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Embodied Capital and the Evolutionary Economics of the Human Life Span

Hillard Kaplan
Jane Lancaster
Arthur Robson

A fundamental question concerning aging is whether the life spans of organisms evolve and, if so, what forces govern their evolution. In this chapter we argue that life spans do evolve and present a general theory of life spans, with a particular focus on humans. We employ a qualitative definition of life span: the amount of time between birth and the age at which the likelihood of death becomes high, relative to the likelihoods at younger ages.[1] Most multicellular organisms exhibit a phase in which mortality decreases with age and then a second phase in which mortality increases with age.[2] Our definition focuses on this second phase, on the age at which death becomes imminent because of physiological deterioration or some environmental condition (such as winter). We chose this approach over the "maximum life span" concept, because it is more biologically meaningful. It focuses on the length of time that organismic function is adequate to sustain life. Since it is concerned with likelihoods rather than actual events, it assumes that many individuals do not live their full life span. For many organisms, including humans, this qualitative definition corresponds to a more precise quantitative definition: the modal age at death, conditional on reaching adulthood. The principal argument we develop is that life spans evolve as part of an integrated life-history program and that the program for development and reproduction is fundamentally related to the age of death.

Our first section outlines an evolutionary economic framework for understanding the effects of natural selection on life histories, previously referred to as "embodied capital theory." It combines the basic structure of life-history theory as developed in biology with the formal analytical approach developed in the analysis of capital in economics. We next dis-

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cuss specialization and flexibility in life histories, with special emphases on the fast–slow continuum and on the relationship between brain evolution and life-history evolution. This is followed by a graphical presentation of analytical models of life-history evolution, based on embodied capital theory.

Our second section focuses on the special features of the human life course. This section briefly reviews a theory of human life-history evolution, developed and partially tested in earlier work. The theory posits that large brains and slow life histories result from a dietary specialization that has characterized the last 2 million years of human evolution. Empirical findings suggest that humans have a particular life course with characteristic schedules of growth, development, fertility, mortality, and aging. The approach here does not assume that those schedules are fixed and unresponsive to environmental variation. Rather it implies structured flexibility based upon the variation experienced in human evolutionary history and a set of specialized anatomical, physiological, and psychological adaptations to the niche humans occupied during that history. Together, those adaptations result in a life span for the species that can vary within a limited range.

We conclude with a discussion of two themes: short- and long-term flexibility in the human life span and the building blocks for a more adequate theory of senescence and life span.

Embodied capital and life-history theory

Fundamental tradeoffs in life-history theory

Life-history theory in biology grew out of the recognition that all organisms face two fundamental reproductive tradeoffs (see Charnov 1993; Lessells 1991; Roff 1992; Sibly 1991; Stearns 1992, for general reviews and Hill and Kaplan 1999, for a review of the application of life-history theory to humans). The first tradeoff is between current and future reproduction. The second is between quantity and quality of offspring. With respect to the first (the principal focus here), early reproduction is favored by natural selection, holding all else constant. This is the result of two factors. First, earlier reproduction tends to increase the length of the reproductive period. Second, shortening generation length by early reproduction usually increases the growth rate of the lineage.

The forces favoring early reproduction are balanced by benefits derived from investments in future reproduction. Those investments, often referred to as "somatic effort," include growth and maintenance. The allocation of energy to growth has three potential benefits. It can increase a) the length of the life span, by lowering size-dependent mortality, b) the efficiency of energy capture, thus allowing for a higher rate of offspring

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production, and c) the rate of success in intrasexual competition for mates. For this reason, organisms typically have a juvenile phase in which fertility is zero until they reach a size at which some allocation to reproduction increases fitness more than it increases growth. Similarly, for organisms that engage in repeated bouts of reproduction (humans included), some energy during the reproductive phase should be diverted from reproduction and allocated to maintenance so that organisms can live to reproduce again. Natural selection is expected to optimize the allocation of energy to current reproduction and to future reproduction (via investments in growth and maintenance) at each point in the life course so that genetic descendents are maximized (Gadgil and Bossert 1970; Sibly et al. 1985; see Hill and Hurtado 1996 for an application of those models to humans).

Specialization and flexibility in life histories and the fast–slow continuum

Variation across taxa and across conditions in optimal energy allocations and optimal life histories is shaped by ecological factors, such as food supply, disease, and predation rates. It is generally recognized that there are species-level specializations that result in bundles of life-history characteristics, which, in turn, can be arrayed on a fast–slow continuum (Promislow and Harvey 1990). For example, among mammals, species on the fast end exhibit short gestation times, early reproduction, small body size, large litters, and high mortality rates, with species on the slow end having opposite characteristics (ibid.). Similarly, among plants, some species that specialize in secondary growth are successful at rapidly colonizing newly available habitats, but their rapid life history means that they invest little in chemical defense and structural cells that would promote longevity. On the other end of the continuum are trees, such as the bristle cone pine, that are slow to mature but suffer very low mortality rates and are very long-lived (Finch 1998).

It is also recognized that many, if not most, organisms are capable of slowing or accelerating their life histories, depending upon environmental conditions such as temperature, rainfall, food availability, density of conspecifics, and mortality hazards. Within-species variation in life-history characteristics can operate over several time scales. For example, there is abundant evidence that allocations to reproduction, as measured by fecundity and fertility, vary over the short term among plants, birds, and humans in response to the balance between food supply and energy output (see, for example, Hurtado and Hill 1990; Lack 1968). The impacts of the environment may extend over longer time intervals through developmental effects. For example, calorie restriction in young rats tends to slow growth rates and leads to reduced adult stature, even when food becomes abundant in

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the later juvenile period (Shanley and Kirkwood 2000). Some intraspecific variation arises at even longer time scales, where this involves differential selection on genetic variants in different habitats. For example, rates of senescence vary across populations of grasshoppers, with those at higher altitudes and experiencing earlier winters senescing faster than those at lower altitudes (Tatar, Grey, and Carey 1997).

A central thesis of this chapter is that both specialization and flexibility are fundamental to understanding the human life span. On the one hand, the large human brain supports the ability to respond flexibly to environmental variation and to learn culturally, facilitating short-term flexibility. On the other hand, the commitment to a large brain and the long period of development necessary to make it fully functional constrains the human life course by requiring specializations for a slow life history.

Embodied capital and life-history theory

The embodied capital theory integrates life-history theory with capital investment theory in economics (Becker 1975; Mincer 1974) by treating the processes of growth, development, and maintenance as investments in stocks of somatic or embodied capital. In a physical sense, embodied capital is organized somatic tissue: muscles, digestive organs, brains, and so on. In a functional sense, embodied capital includes strength, speed, immune function, skill, knowledge, and other abilities. Since such stocks tend to depreciate with time, allocations to maintenance can also be seen as investments in embodied capital. Thus, the present–future reproductive tradeoff becomes a tradeoff between investments in own embodied capital and reproduction, and the quantity–quality tradeoff becomes a tradeoff between the embodied capital of offspring and their number.

The embodied capital theory allows us to treat problems that have not been addressed with standard life-history models. For example, physical growth is only one form of investment. The brain is another form of embodied capital, with special qualities. On the one hand, neural tissue monitors the organism's internal and external environment and induces physiological and behavioral responses to stimuli (Jerison 1973, 1976). On the other hand, the brain has the capacity to transform present experiences into future performance. This is particularly true of the cerebral cortex, which specializes in the storage, retrieval, and processing of experiences. The expansion of the cerebral cortex among higher primates represents an increased investment in this capacity (Armstrong and Falk 1982; Fleagle 1999; Parker and McKinney 1999). Among humans, the brain supports learning and knowledge acquisition during both the juvenile and adult periods, well after the brain has reached its adult mass. This growth in the stock of knowledge and functional abilities is another form of investment.

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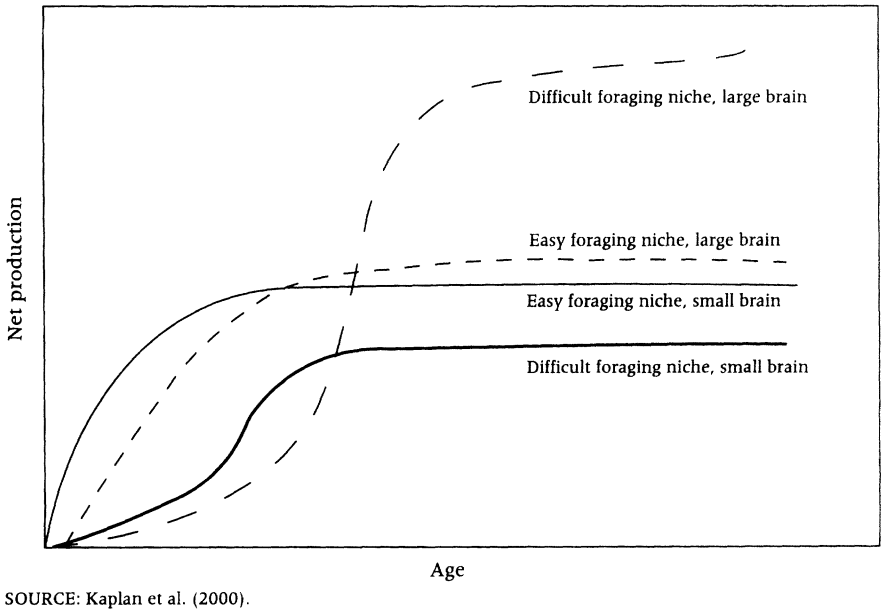
The action of natural selection on the neural tissue involved in learning, memory, and the processing of stored information depends on the costs and benefits realized over the organism's lifetime. There are substantial energetic costs of growing the brain early in life and of maintaining neural tissue throughout life. Among humans, for example, it has been estimated that about 65 percent of all resting energetic expenditure is used to support the maintenance and growth of the brain in the first year of life (Holliday 1978). Another potential cost of the brain may be decreased performance early in life. The ability to learn may entail reductions in "preprogrammed" behavioral routines, thereby decreasing early performance. The incompetence of human infants, even children, in many motor tasks is an example.

Taking these costs into account, the net benefits from the brain tissue involved in learning are only fully realized as the organism ages (see Figure 1). In a niche where there is little to learn, a large brain might have higher costs early in life and a relatively small influence on productivity late in life. Natural selection may then tend to favor the small brain. In a more challenging niche, however, although a small brain might be slightly better early in life, because of its lower cost, it would be much worse later, and the large brain might be favored instead.

The brain is not the only system that learns and becomes more functional through time. Another example is the immune system, which re-

FIGURE 1 Age-specific effects of brains on net production: Easy and difficult foraging niches



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quires exposure to antigens in order to become fully functional. Presumably, indeed, the maturation of the immune system is a primary factor in the decrease in mortality with age from birth until the end of the juvenile period.

Furthermore, a positive relationship between brain size and life span (controlling for body size) is found in empirical studies of mammals (Sacher 1959) and primates (Allman, McLaughlin, and Hakeem 1993; Hakeem et al. 1996; Judge and Carey 2000; Kaplan and Robson 2002). Such considerations led us to propose that brain size and longevity coevolve for the following reasons. Since the returns to a large brain lie in the future, ecological conditions favoring large brains also favor greater expenditure on survival. Conversely, exogenous ecological conditions that lower mortality favor increased expenditure on survival and hence also much greater investment in brain capital (Kaplan et al. 2000; Kaplan and Robson 2002; see Carey and Judge 2001 for an alternative coevolutionary model of human life spans).

This logic suggested an alternative approach to standard treatments of life-history theory. Standard treatments generally define two types of mortality: 1) extrinsic, which is imposed by the environment and is outside the control of the organisms (e.g., predation or weather), and 2) intrinsic, over which the organism can exert some control in the short run or which is subject to selective control over longer periods. In most models of growth and development, mortality is treated as extrinsic and therefore not subject to selection (Charnov 1993; Kozlowski and Wiegert 1986; for exceptions see Janson and Van Schaik 1993; Charnov 2001). Models of aging and senescence (Promislow 1991; Shanley and Kirkwood 2000) frequently treat aging as affecting intrinsic mortality, with extrinsic mortality, in turn, selecting for rates of aging. For example, in the Gompertz–Makeham mortality function where the mortality rate, μ, equals $A + Be^{\mu x}$ (with A, B, and μ being parameters and x referring to age), this entails treating the first term on the right-hand side of the equation, A, as the extrinsic component and the second term as the intrinsic component.

In our view, this distinction between types of mortality is unproductive and generates confusion. Organisms can exert control over virtually all causes of mortality in the short or long run. Susceptibility to predation can be affected by vigilance, choice of foraging zones, travel patterns, and anatomical adaptations, such as shells, cryptic coloration, and muscles that facilitate flight. Each of these behavioral and anatomical adaptations has energetic costs that reduce energy available for growth and reproduction. Similar observations can be made regarding endogenous responses to disease and temperature. The extrinsic mortality concept has been convenient, because it provided a reason for other life-history traits, such as age of first reproduction and rates of aging. However, this has prevented the examination of how mortality rates themselves evolve by natural selection.

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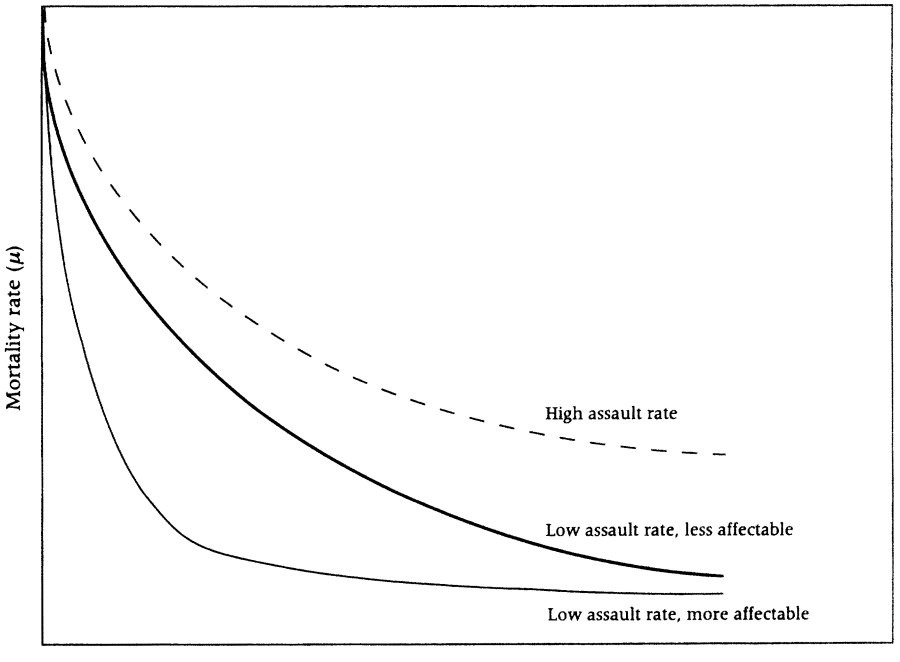
Since all mortality is, to some extent, intrinsic or endogenous, a more useful approach is to examine the functional relationship between mortality and effort allocated to reducing it (see Figure 2). Exogenous variation can be thought of in terms of varying "assault types" and varying "assault rates" of mortality hazards. For example, warm, humid climates favor the evolution of disease organisms and therefore increase the assault rate and diversity of diseases affecting organisms living in those climates. Exogenous variation also may affect the functional relationship between mortality hazards and endogenous effort allocated to reducing them.

The recognition that all mortality is partially endogenous and therefore subject to selection complicates life-history theory because it requires multivariate models, but it also generates insights about evolutionary coadaptation or coevolution among life-history traits. One of the benefits of modeling life-history evolution formally in terms of capital investments is that the analysis of such investments is well developed in economics with many well-established results. The next section summarizes some formal results of applying capital investment theory to life-history evolution.

FIGURE 2 Mortality as a function of investments



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Capital investments and endogenous mortality

As a first step, it is useful to think of capital as the bundle of functional abilities of the soma. Organisms generally receive some energy from their parents, represented as an initial stock of capital, say K_0. Net energy acquired from the environment, F, at each point in time, t, is a positive function of the capital stock, with diminishing returns to capital. This energy can be used in three ways, which are endogenous and subject to selection. It can be reinvested in increasing the capital stock, that is, in growth. Define $v(t)$ as flow of investment at time t, so that dK/dt equals $v(t)$. Since growth and development take time, it is useful to impose a maximal investment rate, $\bar{v}$. Some energy, s, may also be allocated to reducing mortality, μ, for example via increased immune function, as illustrated in Figure 2. The probability of reaching any age, $p(t)$, is then a function of mortality rates at each earlier age, so that

$$p(t) = e^{-\int_0^t \mu(t)dt}.$$

Finally, energy can be used for reproduction, which is the net excess energy available after allocations to capital investments and mortality reduction, y; so $y(t) = F(K) - v(t) - s(t)$.

The dynamic optimization program is to find the largest solution r of

$$\int_0^\infty p(t)y(t)e^{-rt}dt = C_0,$$

where C_0 is the cost of producing a newborn. This equation is an economic extension of the continuous-time Euler–Lotka equation for the long-run growth rate in a species without parental investment after birth. Under most conditions (for example, for most of human evolutionary history), the average r must be close to zero. It can then be shown that an optimal life history would choose capital investment and mortality reduction so as to maximize total expected surplus energy over the life course. The results of the analysis have been presented and proven formally (Robson and Kaplan 2002). At each point in time, the marginal gain from investments in capital and the marginal gain from increased expenditure on survival must equal their marginal costs. During the capital investment period, where v is greater than zero, the value of life, J, which is equal to total expected future net energy, is increasing with age, since productivity is growing with increased capital. The optimal value of s also then increases. At some age, a steady state is reached where capital is at its optimum level and both capital and mortality rates remain constant.

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Two important comparative results emerge from this analysis. An environmental change that increases the productivity of capital has two reinforcing effects: it increases the optimal level of capital investment (and hence the length of the investment period) and it decreases mortality through increases in s. A reduction in mortality rates has two similar effects: it increases the optimal capital stock and produces a reinforcing increase in s.

We note that the model does not result in senescence, as defined by increasing mortality rates with age. Even if capital were to depreciate over time (say, if $dK/dt = (1 - \lambda)K(t) + v(t)$, with λ being the proportional depreciation rate), a steady state still would be achieved where depreciation would be exactly offset by investment (Arrow and Kurz 1970: 85; Intriligator 1971). We address this issue in the final section.

Embodied capital and the evolution of human life histories

There has been a series of radiations within the primate order toward increased brain size, relative to body size, and toward increased longevity. These involve a transition from primitive prosimian primates to monkeys, then from monkeys to apes, and finally from apes to humans. For example, a human has a brain that is roughly three times as big as that of a chimpanzee and lives about twice as long. Can the theory illustrated above explain those radiations resulting in the long lives and large brains characteristic of the genus *Homo* and, particularly, of modern *Homo sapiens*? We posit that this extreme brain size and extreme longevity are coevolved responses to learning-intensive foraging strategies and a dietary shift toward high-quality, nutrient-dense, and difficult-to-acquire food resources. The following logic underlies our proposal. First, high levels of knowledge, skill coordination, and strength are required to exploit the suite of resources humans consume. The attainment of those abilities requires time and a significant commitment to development. This extended learning phase during which productivity is low is compensated for by higher productivity during the adult period, with an intergenerational flow of food from old to young. Since productivity increases with age, the time investment in skill acquisition and knowledge leads to selection for lowered mortality rates and greater longevity, because the returns on the investments in development occur at older ages.

Second, we believe that the feeding niche specializing in large, valuable food packages, particularly hunting, promotes cooperation between men and women and high levels of male parental investment, because it favors sexual specialization in somatic investments and thus generates a complementarity between male and female inputs. The economic and reproductive cooperation between men and women facilitates provisioning of juveniles, which both bankrolls their somatic investments and allows

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lower mortality during the juvenile and early adult periods. Cooperation between males and females also allows women to allocate more time to childcare, increasing both survival and reproductive rates. Finally, large packages also appear to promote interfamilial food sharing. Food sharing reduces the risk of food shortfalls due to the vagaries of foraging, providing insurance against illness and against variance in family size resulting from stochastic mortality and fertility. These buffers favor a longer juvenile period and higher investment in other mechanisms to increase life span.

Thus, we propose that the long human life span coevolved with the lengthening of the juvenile period, with increased brain capacities for information processing and storage, and with intergenerational resource flows—all as a result of a significant dietary shift. Humans are specialists in that they consume only the highest-quality plant and animal resources in their local environment and rely on creative, skill-intensive techniques to exploit them. Yet, the capacity to develop new techniques for extractive foraging and hunting allows them to exploit a wide variety of foods and to colonize all of the Earth's terrestrial and coastal ecosystems. In the following sections we review the specialized adaptations associated with this life history.

Digestion and diet

There is mounting evidence from various sources, including digestive anatomy, digestive biochemistry, bone isotope ratios, archeology, and observations of hunter-gatherers, that humans are specialized toward the consumption of calorie-dense, low-fiber foods that are rich in protein and fat. Contrary to early generalizations based on incomplete analysis and limited evidence (Lee 1979; Lee and DeVore 1968), more than half of the calories in hunter-gatherer diets are derived, on average, from meat. There are ten foraging societies and five chimpanzee communities for which caloric production or time spent feeding has been monitored systematically (Kaplan et al. 2000). All modern foragers differ considerably in diet from chimpanzees. Measured in calories, the major component of forager diets is vertebrate meat. Meat accounts for between 30 percent and 80 percent of the diet in the sampled societies, with most diets being more than 50 percent vertebrate meat, whereas chimpanzees obtain about 2 percent of their food energy from hunted foods. Similarly, using all 229 hunter-gatherer societies described in the Ethnographic Atlas (Murdock 1967) and Murdock's estimates based upon qualitative ethnographies, Cordain et al. (2000) found median dependence on animal foods in the range of 66 to 75 percent.

The next most important food category in the ten-society sample is extracted resources, such as most invertebrate animal products, roots, nuts, seeds and difficult-to-extract plant parts such as palm fiber or growing shoots. These are mostly nonmobile resources embedded in a protective context such as underground, in hard shells, or bearing toxins that must be removed

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before they can be consumed. In the ten-forager sample, extracted foods accounted for about 32 percent of the diet, as opposed to 3 percent among chimpanzees.

In contrast to hunted and extracted resources, which are difficult to acquire, collected resources form the bulk of the chimpanzee diet. Collected resources, such as fruits, leaves, flowers, and other easily accessible plant parts, are simply gathered and consumed. They account for 95 percent of the chimpanzee diet, on average, but only 8 percent of the human forager diet. The data suggest that humans specialize in rare but nutrient-dense resource packages or patches (meat, roots, nuts) whereas chimpanzees specialize in ripe fruit and plant parts with low nutrient density.

Comparative data on digestive anatomy confirm that these contemporary differences reflect long-term adaptations. Gorillas, chimpanzees, and humans can be arrayed along a continuum in terms of their digestive anatomy (Schoeninger et al. 2001). The gorilla has a very long large intestine and caecum in order to use bacterial fermentation for the breakdown of plant cellulose in leaves and other structural plant parts as a source of dietary protein. Although gorillas eat significant quantities of fruit, they derive a large proportion of their calories and most of their protein from leaves and other nonreproductive plant parts. Chimpanzee caeca are somewhat smaller. Chimpanzees supplement leaf consumption with hunted foods, insects, and nuts for fat and protein.

Human digestive anatomy is specialized for a very different diet. Humans have very small large intestines and are incapable of digesting cellulose in large quantities as a source of protein, and they have very long small intestines for the digestion of lipids (ibid.). Moreover, humans are very inefficient at chain elongating and desaturating various carbon fatty acids to produce the fatty acids that are essential cellular lipids (Emken et al. 1992; cited in Cordain et al. 2002, upon which this discussion is based). Since humans share this trait with other obligate carnivores and since those essential fatty acids are found only in animal foods, it appears that human digestion is specialized toward meat consumption and low-fiber diets. If chimpanzees consumed as much meat as humans, the nitrogen would destroy their foregut bacteria; and if they consumed a diet as low in fiber, they would suffer from colonic twisting (Schoeninger et al. 2001). Humans, on the other hand, must reduce dietary fiber. When they acquire foods that are high in fiber, such as roots and palm fiber, they remove the fiber before ingestion (ibid.).

Although the data are still scarce, it appears that this dietary shift occurred at the origin of the genus *Homo* about 2 million years ago. Compared to chimpanzees and australopithecines, early *Homo* appears to have had a reduced gut (Aiello and Wheeler 1995); and radio-isotope data from fossils also suggest a transition from a plant-based diet to greater reliance on meat (see Schoeninger et al. 2001 for a review). There is significant archeological

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evidence of meat eating by *Homo* in the early Pleistocene (Bunn 2001). Finally, radio-isotope evidence from Neanderthal specimens (Richards et al. 2000) and from anatomically modern humans in Europe (Richards and Hedges 2000) during the late Pleistocene shows levels of meat eating that are indistinguishable from carnivores. It is interesting that this dietary transition occurs at about the same time as the hominid brain expanded beyond the size of the ape's brain (Aiello and Wheeler 1995).

The brain and cognitive development

Although it has long been recognized that intelligence is the most distinctive human trait, it is now becoming increasingly clear that our larger brains and greater intellectual capacities depend upon the stretching out of development at every stage. The production of cortical neurons in mammals is limited to early fetal development, and, compared to monkeys and apes, human embryos spend an additional 25 days in this phase (Deacon 1997; Parker and McKinney 1999). The greater original proliferation of neurons in early fetal development has cascading effects in greatly extending other phases of brain development, ultimately resulting in a larger, more complex, and more effective brain. For example, in monkeys, such as macaques, myelination of the brain begins prenatally and is largely complete in 3.5 years, whereas in humans this process continues for at least 12 years (Gibson 1986). Dendritic development is similarly extended to age 20 or later in humans.

The timing of cognitive development is extended in chimpanzees relative to monkeys, and in humans relative to apes (see Parker and McKinney 1999, upon which this discussion is based and references therein for reviews of comparative cognitive development in monkeys, apes, and humans). In terms of Piagetian stages, macaque monkeys traverse only two subperiods of cognitive development regarding physical phenomena by 6 months of age and peak in their logical abilities at around 3 years of age; however, they can never represent objects symbolically, classify objects hierarchically, or recognize themselves in a mirror. Chimpanzees traverse three to four subperiods of cognitive development by about 8 years of age.[3] They can recognize themselves in a mirror and are much better skilled at classification than macaques, but can never construct reversible hierarchical classes or engage in abstract, logical reasoning. Human children traverse eight subperiods of cognitive development over the first 18 to 20 years.

Although humans take about 2.5 times as long to complete cognitive development as do chimpanzees, humans actually learn faster. In most cognitive spheres, especially language, a 2-year-old child has the abilities of a 4-year-old chimpanzee. Humans have much more to learn and their brains require more environmental input to complete development. Formal abstract logical reasoning does not emerge until ages 16 to 18. This is the time when productivity begins to increase dramatically among modern hunter-

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gatherers. The ability to construct abstract scenarios and deduce logical relationships appears to induce a growth in knowledge that results in peak productivity in the mid-30s.

Elongated development in humans appears to be associated with slowed aging of the brain. Macaques exhibit physiological signs of cognitive impairment, as evidenced by Alzheimer-like neuropathology and cerebral atrophy by ages 22 to 25, and chimpanzees exhibit this by age 30. This contrasts with humans, for whom such changes are rare until age 60 (<1 percent) and only common (>30 percent) in their 80s (see Finch 2002; Finch and Sapolsky 1999 for reviews; however, the evidence on chimpanzees is mixed).

Physical growth

Physical growth in humans differs from that in chimpanzees and gorillas. It is first faster, then slower, and finally faster. Human neonates weigh about 3,000 grams (Kuzawa 1998), whereas mean birthweights for gorillas and chimpanzees are 2,327 and 1,766 grams (Leigh and Shea 1996). The differences are due not only to the longer gestation length among humans, but also to weight gain per day (ibid.). The comparison with gorillas is especially pronounced since adult female gorillas weigh about 60 percent more than average women among contemporary hunter-gatherers. Body composition also differs, with human neonates being much fatter (3.75 times more fat than that found in mammals of comparable weight), suggesting an even greater difference in the calories stored in human newborns (Kuzawa 1998).

It seems likely that these differences in neonatal body size are associated with the differences in brain growth rates. A human brain is twice as big at birth as a chimpanzee neonate's; indeed, it is about the same size as a chimpanzee adult's, despite the 15-fold greater total body weight of the latter. The bigger body and greater stores of energy in the form of fat are probably necessary to support the human brain and its rapid postnatal growth.

Following infancy and early childhood, humans grow absolutely more slowly and proportionally much more slowly than do chimpanzees. Growth is almost arrested for human children during middle and late childhood (see Figure 3). By age 10, chimpanzees have caught up with and surpassed human children in body size. Only with the adolescent growth spurt do humans achieve their final larger body size. Children in the foraging societies for which data are available do not acquire enough calories to feed themselves until they have completed growth. Growth is supported through within- and between-family food sharing (Kaplan et al. in press). Middle childhood is generally a time of low appetite, which is then followed by the voracious appetite associated with adolescence.

We propose that human infants grow quickly until both their body and gut are of sufficient size that they can comfortably support the large brain.

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FIGURE 3a Proportional weight change among males

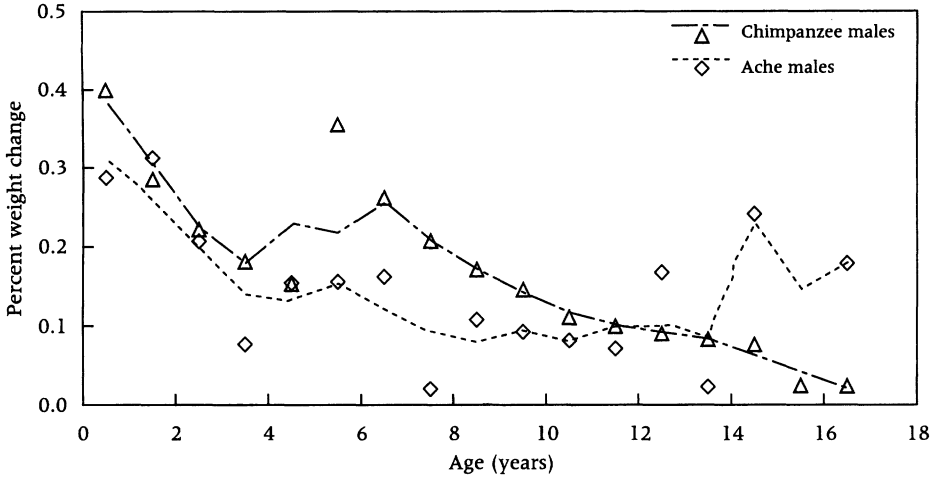
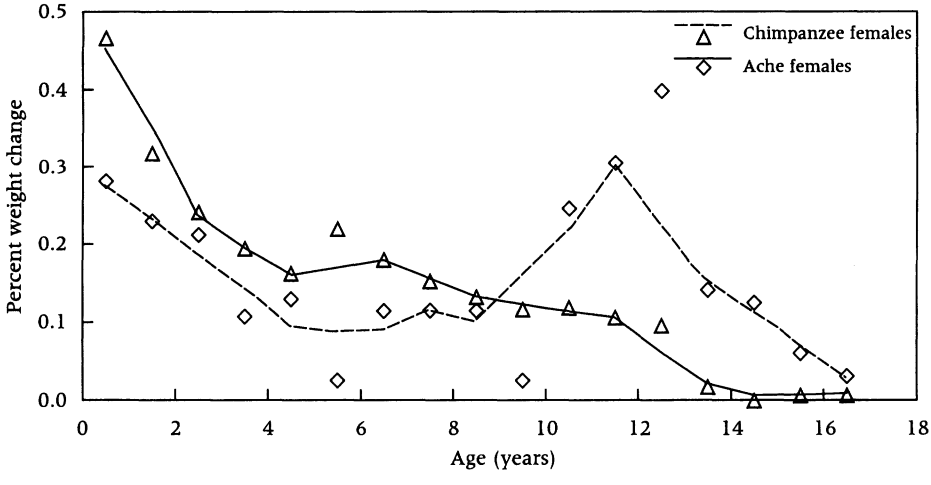


FIGURE 3b Proportional weight change among females



SOURCE: Ache data collected by Hill, Hurtado, and Kaplan. Chimpanzee data collected by Pusey and Williams, personal communication.

Growth rates are then slow, because children do not need large bodies since they do very little work. Instead they learn through observation and through play. When their brains are almost ready for large bodies, growth rates increase rapidly and adult body size is achieved relatively quickly.

Production, reproduction, and energy flows

Figure 4 compares age profiles of production for humans and chimpanzees. The chimpanzee net production curve shows three distinct phases. The first phase, lasting to about age 5, is the period of complete and then partial dependence upon mother's milk. Net production during this phase is nega-

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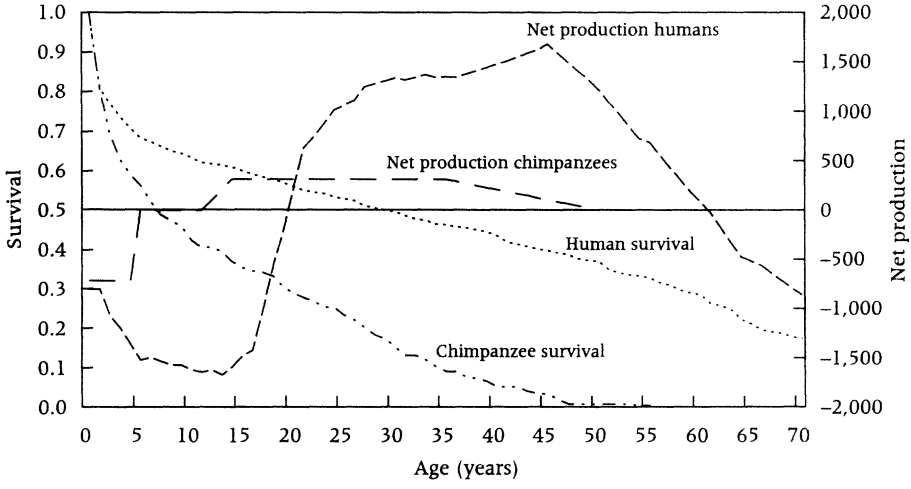
FIGURE 4 Net food production and survival: Human foragers and chimpanzees



SOURCE: Adapted from Kaplan and Robson (2002).

tive. The second phase, during which net production is zero, is one of independent juvenile growth, lasting until adulthood, about age 13 for females. The third phase is reproductive, during which females, but not males, produce a surplus of calories used for nursing.

Humans, in contrast, produce less than they consume for close to 20 years. Net production is negative and falling until about age 14, with the growth in consumption due to increased body size outstripping the growth in production, and only then begins to climb. Net production in adulthood among humans is much higher than among chimpanzees and peaks at a much older age. Peak net production among humans reflects the payoffs to the long dependency period. It is about 1,750 calories per day, but it is not reached until about age 45. Among chimpanzee females, peak net production is only about 250 calories per day, and since fertility decreases with age, net productivity probably decreases during adulthood.

This great increase in net production among humans during adulthood is a consequence of the difficulty of acquiring foods, as shown by the age profiles of production for collected, extracted, and hunted resources. In most environments, fruits are the easiest resources that people collect. Daily production data among Ache foragers show that both males and females reach their peak daily fruit production by their mid to late teens. Some fruits that are simply picked from the ground are collected by 2- to 3-year-olds at 30 percent of the adult maximum rate. Ache children acquire five times as many calories per day during the fruit season as during other seasons of the year (Kaplan 1997). Similarly, among the Hadza, teenage girls acquired 1,650 calories per day during the wet season when fruits were available but only 610 calories per day during the dry season when they were not. If we weight

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the wet and dry season data equally, Hadza teenage girls acquire 53 percent of their calories from fruits, compared to 37 percent and 19 percent for reproductive-aged and post-reproductive women (Hawkes, O'Connell, and Blurton Jones 1989).

In contrast, the acquisition rate of extracted resources often increases through early adulthood as foragers acquire the necessary skills. Data on Hiwi women show that root acquisition rates do not peak until about ages 35 to 45 (Kaplan et al. 2000), and the rate for 10-year-old girls is only 15 percent of the adult maximum. Hadza women appear to attain maximum root digging rates by early adulthood (Hawkes, O'Connell, and Blurton Jones 1989). Hiwi honey extraction rates by males peak at about age 25. Again the extraction rate of 10-year-olds is less than 10 percent of the adult maximum. Experiments among Ache women and girls clearly show that young adult girls are not capable of extracting palm products at the rate attained by older women (Kaplan et al. 2000). Ache women do not reach peak return rates until their early 20s. !Kung (Ju/'hoansi) children crack mongongo nuts at a much slower rate than adults (Blurton Jones, Hawkes, and Draper 1994b), and nut cracking rates among the neighboring Hambukushu do not peak until about age 35 (Bock 1995). Finally, even chimpanzee juveniles focus on more easily acquired resources than adult chimpanzees. Difficult-to-extract resources such as termites and ants and activities such as nut cracking are practiced less by chimpanzee juveniles than adults (Boesch and Boesch 1999; Hiraiwa-Hasegawa 1990; Silk 1978).

The skill-intensive nature of human hunting and the long learning process involved are demonstrated by data on hunting return rates by age (see Kaplan et al. 2001 for details on why hunting is so cognitively demanding). Hunting return rates among the Hiwi do not peak until ages 30 to 35, with the acquisition rate of 10-year-old and 20-year-old boys reaching only 16 percent and 50 percent of the adult maximum. The hourly return rate for Ache men peaks in the mid-30s. The return rate of 10-year-old boys is a mere 1 percent of the adult maximum, and the return rate of 20-year-old juvenile males is still only 25 percent of the adult maximum. Marlowe (unpublished data, personal communication) obtains similar results for the Hadza. Also, boys switch from easier tasks, such as fruit collection, shallow tuber extraction, and baobab processing, to honey extraction and hunting in their mid to late teens among the Hadza, Ache, and Hiwi (Blurton Jones, Hawkes, and O'Connell 1989, 1997; Kaplan et al. 2000). Even among chimpanzees, hunting is strictly an adult or near-adult activity (Boesch and Boesch 1999; Stanford 1998; Teleki 1973).

A complex web of intrafamilial and interfamilial food flows and other services supports this age profile of energy production. First, there is the sexual division of labor. Men and women specialize in different kinds of skill acquisition and then share the fruits of their labor. The specialization generates two forms of complementarity. Hunted foods acquired by men

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complement gathered foods acquired by women, because protein, fat, and carbohydrates complement one another with respect to their nutritional functions (Hill 1988). The fact that male specialization in hunting produces high delivery rates of large, shareable packages of food leads to another form of complementarity. The meat inputs of men shift the optimal mix of activities for women, increasing time spent in childcare and decreasing time spent in food acquisition (Hurtado et al. 1992). They also shift women's time to foraging and productive activities that are compatible with childcare and away from dangerous ones. In the ten-group sample, men, on average, acquired about twice as many calories and seven times as much protein as women (68 percent vs. 32 percent of the calories and 88 percent vs. 12 percent of the protein) (Kaplan et al. 2000). We estimate that on average 31 percent, 39 percent, and 30 percent of those calories support adult female, adult male, and offspring consumption (Kaplan et al. 2001). This implies that after taking into account own consumption, women supply only 3 percent of the calories to offspring and men provide the remaining 97 percent. Men supply not only all of the protein and fat to offspring, but also the bulk of the protein and fat consumed by women. This contrasts sharply with most (>97 percent) mammalian species, among which the female supports all of the energetic needs of an offspring until it begins eating solid foods (Clutton-Brock 1991) and males provide little or no investment. The high productivity of men has probably allowed for the evolution of physiological adaptations among women, such as fat storage at puberty and again during pregnancy—adaptations not found in apes.

Mortality

Figure 5 shows the mortality rates of chimpanzees (synthesized from five chimpanzee sites; Hill et al. 2001) and two foraging groups (Ache: Hill and Hurtado 1996: Table 6.1; Hadza: Blurton Jones et al. 2002: Table 2). Although there are differences among chimpanzee and human foraging populations, the contrast between the two species is clear. Before the age of 5, mortality rates are not very different between human foragers and chimpanzees. The average mortality rate during the adult period differs greatly between the two species, as does the age at which mortality rates rise steeply. Mortality within the two foraging groups remains quite low from adolescence to ages 35 to 40 and rises abruptly after about age 65. Among chimpanzees, mortality rates begin to rise quickly after their lowest point prior to reproduction.

The low adult mortality rates among humans are necessary components of delayed production. Only about 30 percent of chimpanzees ever born reach 20, the age when humans produce as much as they consume, and less than 5 percent of chimpanzees reach 45, when human net production peaks. The relationship between survival rates and age profiles of production is made

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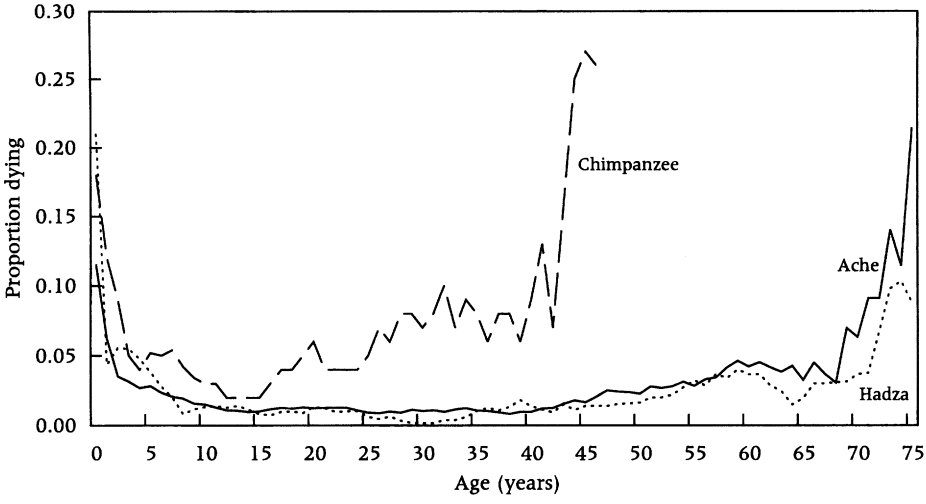
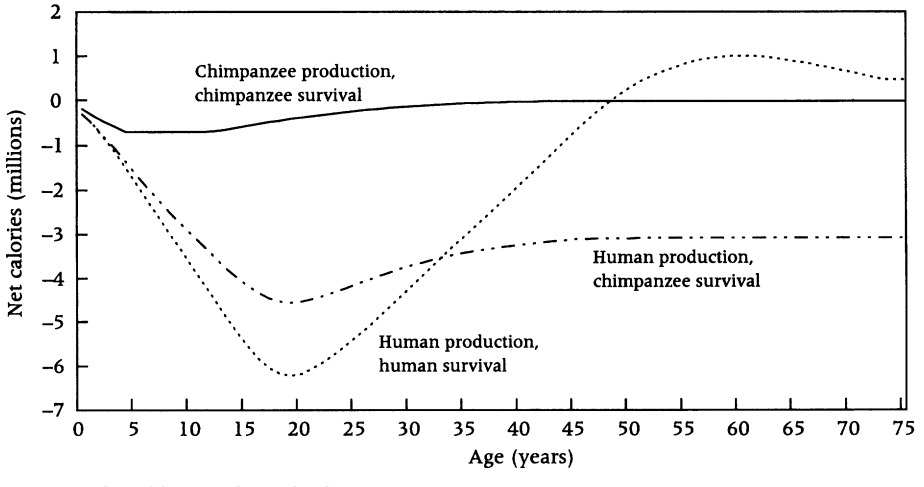
FIGURE 5 Age-specific mortality rates: Chimpanzees and human foragers



even clearer in Figure 6 (adapted from Kaplan and Robson 2002). This plots net expected *cumulative* productivity by age, multiplying the probability of being alive at each age times the net productivity at that age and then summing over all ages up to the present. The unbroken and dotted lines show *cumulative* productivity by age for chimpanzees and humans, respectively. The longer human training period is evident when the troughs in the human and chimpanzee curves are compared. The dashed line is a hypothetical cross of

FIGURE 6 Cumulative expected net caloric production by age: Humans and chimpanzees



SOURCE: Adapted from Kaplan and Robson (2002).

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human production profiles with chimpanzee survival rates. It shows that the human production profile would not be viable with chimpanzee survival rates, because expected lifetime net production would be negative.

Finally, although the mortality data for the Ache and the Hadza are based on small sample sizes and thus subject to considerable sample error, there is some evidence that proportional rates of change in mortality are not constant during the adult period. Figure 7 shows proportional rates of change in mortality. Mean yearly mortality rates were determined for the following age classes (10–14, 15–19, 20–29, 30–39, 40–49, 50–59, 60–69, 70–79) and then transformed into natural logarithms. Differences in those values between an age class and the preceding one were divided by the number of years in the age class to determine an average yearly rate of increase or decrease. For example, the first set of bars compares mortality rates of 10–14-year-olds with those of 15–19-year-olds and shows that rates decreased by 1 percent per year and 6 percent per year among the Ache and Hadza, respectively. For the Ache, the data show virtually no change in mortality rates from age 10 to age 39. From age 40 to age 69, rates of increase vary between 2 and 6 percent per year, with the lowest rate of increase occurring during the 60s. However, mortality rates show a steep rise to about 14 percent per year after age 70. For the Hadza, mortality rates actually decrease during the teens and 20s and stay essentially the same during the 30s. From age 40 to age 69, rates of increase vary between 0 and 9 percent per year, also with the lowest rate of increase during the 60s. Again, mortality rates show a steep rise to about 12 percent per year after age 70.

FIGURE 7 Yearly proportional change in mortality rates

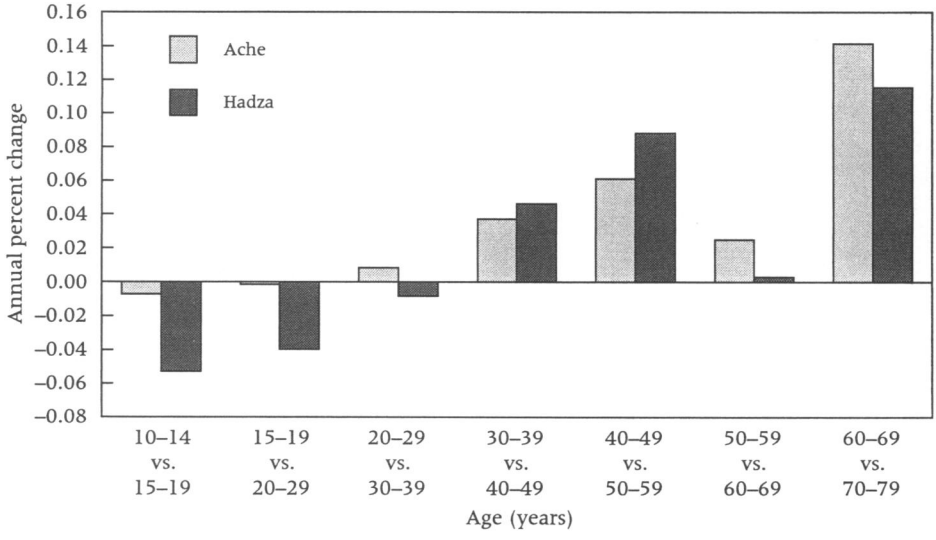

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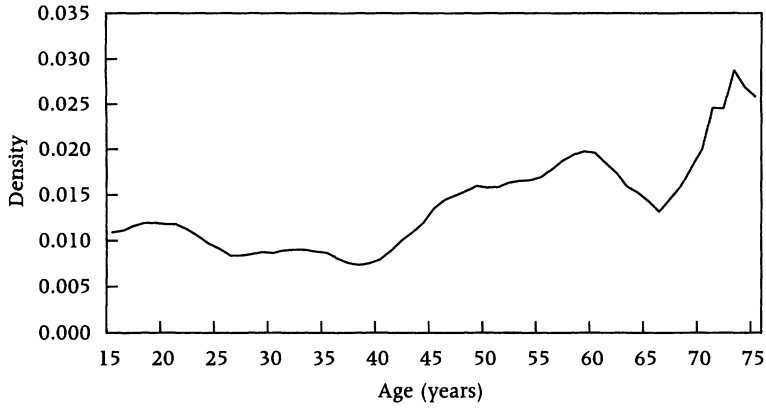
FIGURE 8 Adult mortality density



The relative constancy of mortality rates during prime adulthood is striking in both the Ache and the Hadza, as is the steep increase during the 70s. This suggests that "rectangularization" of the mortality profile among humans (Fries 1980, 1989) predates modernization. It may be a fundamental feature of our species, reflecting both strict genetic quality control before implantation of the fetus (as discussed in detail by Ellison 2001) and increased effort at slowing the rate of senescence during adulthood. Figure 8 shows the adult mortality density function, conditional on surviving to age 15, for the Ache and Hadza (averaging the two groups and then smoothing with a five-year running average). Although samples are small at older ages, there is an apparent mode at 73 years of age. The pattern also might suggest that a good candidate for the natural human life span is about 65 to 75 years, defined in terms of the imminence of death. This corresponds well with our impressions regarding physical deterioration. While some individuals show marked decline in their late 50s to early 60s, others remain vigorous to age 70 or so. After this physical decline, death often follows within a few years. Unfortunately, however, very little is known about the timing and population distribution of physical decline among hunter-gatherers or other traditional subsistence-level peoples.

The life span

The human life course and human life span

Our fundamental thesis in this chapter is that the human life course is an integrated adaptation to a specialized niche. Digestive physiology and anatomy; nutritional biochemistry; brain growth and cognitive development; tempo of body mass increases and appetite; age profiles of productivity, reproduction, parental investment, and mortality; and, ultimately, the life span

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are coadapted to a learning-intensive feeding niche, giving humans access to the most nutrient-dense and highest-quality food resources. The joint examination of these domains suggests a highly structured life course, in which six distinct stages can be recognized.

Brain growth occurs from the early fetal stage to about 5 years of age, with 90 percent occurring by age 3.0 to 3.5. Human mothers and their babies maintain large fat reserves to support this brain growth (Ellison 2001; Kuzawa 1998). While cognitive development unfolds over many more years, a great deal of linguistic competence, especially comprehension, is achieved during this first stage of life. Thus, it would seem that the human specialization evident in the first stage of life is building the "physical plant" (i.e., the brain) and the knowledge acquisition pathway (i.e., language ability) to support a long period of learning.

The second stage, childhood, is characterized by very slow physical growth, a large energetic allocation to building the immune system (McDade and Worthman 1999; Worthman 1999a), several important phases of cognitive development facilitated by play and other forms of practice, very low productivity, and very low mortality. Parents insist that children remain in safe places and encourage them to produce food only when it is easily and safely acquired (Blurton Jones, Hawkes, and Draper 1994a). The unique feature of human childhood is that it is fully supported by familial energy inputs, reducing exposure to mortality hazards and allowing time for learning. Faster physical growth would only make children more expensive before their brains were ready for food production.

Adolescence follows, during which physical growth is accomplished rapidly, the reproductive system matures, and the final phases of cognitive development occur. It is during this phase that the brain and the rest of the body become ready for adult productivity. While productivity increases during adolescence, it is also largely supported by familial food inputs.

For males, "on-the-job training" characterizes the period from early adulthood to prime adulthood in the mid to late 30s. Both physical strength and information-processing speed (fluid intelligence) peak in early adulthood, but knowledge-based abilities (crystallized intelligence) continue to rise (Horn 1968). As a result, productivity increases many-fold during this period. Mortality rates remain virtually constant and low. Resource production at this stage in the life course of women is very different. It is characterized by a reduction in productivity in the interests of fertility and parental investment. Women face a tradeoff between resource acquisition and childcare because the burden of children reduces foraging efficiency and because foraging often exposes children to environmental hazards. As a result, women reduce their work or shift their activities toward food processing and other, less dangerous, efforts.

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Middle age is a period of simultaneous parenthood and grandparenthood. Dependency loads on parents peak around age 40, just before grandparenthood begins. Although productivity is also at a peak level, net productivity including dependency loads is negative, supported by resource transfers from other families. Through middle age, dependency loads diminish, as does productivity.

Old age commences around age 60, and during this seventh decade of life physical deterioration proceeds rapidly and brain aging becomes evident, followed by a sharp increase in mortality rates. Parenting is finished and work effort decreases along with productivity. This is not to say that there are no positive contributions to fitness during this phase. Older adults attempt to be as productive as possible, reallocating their time to skill-intensive but less energy-intensive activities (e.g., craft production and childcare; Gurven and Kaplan 2001). They may also affect the productivity of the younger population through their knowledge of the habitat and through their political skills.

If this set of stages characterized the average life course of the last 100,000 to 200,000 years, it is likely that the processes of maintenance and repair from the intracellular level to the whole-organism level were shaped by selection to achieve a life span including all six stages. One possibility is that the sixth stage is actually an artifact of selection intended to maintain the body and mind in good condition through the first five stages of life. The sixth stage would then be the result of the impossibility of nature's designing a body that collapses the moment high functionality is no longer needed. Another possibility is that this sixth stage has been positively selected because of the fitness benefits produced during it.

Some (e.g., Hawkes et al. 1998; Williams 1957) have hypothesized that the benefits provided by grandmothers to grandchildren have selected for menopause and the long human life span. The data on child dependency burdens suggest an alternative hypothesis: the life span is the result of selection on adults, allowing them to support all of their children through adolescence, and this requires high productivity to about age 60 (Lancaster and King 1985; see Peccei 2001 for a recent statement and review). The post-reproductive period might then not begin until children are fully reared. Grandchildren are only half as related to grandparents as children are to parents. Therefore, at the margin, the benefits to grandchildren would have to be twice as great as those to children to favor the same investment in continued life.

This hypothesis of course depends upon menopause, which must be explained as well. It is still unclear whether menopause is the result of tradeoffs between egg quality and reproductive life span, tradeoffs between supporting multiple dependent young and production of additional offspring, tradeoffs between grandparental investment and reproduction, or something else (see Kaplan 1997 for a discussion).

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Flexibility and variation in the human life span

The human brain is the physical medium through which culture is maintained and transmitted. As such, it is generally thought to have greatly expanded the behavioral flexibility of our species relative to other animals. However, the commitment to building and programming the brain requires a highly structured life history that places constraints on the timing of life events. Our species is committed to long-term neural and cognitive capital accrual and to a long life span. The characteristic life history of our ancestors has shaped age profiles of growth, tissue repair, and physical decline.

Nevertheless, human life histories show evidence of systematic variation in response to environmental variation. Those effects appear to be the result of the interaction between changes in environmental conditions and human physiology and behavior. Perhaps the most striking example of that interaction is the pattern of changes accompanying the secular trend toward modernization. Increased nutrition and decreased work and disease loads have systematic effects on human developmental physiology. Physical growth rates have increased and maturation now begins earlier, resulting in greater stature, higher body weight, and earlier age of menarche in girls (Eveleth 1986; Lancaster 1986; Worthman 1999b). This response is very likely the result of adaptive flexibility in growth and maturation in the face of variation in food supply and in disease assault rates that was experienced during human evolutionary history.

In contrast to this increase in the rate of physical development, aging may be slowed in response to better nutrition and decreased work and disease loads. Although it is possible that humans would also show slowed aging in response to radical reductions in caloric intake (as do rats in feeding experiments—see Shanley and Kirkwood 2000 for a review), it is also possible that within the usual range of variation, rates of aging are slowed and life spans are lengthened when nutrition is better and disease loads are lower (Fogel and Costa 1997). This outcome would also be adaptive. The changing mortality rates among older people accompanying modernization and the fact that some chronic diseases occurred at earlier ages in the nineteenth-century United States than today (Costa 2000) are consistent with this possibility.

On the other hand, increased risk of heart disease, diabetes, and cancer from overweight and lack of exercise may also be the result of evolved responses. Given the common activity regimes in our past and the variability in food supply, human appetites and nutritional biochemistry may be designed to store fat and increase blood lipid levels when food is abundant. Those adaptations might reduce the life span in the context of modern patterns of activity levels and food access and consumption.

In addition to these physiological adaptations, there are also behavioral responses to modernization. The models outlined in our first section

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above are as applicable to short-term behavioral variation as they are to long-term life-history adaptations. Two noteworthy effects of modernization are increased economic payoffs to educational capital and decreased mortality owing to improvements in public health. The present models predict reinforcing endogenous behavioral responses to such changes. Increased payoffs to education should promote increased investment both in educational capital and in staying alive. Improvements in public health should also promote reinforcing increases in capital investment and staying alive. It is an open question whether we are reaching the upper limit of our flexibility in the life span. It appears that with respect to stature and perhaps age of menarche, we have reached the limit. There appears to be more scope for variation in the life span, given investments in medical technology designed to reduce disease and the effects of aging. In any case, knowledge about the human genome is likely to lead to manipulations of genes and gene products, resulting in life span increases of very large magnitude.

Building blocks for an adequate theory of senescence and the life span

Given the definition of life span guiding this discussion (the span from birth to the age when death is imminent as a result of physical deterioration), an adequate theory of life span will require a theory of senescence. Each of the principal theories suffers from important weaknesses, rendering the theory incomplete. This section first reviews those weaknesses and then offers a framework intended to remedy them.

Senescence is generally defined as an increasing mortality rate with age. The theories proposed by Medawar (1952), Williams (1957), and Hamilton (1966) share the common premise that senescence is the manifestation of the decreasing force of selection with age, resulting from extrinsically imposed hazards of mortality. In essence, their argument is that sources of mortality that strike when the individual is old are less stringently selected against than those striking when young, simply because an individual has a greater probability of being young than old.

According to Williams (1957), antagonistic pleiotropy accounts for the increase in mortality with age. Senescence is due to the presence of genes with opposing pleiotropic effects at different ages, increasing survival at younger ages but decreasing survival at older ages or increasing fertility at younger ages at the expense of reduced survival at older ages. Since extrinsically imposed mortality decreases the force of selection with age, genes with such positive effects at younger ages and negative effects at older ages will accumulate through selection, producing increasing mortality rates with age. He speculated that selection on mortality at each age would vary with reproductive value (i.e., expected future reproduction conditional on being alive at that age). Hamilton (1966) formalized this argument and showed

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that sensitivities of fitness to changes in mortality rates decrease with the age of action. However, his results suggested that reproductive value is not the critical determinant, because the sensitivity of fitness to changes in mortality rates depends both on the probability of reaching that age and on expected future reproduction at that age.

According to the formal model discussed above, the fact that mortality discounts the future does not, in itself, select for increasing mortality rates with age. Our approach allows both capital investments and expenditure on mortality reduction to be subject to natural selection. If aging, in the sense of explicit time dependence, is not built-in, then positive but constant mortality rates and a constant capital stock are optimal. This is so because our model assumes that current mortality can be reduced by current energy expenditure. From an initial viewpoint, reducing future mortality is indeed less important than reducing present mortality, just because survival is not certain. However, future expenditures on mortality reduction should be discounted for the same reason, and in the long run these two effects are precisely offsetting. If mortality is avoided during a time interval, the organism faces the same tradeoff between reproduction and survival as earlier.

This result does not depend on the assumption that current mortality is reduced by current expenditures. An alternative model might assume the existence of a stock of somatic capital for determining mortality, as seems a plausible explicit interpretation of Kirkwood's (1990) "disposable soma theory." However, it can be shown that the optimal life history may again entail a steady state in which this stock, and hence the mortality rate, are constant. From a current viewpoint, the tradeoff between the present and the future, as reflected in the decision about the size of the stock, is stationary. Even when decisions are made at the beginning of life, both the costs and benefits of future mortality control are deflated in the same way. Finally, even if somatic capital depreciates over time, results from capital investment theory show that the stock will be maintained at an optimal level (Arrow and Kurz 1970; Intriligator 1971). Optimal investment in the capital stock will continue to precisely offset depreciation. As long as production is constant with a constant capital stock, optimal mortality rates remain constant as well.[4]

What then causes senescence? Suppose there are cost functions for building embodied capital and for repairing and maintaining embodied capital. Imagine that the embodied capital stock is described in terms of two state variables, the quantity of the stock and its efficiency. From its inception, the organism builds somatic capital, adding to its quantity until the optimal quantity is reached. However, because of its own metabolic activity and assaults from outside agents, the efficiency of the capital stock is subject to decay. For example, free radicals and other harmful molecules may accumulate in cells, accidents may cause tissue damage, and pathogens may

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disrupt physiological function and damage cells. In the same sense that the costs of producing new car seats may be different from the costs of repairing tears in those seats, the costs of producing new cells and adding to the quantity of embodied capital may be different from the costs of repairing damage to them. Thus, during development, the optimal life-history program will have to equalize marginal fitness returns from three different investments: adding to new capital, repairing existing capital, and reducing current mortality. When the capital stock reaches the level at which some allocation to reproduction is also optimal, marginal returns from investments in producing new capital in the form of descendants must also be equalized with the marginal returns from the other three investments.

It is possible that for many capital stocks, quantity increases with age while efficiency decreases because, as suggested by Kirkwood (1990), optimal repair is not complete. Such a process appears to characterize cognitive development and aging. For example, verbal knowledge tends to increase, at least through middle age, but information-processing speed and memory peak during the third decade and decline thereafter (Schaie 1996). During the first phase of life, those increases in quantity have larger effects on the value of life (i.e., the expected future contribution to fitness) than do the decreases in efficiency. Thus productivity increases and optimal mortality decreases. However, at some age, the effects of disrepair overwhelm the effects of growth and learning, and overall productivity and the ability to fend off mortality hazards are lessened. This would lead to increasing optimal mortality with age.

This framework, with four kinds of investment, may provide a more adequate basis for a general theory of life-history evolution, and of life span in particular. Without making the unrealistic assumption that any life-history component, such as mortality, is extrinsic, selection can act to optimize and coadapt each of those components. Given the results discussed above, it is plausible that exogenous factors increasing the productivity of capital should select not only for increased capital investments and reduced mortality rates, but also for higher optimal allocations to repair, because of the higher probability of reaching old age. This would then lead to a longer life span. Similarly, exogenous factors that affect mortality rates may select not only for greater capital investment and reinforcing increases in optimal allocations to reducing mortality rates, but also for slower senescence. Given niche differentiation in the payoffs to body size and learning, in the payoffs to investment in offspring, in the payoffs to reducing mortality rates, and possibly in the payoffs to repair, the wide array of life histories found in nature is not surprising.

Whether such a framework will be useful awaits the formal development of models designed to analyze the effects of selection on those investment functions. It does, however, direct empirical attention to measuring

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the shapes of those functions. What are the costs and benefits of reducing the quantities of harmful molecules in cells, of DNA repair, and of differing numbers and kinds of immune cells? How do those costs and benefits compare to those associated with increases in muscle mass, brain mass, and learning? And, how do each of those functional relationships compare with those characterizing the production of offspring of different sizes and different functional abilities? Regardless of the ultimate productivity of this evolutionary economic life-history framework, understanding those relationships is likely to be illuminating.

Notes

This chapter was written with support from the National Institute on Aging, grant number AG15906. The authors acknowledge the contributions of Kim Hill to the data sets and their analyses on the comparative diets and demography of chimpanzees and foragers published previously (Kaplan et al. 2000). We also thank Kim Hill and Magdalena Hurtado for their data on resource acquisition by age and sex among the Hiwi and Ache (Kaplan et al. 2000). These data sets and analyses form a critical base for the second part of this chapter.

1 Of course, a quantitative definition would require specification of that ratio.

2 In some cases, there may be some additional mortality increases and drops due to phase transitions such as weaning.

3 The fourth subperiod, covering abilities such as recognition of conservation of quantities of liquids under container transformations, seems to require tutelage and symbolic training.

4 Medawar's (1952) model, in which deleterious mutations with age-specific effects late in life accumulate owing to the weaker force of selection at older ages, does not suffer from this problem. However, it can only account for specific diseases with late onset, not for the general and progressive deterioration of the soma with age, nor the physiological processes underlying aging.

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