning for the approach of Philip A. Trostel and Grant A. Taylor (2001). They propose that discounting arises because mental and physical capacities decline with age, because this deterioration reduces the marginal utility of consumption. Further, they propose that the rate of time discount might increase over the life cycle. An evolutionary approach would provide a more fundamental perspective on this. That is, if marginal utility declines with age, it could address *why* this would be evolutionarily optimal.

Finally, Models 1 and 2 sharpen the question of why aging occurs at all. Model 1 shows that, assuming that production and mortality are time-invariant functions, an optimal life history will consist of an investment period during which capital grows and endogenous mortality falls and then a steady state in which capital and mortality remain at constant optimal levels. Capital depreciation does not lead to a qualitative change in this conclusion. Model 2 shows that if aging is simply *assumed* to decrease productive output, mortality will ultimately increase with age. At the present, however, there is not a deep understanding of why capital should be allowed to age, whether this concerns machines or biological organisms. Deeper explanations of aging would be of considerable interest in both biology and economics.

Appendix

PROOF OF LEMMA 2:

If $K > 0$, $Z_s(K, s) = -\frac{1}{\mu(s)} - \frac{(F(K) - s)\mu'(s)}{\mu(s)^2}$, and $Z_s = 0$ implies $s \in (0, F(K))$. Moreover, if $Z_s(K, s) = 0$, then $Z_{ss}(K, s) = -\frac{(F(K) - s)\mu''(s)}{\mu(s)^2} < 0$. There is then a unique maximizer $s(K) \in (0, F(K))$, such that $Z_{ss}(K, s(K)) < 0$; $Z_s(K, s) > 0$ for all $s < s(K)$ and $Z_s(K, s) < 0$ for all $s > s(K)$. Since $Z_{sK} = -\frac{F'(K)}{\mu(s)^2}\mu'(s) > 0$, it follows that $\frac{ds(K)}{dK} = -\frac{Z_{sK}}{Z_{ss}} > 0$.

Remark: Since $W''(K) = \frac{F''\mu''(F - s) + F'(\mu')^2}{\mu\mu''(F - s)}$, the condition $W''(K^*) < 0$ is equivalent to

[31] Rogers (1994) adopts an approach based on reproductive value. However, he allows a given female, for example, to contemplate transfers to a sequence of descendants—daughter, granddaughter, and so on. This explicitly involves the female being of reproductive age. However, the sequence of weights obtained like this seem to be appropriate relative weights for a female beyond reproductive age contemplating a transfer away from her daughter in favor of her granddaughter, for example.

(A1) $$\Delta = F''\mu''(F - s) + F'(\mu')^2 < 0, \text{ at } K^*, s^*.$$

PROOF OF LEMMA 3:

Eliminating J from (2) yields the candidate path of mortality expenditure, s, as the continuous solution of:

(A2) $$\frac{ds}{dt} = f(s, t) = -\frac{(\mu'(s))^2}{\mu''(s)} \times \left(F(K_0 + \bar{v}t) - \bar{v} - s + \frac{\mu(s)}{\mu'(s)} \right), \quad \text{for all } t < t^*;$$

and $s(t) = s^*$, for all $t \geq t^*$. Define $\varphi(s, t) = F(K_0 + \bar{v}t) - s + \frac{\mu(s)}{\mu'(s)}$, for all $(s, t) \in S \equiv (0, \bar{s}) \times [0, t^*]$. It follows that $\frac{d\varphi(s(t), t)}{dt} = F'\bar{v} - \frac{\mu\mu''}{(\mu')^2}\frac{ds}{dt} = F'\bar{v} - \mu(\bar{v} - \varphi)$. In particular, if $0 < K < K^*$ and $\varphi = 0$, so that $s = s(K)$, then

(A3) $$\frac{d\varphi}{dt} = (F' - \mu)\bar{v} > 0.$$

Furthermore, $\frac{d^2\varphi(s(t), t)}{dt^2} = F''\bar{v}^2 - \mu'\frac{ds}{dt}(\bar{v} - \varphi) + \mu\frac{d\varphi}{dt}$. Hence at t^*, where $K = K^*$ and $s = s^*$,

(A4) $$\frac{d\varphi^-}{dt} = \varphi = 0,$$

$$\text{so } \frac{d^2\varphi^-}{dt^2} = \left(F'' - \frac{(\mu')^3}{\mu''} \right)\bar{v}^2 < 0, \text{ using (A1).}$$

There is a unique solution for s of (A2) in some neighborhood of (s^*, t^*). Further, (A3) and (A4) imply this extends to a unique solution for all $t \in [0, t^*]$, that stays in the region where $\varphi < 0$ and $s \in (0, s^*)$. Hence $\frac{ds}{dt} > 0$, for all $t \in [0, t^*]$. Finally, if $(s, t) \in S$, then $Z_s(K, s) = -\frac{\mu'}{\mu^2}\left(F(K) - s + \frac{\mu}{\mu'} \right) = -\frac{\mu'}{\mu^2}\varphi$. Hence $(s, t) \in S$ and $\varphi < 0$ imply $Z_s(K, s) < 0$ so that $s > s(K) > 0$.

PROOF OF THEOREM 1(a):

If the candidate path is given by v, s, K, and p, define $V(K_0) = \int_0^\infty p(F(K) - v - s)\,dt$, where $K(0) = K_0 > 0$ and $p(0) = 1$. Since $V(K_0) = W(K_0)$, for all $K_0 \geq K^*$

(A5) $$0 = -F(K_0) + s + \mu(s)V(K_0).$$

For any $0 < K_0 < K^*$ and any $\delta \in (0, t^*)$, $V(K_0) = \int_0^\delta p(F(K_0 + \bar{v}t) - \bar{v} - s)\,dt + \exp(-\int_0^\delta \mu(s)\,d\tau)V(K_0 + \bar{v}\delta)$, since the value for initial capital stock, K', and initial probability of survival, p, is $pV(K')$. It follows that

(A6) $$V'(K_0)\bar{v} = -F(K_0) + \bar{v} + s + \mu(s)V(K_0).$$

It is now shown that, for all $\hat{v} \in [0, \bar{v}]$ and all $\hat{s} \geq 0$,

(A7) $$F(K) - \hat{v} - \hat{s} + V'(K)\hat{v} - V(K)\mu(\hat{s}) \leq 0,$$

for all $K > 0$. This inequality (A7), together with (A5) and (A6), is Bellman's equation, and it follows because:

(i) The control s satisfies $\mu'(s)V(K) = -1$, so that $V(K) > 0$, for all $K > 0$.
(ii) Since $V(K) = W(K)$, for all $K > K^*$, and $W'(K) < 1$, if $K > K^*$, $v = 0$ maximizes the left-hand side (LHS) of (A7) there. Since $V'(K^*) = 1$, $v = 0$ is also maximal here. All that remains is to show that $v = \bar{v}$ is maximal for all $t < t^*$, which follows if $V'(K) > 1$, for all $0 < K < K^*$.

PROOF OF (i):

If $K \geq K^*$, then $\mu'(s)V(K) =$

$\mu'(s(K))\dfrac{F(K)-s(K)}{\mu(S(K))} = -1$. If $0 < K(t) < K^*$, define $\lambda(t) = V(K(t)) + \dfrac{1}{\mu'(s(t))}$, so that $\dfrac{d\lambda}{dt} = V'(K)\bar{v} - \dfrac{\mu''}{(\mu')^2}\dfrac{ds}{dt} = \mu\lambda$, for all $0 \le t < t^*$, using (A2) and (A6). However, $\lambda(t^*) = W(K^*) + \dfrac{1}{\mu'(s^*)} = 0$, so that $\lambda(t) \equiv 0$, for all $0 \le t < t^*$.

PROOF OF (ii):

If $0 < K < K^*$, it follows that $(V'(K) - 1)\bar{v} = -F(K) + s + \mu(s)V(K) = -\varphi(t)$. Lemma 3 shows $\varphi(t) < 0$, for all $0 \le t < t^*$, so that $V'(K) > 1$, for all $0 < K < K^*$.

Suppose now that $\tilde{K}$ and $\tilde{p}$ is any feasible absolutely continuous path such that $\dfrac{d\tilde{K}}{dt} = \tilde{v}$ and $\dfrac{d\tilde{p}}{dt} = -\tilde{p}\mu(\tilde{s})$, almost always, where $\tilde{v} \in [0, \bar{v}]$ and $\tilde{s} \in [0, \bar{s}]$ are measurable functions of time. Using (A7), $\int_0^T \tilde{p}(F(\tilde{K}) - \tilde{v} - \tilde{s})dt + \int_0^T \dfrac{d}{dt}(\tilde{p}V(\tilde{K}))\,dt \le 0$, so that, since $V(K) > 0$, for all $K > 0$, $V(K_0) > \int_0^T \tilde{p}(F(\tilde{K}) - \tilde{v} - \tilde{s})\,dt$, for all $T > 0$. Hence $V(K_0)$ is the optimal value function and the candidate path is optimal. This argument also shows that any path which fails to maximize the LHS of (A7) on a set of times of positive measure cannot be optimal.

PROOF OF THEOREM 1(b):

The above proof indeed shows $\max_{p,y} \int_0^\infty e^{-\rho t}p(t)y(t)\,dt \equiv V(K_0, \rho)$ exists, for all $\rho > -r$, since μ could then be redefined to include ρ. By the envelope theorem, $\dfrac{\partial V}{\partial \rho} = -\int_0^\infty te^{-\rho t}p(t)y(t)\,dt$, for the optimal p and y.[32] Since $\dfrac{d}{dt}\left[t\int_t^\infty e^{-\rho\tau}p(\tau)y(\tau)\,d\tau\right] = \int_t^\infty e^{-\rho\tau}p(\tau)y(\tau)\,d\tau - te^{-\rho t}p(t)y(t)$, integration by parts implies $\int_0^\infty te^{-\rho t}p(t)y(t)\,dt = \int_0^\infty \left(\int_t^\infty e^{-\rho\tau}p(\tau)y(\tau)\,d\tau\right)dt > 0$, because $\int_t^\infty e^{-\rho\tau}p(\tau)y(\tau)\,d\tau > 0$, for all $t \ge 0$, since $V(K_0, \rho) > 0$. Given $V(K_0, 0) = C_0$, then $V(K_0, \rho) < C_0$, for all $\rho > 0$. Thus a growth rate of zero is possible, but any strictly positive growth rate is not.

PROOF OF THEOREM 2(a):

Let $J(t, \alpha) = V(K_0 + \bar{v}t, \alpha)$, for all $t \in [0, t^*(\alpha))$ and $J(t, \alpha) = W(K^*(\alpha), \alpha)$, for all $t \ge t^*(\alpha)$. By the envelope theorem, $J_\alpha(t, \alpha) = (p(t))^{-1}\left(\int_t^{t^*} p(\tau)F_\alpha(K(\tau), \alpha)\,d\tau + p(t^*)W_\alpha(K^*, \alpha)\right) > 0$, for all $t \in [0, t^*)$. From $W_K(K^*, \alpha) = 1$, it follows that $\dfrac{dK^*}{d\alpha} = -\dfrac{W_{K\alpha}(K^*, \alpha)}{W_{KK}(K^*, \alpha)} > 0$ and $\dfrac{dt^*}{d\alpha} > 0$, since $W_{K\alpha}(K, \alpha) = \dfrac{F_{K\alpha}(K, \alpha)}{\mu(s(K))} - \dfrac{F_\alpha(K, \alpha)\mu'(s(K))}{\mu(s(K))^2}\dfrac{ds(K)}{dK} > 0$. Also $J_\alpha(t, \alpha) = W_K(K^*, \alpha)\dfrac{dK^*}{d\alpha} + W_\alpha(K^*, \alpha) > 0$, for all $t > t^*$. Since $\mu'(s(t, \alpha))J(t, \alpha) = -1$, it follows that $\dfrac{\partial s(t, \alpha)}{\partial \alpha} > 0$, for all $t \ge 0$.

PROOF OF THEOREM 2(b):

Let $J(t, \beta)$ be defined similarly. Using the envelope theorem, $J_\beta(t, \beta) = \int_t^\infty \dfrac{\partial}{\partial \beta}\left[\dfrac{p(\tau)}{p(t)}\right] \times (F(K(\tau), \tau) - s(\tau) - v(\tau))\,d\tau = -\int_t^\infty \dfrac{p(\tau)}{p(t)}(\tau - t)(F(K(\tau), \tau) - s(\tau) - v(\tau))d\tau$, for all $t \in [0, t^*)$. Integration by parts implies $J_\beta(t, \beta) = -(p(t))^{-1}\int_t^\infty p(\tau)J(\tau, \beta)\,d\tau < 0$, for all $t \in [0, t^*)$, since $J(\tau, \beta) > 0$, for all $\tau \ge 0$. From $W_K(K^*, \beta) = 1$, it follows that $\dfrac{dK^*}{d\beta} = -\dfrac{W_{K\beta}(K^*, \beta)}{W_{KK}(K^*, \beta)} < 0$, and $\dfrac{dt^*}{d\beta} < 0$, since $W_{K\beta}(K, \beta) = -\dfrac{F'(K) + ds(K)/dK}{(\mu(s(K)) + \beta)^2} < 0$. Also $J_\beta(t, \beta) = W_K(K^*, \beta)\dfrac{dK^*}{d\beta} + W_\beta(K^*, \beta) < 0$, for all $t > t^*$. From $\mu'(s(t, \beta))J(t, \beta) = -1$, it follows that $\dfrac{\partial s(t, \beta)}{\partial \beta} < 0$, for all $t \ge 0$.

[32] There are a number of similar appeals to the envelope theorem in this Appendix. Rigorous proofs can use results from Earl A. Coddington and Norman Levinson (1955, Ch. 1.7), concerning the dependence of the solution to a differential equation on various parameters.

PROOF OF LEMMA 4:

Requiring $\mu'(s)J = -1$, for all $J > 0$ and $s = 0$ for all $J \leq 0$ defines $s(J)$ as a continuous function. Now if $\frac{dJ}{dt} = -F(K, t) + s(J) + \mu(s(J))J = \Psi(J, t)$, then Ψ satisfies a Lipshitz condition in J and there is a unique solution for J. Since $J \leq 0$ implies that $\frac{dJ}{dt} \leq -F(K, t) < 0$, if $t \in (0, T)$, it follows that $J > 0$ for all $t \in [0, T)$. Hence $-J'(t) \geq -\mu(\hat{s})J(t) - \hat{s} + F(K, t)$, for all feasible $\hat{s}$ over $[0, T)$. If $\hat{p}(\tau, t) = \exp -\int_t^\tau \mu(\hat{s}(\tau'))\, d\tau'$, it follows that $\int_t^T \hat{p}(\tau, t)(F(K, \tau) - \hat{s}(\tau))\, d\tau \leq -\hat{p}(\tau, t)J(\tau)|_t^T = J(t)$. Hence s is optimal and $J(t)$ is the value function, $W(K, t)$. Also, any $\hat{s}$ that differs from s on a set of positive measure is strictly inferior. The value of $W_K(K, t)$ follows from the envelope theorem, as does that of $\frac{dM(t)}{dt}$.

PROOF OF THEOREM 3(a):

Define $s(J)$ as in the proof of Lemma 4. Now let $\frac{dJ}{d\tau} = \Psi(J, \tau) = -F(K_0 + \bar{v}\tau, \tau) + \bar{v} + s(J) + \mu(s(J))J$, for all $\tau \in [0, t]$, with terminal condition $J(t) = W(K_0 + \bar{v}t, t)$, for W as in equation (4). Since Ψ is Lipshitz, there is a unique solution for J, although, for nonoptimal t, this J may be nonpositive, so that $s(J)$ is zero. Nevertheless, $-J'(t) \geq -\mu(\hat{s})J(t) - \hat{s} - \bar{v} + F(K, t)$, for all feasible $\hat{s}$ over $[0, t)$, so that, as in the proof of Lemma 4, s is the essentially unique optimal path and J is the value function, $J(0) = M(t) > 0$. Note that $\frac{dJ(0)}{dt} = \mu(s(0))J + s(0) + \bar{v} > 0$, so that $\frac{d\mu(s(t))}{dt} < 0$, for all t close enough to 0. Further, equation (5) implies that, if $t \geq \hat{t} > t^*$, then $\frac{dJ}{dt} < 0$, so that $\frac{d\mu}{dt} > 0$. Indeed, note that, if $\frac{dJ}{dt} \geq 0$, at any $t > \hat{t}$, then $\frac{d^2J}{dt^2} = -F_t(K^*, t) + \mu\frac{dJ}{dt} > 0$, so that $\frac{dJ}{dt} > 0$, for all sufficiently large t, contradicting $J(T) = 0$. If, on the other hand, $\frac{dJ}{dt} = 0$ at $\hat{t}$, then $\frac{d^2J}{dt^2} = 0$, but $\frac{d^3J}{dt^3} > 0$, since $F_{tt}(K^*, \hat{t}) < 0$, so this contradiction still follows. At $t^* < \hat{t}$, the drop in y implies that $\frac{dJ^+}{dt} < \frac{dJ^-}{dt}$. Further, if $\frac{dJ}{dt} = 0$ at some $\bar{t} < \hat{t}$, $\bar{t} \neq t^*$, then $\frac{d^2J}{dt^2} = -F_t(K^*, \bar{t}) < 0$ at $\bar{t}$. It follows that J is strictly increasing for $t \in [0, \bar{t})$ and strictly decreasing for $t \in (\bar{t}, T]$, for some $\bar{t} < \hat{t}$, where $\bar{t}$ may or may not be t^*.

PROOF OF THEOREM 3(b):

This is analogous to the proof of Theorem 1(b).

PROOF OF THEOREM 4(a):

Note that $W_\alpha(K^*, t, \alpha) = (p(t))^{-1} \times \int_t^T p(\tau)F_\alpha(K^*, \tau, \alpha)\, d\tau$, for all $t \in [t^*, T]$ and $W_{\alpha t}(K^*, t, \alpha) = \mu(s(t))W_\alpha(K^*, t, \alpha) - F_\alpha(K^*, t, \alpha)$. It is immediate that $W_\alpha(K^*, t, \alpha) > 0$, for all $t \in [\check{t}, T)$. Further, if $W_\alpha(K^*, t, \alpha) = 0$, for any $t' \in (t^*, \check{t})$, then $W_{\alpha t}(K^*, t', \alpha) > 0$, so that $W_\alpha(K^*, t, \alpha) < 0$ for all $t \in [t^*, t')$. Since, by assumption, $W_\alpha(K^*, t^*, \alpha) \geq 0$, it follows that $W_\alpha(K^*, t, \alpha) > 0$, for all $t \in (t^*, T)$. Since $\mu'(s(t, \alpha))W(K^*, t, \alpha) = -1$, it also follows that $\left.\frac{\partial s(t, \alpha)}{\partial \alpha}\right|_{K = K^*} > 0$, for all $t \in (t^*, T)$. Given $W_K(K^*, t^*, \alpha)$ as in Lemma 4, $W_{K\alpha}(K^*, t^*, \alpha) = \int_{t^*}^T \frac{d}{d\alpha}\left[\frac{p(\tau)}{p(t^*)}\right]\Big|_{K = K^*} F_K(K^*, \tau, \alpha)\, d\tau + (p(t^*))^{-1} \times \int_{t^*}^T p(\tau)F_{K\alpha}(K^*, \tau, \alpha)\, d\tau > 0$. From $W_K(K^*, t^*, \alpha) = 1$ and Assumption 6, $\frac{dt^*}{d\alpha} = -\frac{W_{K\alpha}(K^*, t^*, \alpha)}{W_{KK}(K^*, t^*, \alpha)\bar{v} + W_{Kt}(K^*, t^*, \alpha)} > 0$, and hence $\frac{dK^*}{d\alpha} > 0$. Hence $\frac{\partial s(t, \alpha)}{\partial \alpha} = \left.\frac{\partial s(t, \alpha)}{\partial \alpha}\right|_{K = K^*} - \frac{\mu'(s(t, \alpha))W_K(K^*, t, \alpha)}{\mu''(s(t, \alpha))W(K^*, t, \alpha)}\frac{dK^*}{d\alpha} > 0$, for all $t \in (t^*, T]$. Finally, since $J_\alpha(t, \alpha) \geq 0$, for

J as in the proof of Theorem 3(a), $\frac{\partial s(t, \alpha)}{\partial \alpha} \geq 0$, for all $t \in [0, t^*)$.

PROOF OF THEOREM 4(b):

Using the envelope theorem, $W_\beta(K^*, t, \beta) = \int_t^T \frac{\partial}{\partial \beta}\left[\frac{p(\tau)}{p(t)}\right](F(K^*, \tau) - s(\tau))\, d\tau = -\int_t^T \frac{p(\tau)}{p(t)}(\tau - t)(F(K^*, \tau) - s(\tau))\, d\tau$, for all $t \in [t^*, T]$. Applying integration by parts, it follows that $W_\beta(K^*, t, \beta) = -(p(t))^{-1} \times \int_t^T p(\tau)W(K, \tau, \beta)\, d\tau < 0$, for all $t \in [t^*, T)$, since $W(K, \tau, \beta) > 0$, for all $\tau \in [0, T)$. From $\mu'(s(t, \beta))W(K^*, t, \beta) = -1$, it follows that $\left.\frac{\partial s(t, \beta)}{\partial \beta}\right|_{K = K^*} < 0$, for all $t \in [t^*, T)$. Given $W_K(K^*, t^*, \beta)$ as in Lemma 4, $W_{K\beta}(K^*, t^*, \beta) = \int_{t^*}^T \frac{d}{d\beta}\left[\frac{p(\tau)}{p(t^*)}\right]\Big|_{K = K^*} F_K(K^*, \tau)\, d\tau < 0$. The results then follow as in (a).

REFERENCES

Allman, John; McLaughlin, Todd and Hakeem, Atiya. "Brain Weight and Life Span in Primate Species." *Proceedings of the National Academy of Sciences of the USA*, January 1993, *90*(1), pp. 118–22.

Barton, Robert A. "The Evolutionary Ecology of the Primate Brain," in Phyllis C. Lee, ed., *Comparative primate socioecology*. Cambridge: Cambridge University Press, 1999, pp. 167–203.

Becker, Gary S. *A treatise on the family*, 2nd Ed. Cambridge, MA: Harvard University Press, 1991.

Cichon, Mariusz. "Evolution of Longevity through Optimal Resource Allocation." *Proceedings of the Royal Society of London, Series B*, September 1997, *264*(1386), pp. 1383–88.

Coddington, Earl A. and Levinson, Norman. *Theory of ordinary differential equations*. New York: McGraw-Hill, 1955.

Ehrlich, Isaac and Chuma, Hiroyuki. "A Model of the Demand for Longevity and the Value of Life Extension." *Journal of Political Economy*, August 1990, *98*(4), pp. 761–82.

Galor, Oded and Moav, Omer. "Natural Selection and the Origin of Economic Growth." *Quarterly Journal of Economics*, November 2002, *117*(4), pp. 1133–92.

Galor, Oded and Weil, David N. "Population, Technology, and Growth: From Malthusian Stagnation to the Demographic Transition and Beyond." *American Economic Review*, September 2000, *90*(4), pp. 806–28.

Hill, Kim and Hurtado, A. Magdalena. *Ache life history*. New York: Aldine de Gruyter, 1996.

Holliday, Malcolm A. "Body Composition and Energy Needs During Growth," in Frank Falker and James M. Tanner, eds., *Human growth*. New York: Plenum, 1978, pp. 117–39.

Jerison, Harry J. *Evolution of the brain and intelligence*. New York: Academic Press, 1973.

Jurmain, Robert; Nelson, Harry; Kilgore, Lynn and Trevathan, Wenda. *Introduction to physical anthropology*. Belmont, CA: Wadsworth, 2000.

Kaplan, Hillard S. and Hill, Kim. "Food-Sharing Among Ache Foragers: Tests of Explanatory Hypotheses." *Current Anthropology*, April 1985, *26*(2), pp. 223–45.

Kaplan, Hillard S.; Hill, Kim; Hurtado, A. Magdalena and Lancaster, Jane B. "The Embodied Capital Theory of Human Evolution," in P. T. Ellison, ed., *Reproductive ecology and human evolution*. Hawthorne, NY: Aldine de Gruyter, 2001, pp. 293–318.

Kaplan, Hillard S.; Hill, Kim; Lancaster, Jane B. and Hurtado, A. Magdalena. "A Theory of Human Life History Evolution: Diet, Intelligence, and Longevity." *Evolutionary Anthropology*, August 2000, *9*(4), pp. 156–85.

Kaplan, Hillard S. and Robson, Arthur J. "The Emergence of Humans: The Coevolution of Intelligence and Longevity with Intergenerational Transfers." *Proceedings of the National Academy of Sciences of the USA*, July 2002, *99*(15), pp. 10221–26.

Manton, Kenneth G.; Stallard, Eric and Tolley, H. Dennis. "Limits to Human Life Expectancy: Evidence, Prospects, and Implications." *Population and Development Review*, December 1991, *17*(4), pp. 603–37.

Martin, Robert D. "Relative Brain Size and Basal Metabolic Rate in Terrestrial Vertebrates." *Nature*, September 1981, *293*(5827), pp. 57–60.

Ofek, Haim. *Second nature: Economic origins of human evolution.* Cambridge: Cambridge University Press, 2001.

Parker, Sue T. and McKinney, Michael L. *Origins of intelligence: The evolution of cognitive development in monkeys, apes, and humans*. Baltimore, MD: Johns Hopkins Press, 1999.

Reader, Simon M. and Laland, Kevin N. "Social Intelligence, Innovation, and Enhanced Brain Size in Primates." *Proceedings of the National Academy of Sciences of the USA*, April 2002, *99*(7), pp. 4436–41.

Robson, Arthur J. "The Biological Basis of Economic Behavior." *Journal of Economic Literature*, March 2001, *39*(1), pp. 11–33.

Rogers, Alan. "Evolution of Time Preference by Natural Selection." *American Economic Review*, June 1994, *84*(3), pp. 460–81.

Rowe, Noel. *Pictorial guide to the living primates*. New York: Pogonius Press, 1996.

Sibley, Richard; Calow, Peter and Nichols, Nancy K. "Are Patterns of Growth Adaptive?" *Journal of Theoretical Biology*, February 1985, *112*(3), pp. 553–74.

Smith, B. Holly. "Dental Development and the Evolution of Life History in Hominidae." *American Journal of Physical Anthropology*, October 1991, *86*(2), pp. 157–74.

Stearns, Stephen C. *The evolution of life histories*. Oxford: Oxford University Press, 1992.

Trostel, Philip A. and Taylor, Grant A. "A Theory of Time Preference." *Economic Inquiry*, July 2001, *39*(3), pp. 379–95.