

An Evolutionary Theory of Human Life Span: Embodied Capital and the Human Adaptive Complex

3

Hillard Kaplan
Michael Gurven
Jeffrey Winking

The future of human longevity remains an open question and is the subject of a vigorous debate among population scientists. There are scientists who propose that human life expectancy is not likely to exceed 85 years (Fries, 1989; Hayflick, 2007). Others suggest that life expectancy may reach 100 years in the 21st century (e.g., Vaupel, 1997). The difference between these two positions stems from beliefs about the relative importance of environmental and genetic variance in determining life expectancy. Those who expect limited future gains in life expectancy believe that most of the existing gains came from improvements in preadult survivorship and that there is an innate program of physiological decay. On the other hand, those who expect greater improvements base their views on evidence suggesting that secular trends in life expectancy due to improvements in the environment (broadly conceived in terms of public health,

social services, and so on) show no signs of deceleration to date. At the same time, even as medical science is advancing, there are trends toward increasing obesity, diabetes, and hypertension in both developed and developing nations (Crimmins et al., 2005; Hossain, Kavar, & El Nahas, 2007; Seidell, 2000), causing concern that those environmental improvements may be offset by food abundance and greater ensuing risk for those diseases.

An understanding of how age-specific mortality, the aging process, and behavior respond to novel environmental variation requires a theory of how natural selection has acted on our biology over the course of human evolution. This chapter presents such a theory and attempts to make sense of those two opposing views, suggesting that both are partially correct and partially incorrect. We argue that in the environments in which humans spent the majority of their evolutionary history, natural selection has resulted in a species-typical human life span of about seven decades, as part of a larger adaptive complex. We also argue that the human response to the novel environments of today grows out of our evolutionary history and, to a large extent, is predictable.

The chapter begins with a general framework for understanding gene-environment interactions affecting age-specific mortality and longevity and the role of natural selection in determining the evolution of population gene distributions over time. We then present an extension of standard life history theory, which we term the “embodied capital theory of life history evolution.” These ideas are then applied to an understanding of the human case, and we argue that the adaptive niche occupied by our species selected for a coevolved suite of characteristics, or the *human adaptive complex*. We then present the theory about why the ability to live at least to age 65 has played an important role in the human adaptation, without which many other human characteristics could not have evolved. Together with a long life span, these characteristics form an adaptive peak that is unique to humans, with other species being at other peaks in the adaptive landscape. The chapter concludes with a discussion of the present and future that derives from this theory.

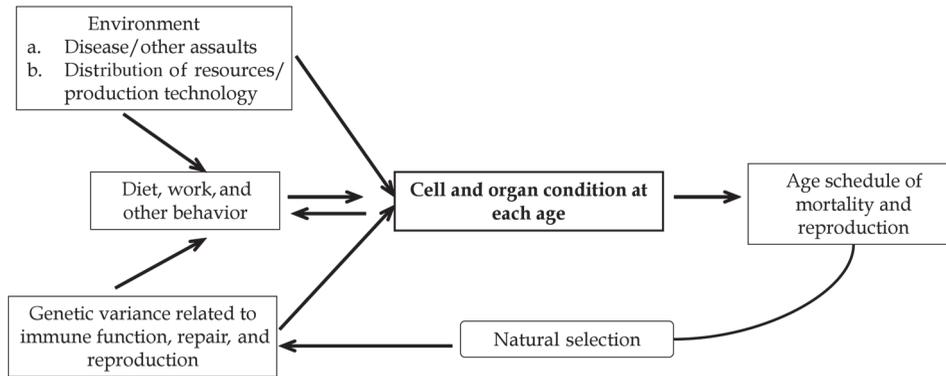
A General Framework

Figure 3.1 depicts graphically a conceptual framework for analyzing the factors underlying the evolutionary process relevant to aging and its demographic and physiological outcomes. The two boxes to the left represent the gene-environment interactions that determine diet, work effort, and state of cell and organ tissue by age. Beginning with the upper box, human populations, over evolutionary time, experienced distributions of environmental assaults, including viral and bacterial disease, parasitism, predation, accidents, and trauma. While those assaults undoubtedly varied over time and geography, there was likely a characteristic range of variation to which evolving humans were exposed during the 2 million years our species lived as nomadic foragers.

With respect to pathogen burden, it is likely that evolving hominids were exposed to an array of pathogens, many common to other wild primate species (Nunn et al., 2004). A general assumption is that virgin-soil epidemics were mostly nonexistent in isolated populations prior to modernization and the advent of agriculture and large-scale settlement (Fiennes, 1978; McNeil, 1989).

3.1

Effects of natural selection on organ condition and age schedules of mortality and reproduction.



While there is a great deal of uncertainty regarding disease exposure during our evolutionary past, recent work suggests that common infections that have been traditionally associated with high population density, such as tuberculosis (Buikstra, 1981; Clark, Kelley, Grange, & Hill, 1987) and trypanosomiasis (Coimbra, 1988), were present in pre-Columbian South America. The presence of antibodies to viral infections, such as herpes, Epstein-Barr, and varicella, has been documented in relatively isolated Amazonian groups (Black, Woodall, Evans, Liebhaver, & Henle, 1970; Salzano & Callegari-Jacques, 1988). There is strong evidence that cytomegalovirus, Epstein-Barr virus, pneumonias, intestinal geohelminths, herpes, hepatitis B, and arboviruses have long coexisted among precontact Amazonian populations (Black, 1975). More important, recent phylogenetic evidence for a variety of pathogens that were previously assumed to postdate the advent of agriculture and animal domestication also strongly suggests an earlier evolutionary history of exposure to many common pathogens, including smallpox, falciparal malaria, and tuberculosis (see review in Pearce-Duvet, 2006). Sexually transmitted diseases also likely have a long evolutionary history among humans (Donovan, 2000). While virulence of some pathogens may have changed with dense reservoirs of human and animal hosts, ancestral humans were likely exposed to a wide diversity of viruses, bacteria, protozoa, and parasites (Finch & Stanford, 2004).

[AuQ1]

At the same time as evolving humans faced a distribution of environmental assaults, they also lived in environments with characteristic distributions of food resources and characteristic technologies that changed over evolutionary time. It appears that humans, at least over the past several hundred thousand years, have eaten an omnivorous diet based on both hunting and gathering, with much higher amounts of meat than other primates (Kaplan, Hill, Lancaster, & Hurtado, 2000). This, along with advents in cooking technology (Wrangham, Jones, Laden, Pilbeam, & Conklin-Brittain, 1999), allowed them to do much less physiological processing, such as bacterial fermentation of leaves, in order to have adequate amounts of fat and protein in their diet.

Genes (depicted in the lower left of Figure 3.1) that control defenses against pathogens, repair of cell damage, and reproduction interact with those environmental assaults to determine population distributions of individuals of different ages and their associated physical states at the cellular and organ levels. Genes also interact with environmental conditions (including distributions of energy in the environment and production technologies) and with physical state to determine diet, work, energy budget, and reproductive behavior. Those behavioral patterns have feedback effects on physical state since work exposes people to risks of injury and physical stress but also provides energy to support repair, immune defenses, and reproduction. Both changing physical states with age and behavior result in mortality and reproductive schedules with age.

Population variance in age-specific mortality and reproductive schedules associated with genetic variation in turn result in natural selection since some genotypes are associated with lower or higher fitness than others. Ultimately, this process of natural selection feeds back on the distribution of genotypes in subsequent generations. Presumably, this process produces a distribution of genotypes that tends to maximize fitness over generational time (Hamilton, 1966).

Embodied Capital, Development, and Aging

Two fundamental trade-offs determine the action of natural selection on reproductive schedules and mortality rates. The first trade-off is between current and future reproduction. By growing, an organism can increase its energy capture rates in the future and thus increase its future fertility. 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 lifetime fitness more than continued growth. Similarly, among organisms that engage in repeated bouts of reproduction (humans included), some energy during the reproductive phase is diverted away from reproduction and allocated to maintenance so that they 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 descendants are maximized (Gadgil & Bossert, 1970). Variation across taxa and across conditions in optimal energy allocations is shaped by ecological factors, such as food supply, disease, and predation rates.

A second fundamental life history trade-off is between offspring number (quantity) and offspring fitness (quality). This trade-off occurs because parents have limited resources to invest in offspring and each additional offspring produced necessarily reduces average investment per offspring. Most biological models (Lack, 1954; Lloyd, 1987; Smith & Fretwell, 1974) operationalize this trade-off as number versus survival of offspring. However, parental investment may affect not only survival to adulthood but also the adult productivity and fertility of offspring. This is especially true of humans. Thus, natural selection is expected to shape investment per offspring and offspring number so as to maximize the product of offspring number and average per-offspring lifetime fitness.

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 trade-off becomes a trade-off between investments in own embodied capital and reproduction, and the quantity–quality trade-off becomes a trade-off 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, 1982; Fleagle, 1999; Parker & McKinney, 1999). Among humans, the brain supports learning and knowledge acquisition during both the juvenile and the adult period, well after the brain has reached its adult mass. This growth in the stock of knowledge and functional abilities is another form of investment.

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% 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 and thus decrease early performance. The incompetence of human infants—and 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 then fully realized only as the organism ages. In a niche where there is little to learn, a large brain might have higher costs early in life and a relatively small impact 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 requires exposure to antigens in order to become fully functional. 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.

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, & Hakeem, 1993; Hakeem, Sandoval, Jones, & Allman, 1996; Judge & Carey, 2000). 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 (Abrams 1993; Carey, 2001; Williams 1957).

This logic suggested an alternative approach to standard evolutionary treatments. Standard treatments generally define two types of mortality: (a) extrinsic, which is imposed by the environment and is outside the control of the organisms (e.g., predation or weather), and (b) intrinsic, over which the organism can exert some control over 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 & Berrigan, 1993; Kozlowski & Wiegert, 1986). Models of aging and senescence (Promislow, 1991; Shanley & Kirkwood, 2000) frequently treat aging as affecting intrinsic mortality, with extrinsic mortality, in turn, selecting for rates of aging. For example, in the Makeham-Gomperz 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 misleading 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 facilitating flight. Each of those 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.

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. 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 analysis 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 their analysis 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 with informal graphical illustrations.

Capital Investments and Endogenous Mortality

As a first step, it is useful to think of capital generally 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 that are endogenous and subject to selection. It can be reinvested in increasing the capital stock, that is, in growth. Define $\nu(t)$ as flow of investment at time t , so that dK/dt equals $\nu(t)$. Since growth and development take time, it is useful to impose a maximal investment rate, $\bar{\nu}$. Some energy, s , may also be allocated to reducing mortality, μ , for example, via increased immune function. 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 also be used for reproduction, which is the net excess energy available after allocations to capital investments and mortality reduction, y ; thus, $y(t) = F(K) - \nu(t) - s(t)$.

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 (e.g., 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 (Kaplan & Robson, 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 ν 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 when capital is at its optimum level, and both capital and mortality rates remain constant.

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 decreases mortality through increases in s because it increases the value of life. A reduction in mortality rates has two similar effects. It increases the optimal capital stock and produces a reinforcing increase in s . Overall, these two effects would tend to increase the expected life span.

It is interesting to 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 $dK/dt = (1 - \lambda)K(t) + \nu(t)$, with λ being the proportional depreciation rate—a steady state still would be achieved where depreciation would be exactly offset by investment (Arrow & Kurz, 1970; Intriligator, 1971).

In a recent paper, Kaplan and Robson (n.d.) provide a model of senescence by disaggregating embodied capital into its two dimensions of *quantity* and *quality*. The quantity of embodied capital can be defined in terms of the number of cells in the soma, and cell quality, interpreted as functional efficiency in our model, is endogenous. Its deterioration can always be slowed or reversed by investment in repair. Without such investment, cell quality depreciates over time because of the buildup of deleterious by-products of cell metabolism. For example, somatic capital can be disaggregated into its two components, the quantity and quality of tissue, where optimal levels of quality decline with age, even when quantity grows and then remains stable.

A key result from these analyses is that key life history variables under selection, such as time spent growing (i.e., the capital investment period), mortality rates, and reproductive rates, jointly respond to ecological parameters and therefore coevolve. The settings of different ecological parameters, such as the productivity of somatic capital and the costs of mortality reduction, jointly determine the *complex* of life history characteristics exhibited by a species. In the next section, we illustrate the components of an adaptive complex for the human case.

Life Span and the Human Adaptive Complex

Our proposal is that natural selection impacting immune function, DNA repair, energy metabolism, growth, and behavior produced a specific life history pattern in the environmental context in which humans evolved. In particular, we hypothesize that the ecological niche that our ancestors occupied in the majority of the world's environments consisted of a coadapted set of traits, including (a) the life history of development, aging, and longevity; (b) diet and dietary physiology; (c) energetics of reproduction; (d) social relationships among men and women; (e) intergenerational resource transfers; and (f) cooperation among related and unrelated individuals (Gurven & Kaplan, 2006; Gurven, Kaplan, & Gutierrez, 2006; Gurven & Walker, 2006; Kaplan, 1997; Kaplan & Gurven, 2005; Kaplan, Lancaster, & Hurtado, 2001; Kaplan, Mueller, Gangestad, & Lancaster, 2003; Kaplan & Robson, 2002; Kaplan et al., 2000, Robson & Kaplan, 2003). We refer to this set of traits as the *human adaptive complex* (HAC).

According to the theory, the HAC is a very specialized niche, characterized by (a) the highest-quality, most nutrient-dense, largest-package-size food resources; (b) learning-intensive, sometimes technology-intensive, and often cooperative food acquisition techniques; (c) a large brain to learn and store a great deal of context-dependent environmental information and to develop creative food acquisition techniques; (d) a long period of juvenile dependence to support brain development and learning; (e) low juvenile and even lower adult mortality rates, generating a long productive life span and population age structure with a high ratio of adult producers to juvenile dependents; (f) a three-generational system of downward resource flows from grandparents to parents to children; (g) biparental investment with men specializing in energetic support and women combining energetic support with direct care of children; (h) marriage and long-term reproductive unions; and (i) cooperative arrangements among kin and unrelated individuals to reduce variance in food

availability through sharing and to more effectively acquire resources in group pursuits. Following is a review of some evidence supporting this view.

Diet, Net Food Production Over the Life Course, and Intergenerational Transfers

There is now mounting evidence from various sources, including digestive anatomy, digestive biochemistry, bone isotope ratios, archaeological assemblages, and observational data on 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 (Dunn, 1968), more than half the calories in hunter-gatherer diets are derived from animal meat. There are 10 foraging societies and five chimpanzee communities for which caloric production or time spent feeding were monitored systematically (Kaplan et al. 2000). Modern foragers all differ considerably in diet from chimpanzees. Measured in calories, the major component of forager diets is vertebrate meat. This ranges from about 30% to around 80% of the diet in the sampled societies with most diets consisting of more than 50% vertebrate meat (equally weighted mean = 60%), whereas chimpanzees obtain about 2% 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 on qualitative ethnographies, Cordain et al. (2000) found median dependence on animal foods of 66% to 75%.

The next most important food category in the 10-society sample is extracted resources, such as roots, nuts, seeds, most invertebrate products, and difficult-to-extract plant parts, such as palm fiber or growing shoots. They may be defined as nonmobile resources that are embedded in a protective context such as underground or in hard shells or bearing toxins that must be removed before they can be consumed. In the 10-forager sample, extracted foods accounted for about 32% of the diet, as opposed to 3% 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% of the chimpanzee diet, on average, and only 8% of the human forager diet. The data suggest that humans specialize in rare but nutrient-dense resource packages or patches (meat, roots, and nuts), whereas chimpanzees specialize in ripe fruit and low-nutrient-density plant parts.

Although the data are still relatively thin, it appears that this dietary shift can be traced to the origins of the genus *Homo* about 2 million years ago. Compared to chimpanzees and australopithecines, early *Homo* appears to have a reduced gut (Aiello & Wheeler, 1995), and radio-isotope data from fossils also suggest a transition from a more plant-based diet to greater reliance on meat (Schoeninger, Bunn, Murray, Pickering, & Moore, 2001). There is significant archaeological evidence of meat eating by *Homo* in the early Pleistocene (Bunn, 2001), and radio-isotope evidence from Neanderthal specimens (Richards & Hedges, 2000) and anatomically modern humans in Europe (Richards & Hedges, 2000) during the late Pleistocene show levels 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 brain around 2

million years ago (Aiello & Wheeler, 1995). The next section discusses the comparative evidence on brain development and its psychological correlates.

Figure 3.2 compares human and chimpanzees in terms of age profiles of net production (food produced minus food consumed) and mortality rates. Seen on the right vertical axis, 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 on mother's milk. Net production during this phase is negative. The second phase during which net production is zero is 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 that they allocate to nursing.

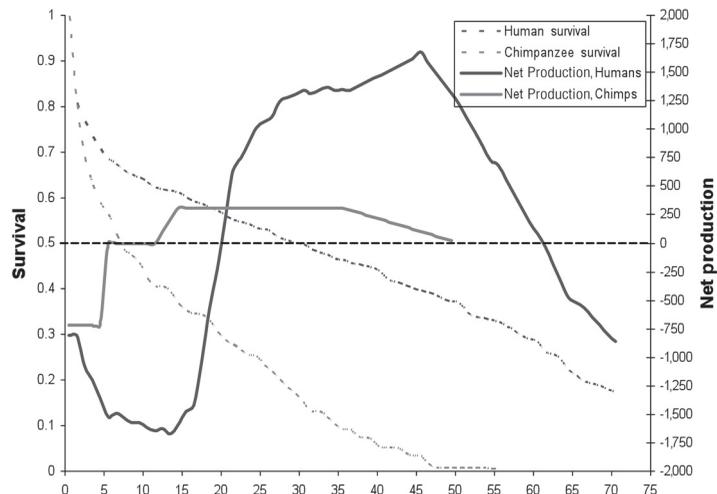
Humans, in contrast, produce less than they consume for close to 20 years. Net production becomes increasing negative until about age 14 (with growth in consumption due to increased body size outstripping growth in production) and then begins to climb. Net production in adulthood among humans is much 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 the adult period.

This delay in productivity and then the great increase during adulthood is due to the difficulty involved in acquiring foods. In most environments, fruits are the easiest resources that people acquire. 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% 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, teen girls acquired 1,650 calories per day during the wet season when fruits were available and only 610 calories per day during the dry season when fruits were not. If we weight the wet- and dry-season data equally, Hadza teen

3.2

Net food production and survival: Human foragers and chimpanzees.

Adapted from Kaplan et al. (2000).



girls acquire 53% of their calories from fruits, compared to 37% and 19% for reproductive-aged and postreproductive women, respectively (Blurton Jones, Hawkes, & O'Connell, 1989).

In contrast to fruits, the acquisition rate of extracted resources often increases through early adulthood as foragers acquire necessary skills. Data on Hiwi women show that root acquisition rates do not asymptote until about age 35 to 45, and the rate of 10-year-old girls is only 15% of the adult maximum. Hadza women appear to obtain maximum root digging rates by early adulthood (Blurton Jones et al., 1989). Hiwi honey extraction rates by males peak at about age 25. Again the extraction rate of 10-year-olds is less than 10% of the adult maximum (Kaplan et al., 2000). Experiments done with Ache women and girls clearly show that young adult girls are not capable of extracting palm products at the rate obtained by older Ache 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, & Draper, 1994), and Bock (1995) has shown that nut-cracking rates among the neighboring Hambukushu do not peak until about age 35. Finally, chimpanzee juveniles also focus on more easily acquired resources than adult chimpanzees. Difficult-to-extract activities, such as termite and ant fishing or nut cracking, are practiced less by chimpanzee juveniles than adults (Boesch & Boesch, 1999; Hiraiwa-Hasegawa, 1990; Silk, 1978).

The skill-intensive nature of human hunting and the long learning process involved are demonstrated dramatically by data on hunting return rates by age (for details regarding why hunting is so cognitively demanding, see Kaplan et al., 2001). Hunting return rates among the Hiwi do not peak until age 30 to 35 with the acquisition rate of 10-year-old and 20-year-old boys reaching only 16% and 50% of the adult maximum, respectively. The hourly return rate for Ache men peaks in the mid-30s. The return rate of 10-year-old boys is about 1% of the adult maximum, and the return rate of 20-year-old juvenile males is still only 25% of the adult maximum (Walker et al., 2002). Marlowe (unpublished data) obtains similar results for the Hadza. In addition, 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 et al., 1989, Blurton Jones, Hawkes, & O'Connell, 1997; Kaplan et al., 2000). Even among chimpanzees, hunting is strictly an adult or subadult activity (Boesch & Boesch, 1999; Stanford, 1998; Teleki, 1973).

A complex web of intra- 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, however, specialize in different forms of skill acquisition with correspondingly different foraging niches and activity budgets and then share the fruits of their labor. The specialization generates two forms of complementarity. Hunted foods acquired by men complement gathered foods acquired by women because protein, fat, and carbohydrates complement one another with respect to their nutritional functions (Hill, 1988) and because most gathered foods, such as roots, palm fiber, and fruits, are low in fat and protein (nuts are an exception). 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 child care and decreasing time spent in food

acquisition. They also shift women's time to foraging and productive activities that are compatible with child care and away from activities that are dangerous to them and their children (Brown, 1970; Hurtado, 1992).

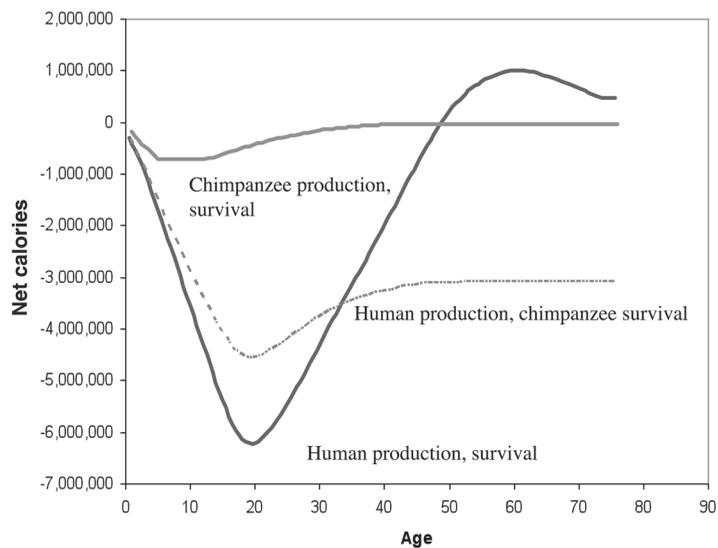
On average among adults in the 10-group sample, men acquired 68% of the calories and almost 88% of the protein; women acquired the remaining 32% of calories and 12% of protein. Given that, on average, these calories are distributed to support adult female consumption (31%), adult male consumption (39%), and offspring (31%), respectively, women supply 3% of the calories to offspring, and men provide the remaining 97%. Men supply not only all the protein and fat to offspring but also the bulk of the protein and fat consumed by women. This contrasts sharply with most mammalian species (>97%), where the female supports all the energetic needs of the offspring until it begins eating solid foods (Clutton-Brock, 1991) and males provide little or no investment. It is the high productivity of men that has probably allowed for the evolution of physiological adaptations among women, such as fat storage at puberty and again during pregnancy, which is not found in apes.

Strikingly, the survival curves in Figure 3.2 depicted with dashed lines and scaled on the left vertical axis show that only about 30% of chimpanzees ever born reach 20, the age when humans produce as much as they consume, and that less than 5% ever born reach 45, when human net production peaks. The relationship between survival rates and age profiles of production is made even clearer in Figure 3.3. This panel 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 cumulating over all ages up to the present age. The thin and bold lines show cumulative productivity by age for chimpanzees and humans, respectively. The long human training period is evident when the troughs in the human and chimpanzee curves are compared. The dashed line is a hypothetical cross of human production profiles with chimpanzee survival rates. It shows that the human production profile would not be viable with

3.3

Cumulative expected net caloric production by age: Humans and chimpanzees.

Adapted from Kaplan et al. (2000).



chimpanzee survival rates because expected lifetime net production would be negative. The next section examines human longevity in greater detail.

Traditional Human Life History and Demography

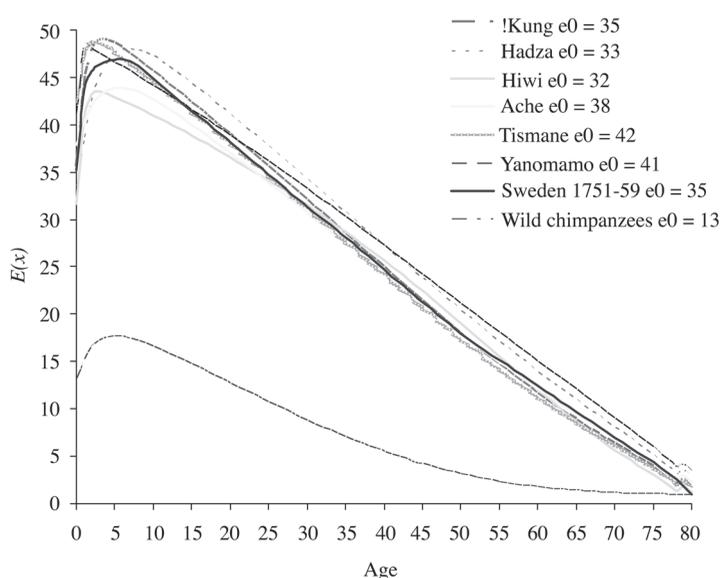
Figure 3.4 shows expected future years of life remaining (e_x), conditional on living to each age for the human groups with the most reliable data and for wild and captive chimpanzees. While there is significant variation across groups in life expectancy at early ages, there is significant convergence after about age 30. With the exception of the Hiwi, who show more than 10 years less remaining during early ages and more than 5 years less remaining during adulthood, and of the Hadza, whose life expectancy at each age is about 2 years longer than the rest at most adult ages, all other groups, including 18th-century Sweden, are hardly distinguishable. This figure also shows that at age 40, the expected age at death is about 63 to 66 (i.e., 23 to 26 additional expected years of life), whereas by age 65, expected age at death is only about 70 to 76 years of age. By that age, death rates become very high.

In contrast, chimpanzees show a very different life course, with higher mortality and lower age-specific survival, especially during adulthood. Even placing chimpanzees in protected environments and modern medical care, which greatly reduces juvenile mortality, does not achieve the longevity experienced by traditional humans without medical care. Captivity raises infant and juvenile survival greatly, from 37% surviving to age 15 to 64%, similar to the human averages. However, while the probability of reaching 45 increases 10-fold from 3% in the wild to 20% with captivity, it is still just half as high as humans living in premodern conditions. The difference between chimpanzees and humans after age 45 is even greater, with an expected future life span of chimpanzees in captivity of only 7 years, about a third of the human expectation. It appears

3.4

Age-specific life expectancy among foragers and chimpanzees.

Expected number of years remaining for six sample populations with sufficient data quality and Sweden, 1751–1759. Curves are based on life table estimates using the Siler model. Adapted from Gurven and Kaplan (2007).



that chimpanzees simply age much faster than humans and die earlier, even in protected environments.

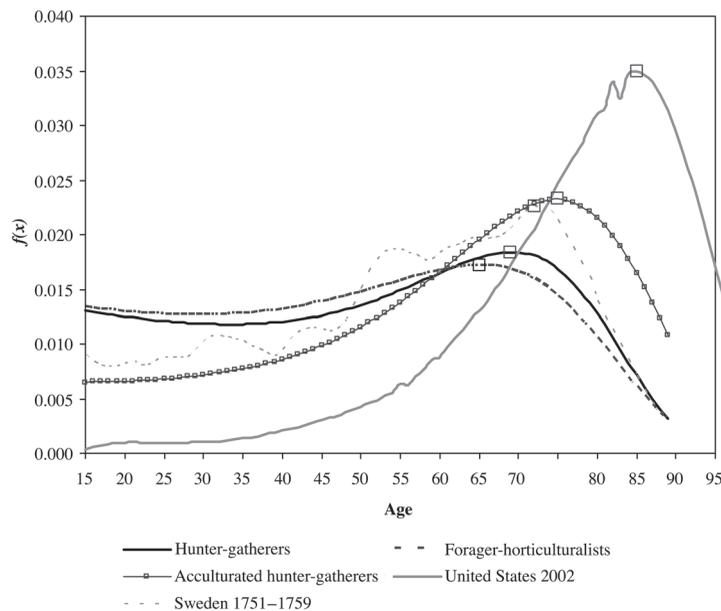
Human adult mortality rates in traditional groups also do not rise at a constant proportional rate. Senescence is usually defined as an increase in the endogenous rate of mortality (Finch, Pike, & Whitten, 1990; Rose, 1991). It has been reported that in many populations, mortality reaches its minimum at reproductive maturity and then increases thereafter at a constant proportional (Gompertz) rate, although noticeable decreases in vital functions do not occur until at least age 30 (Shock, 1981; Weale, 2004).

For most traditional human groups, however, we find strong evidence of departure from linearity. The slope of mortality increase is greater after age 40 than before age 40 (Gurven & Kaplan, 2007). From age 15 until about 35, we see virtually no change in mortality rates with age. This is a period of prime adulthood. However, after age 40, mortality rates rise steadily with age. This delay in senescent decline may play an important role in the greater longevity of our species.

The effective end of the human life span under traditional conditions seems to be just after 70 years of age. Following the lead of Kannisto (2001) and Lexis (1878), we examine the modal ages of "normal" adult death and the variance around these modes to examine the extent of stability in adult life spans among and within our study populations (see Figure 3.5 and Table 3.1). Figure 3.5

3.5

Modal ages of adult death. Adapted from Gurven and Kaplan (2007).



Frequency distribution of ages at death $f(x)$ for individuals over age 15 show strong peaks for hunter-gatherers, forager-horticulturalists, and acculturated hunter-gatherers, Sweden 1751-1759, and the United States, 2002. All curves except for the United States are smoothed by using Siler estimates. Adapted from Gurven and Kaplan (2007).

3.1 Modal Ages of Adult Death^a

Population	Modal age at death	Standard deviation	% of adult deaths at mode	% adult deaths at and above mode
Hadza	76	6.0	2.5	24.1
Hiwi	68	3.3	3.3	17.9
Ache	71	7.7	2.1	24.5
Yanomamo Xilixana	75	7.3	1.9	22.8
Tsimane	78	5.9	3.0	30.5
!Kung, 1963–1974	74	7.8	2.7	35.4
Ache reservation	78	5.9	3.0	30.5
Aborigines	74	7.8	2.7	35.4
Wild chimpanzees	15	16.8	4.6	100.0
Captive chimpanzees	42	7.5	2.6	38.5
Sweden, 1751–1759	72	7.4	2.3	24.3
United States, 2002	85	1.7	3.5	35.3

^a The extent of variation around the mode is usually defined as four standard deviation units around the mode (Cheung et al., 2005). Adapted from Gurven and Kaplan (2007).

shows the frequency distribution, $f(x)$, of deaths at age x , conditional on surviving to age 15, for our composite categories of hunter-gatherer, forager-horticulturalists, and acculturated hunter-gatherer samples using all populations with high data quality and age specificity. All curves (except Sweden and the United States) are based on the Siler models. Data from prehistoric Sweden and the modern United States are shown for comparative purposes. This sample of pre-modern populations show an average modal adult life spans of about 72 years of age (range: 68 to 78; Table 3.1).

While modal age at death is not the same as the effective end of the life span because it refers to a peak in the population distribution of adult deaths, it may reflect an important stage in physiological decline. While there is significant individual variation in rates of aging, the modal age at death may be the age at which most people experience sufficient decline that if they do not die from one cause, they are soon to die from another. This is consistent with our anecdotal impressions of frailty and work in foraging societies. While many individuals remain healthy and vigorous workers through their 60s, few are in good health and capable of significant work in their 70s, and it is the rare individual who survives to age 80.

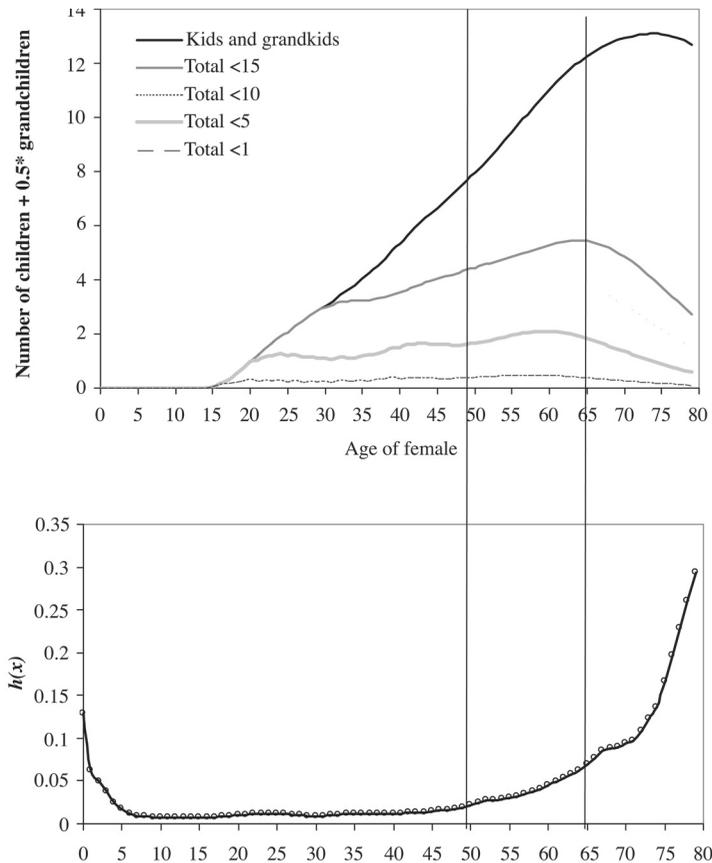
The Evolved Human Life Span

A fundamental conclusion to be drawn from this analysis is that extensive longevity appears to be a novel feature of *Homo sapiens*. The demographic data derived from foragers and forager-horticulturalists allow us to assess how those benefits can change with age. The two panels in Figure 3.6, derived from data on Tsimane forager-horticulturalists, compare age-specific numbers of dependent descendants with mortality rates. The top panel shows the weighted sum of children and one-half the number of grandchildren by age (since grandchildren share, on average, a quarter of their genes with grandparents, whereas parents share half with their own children). The bottom panel shows age-specific mortality rates. Even though a woman still has descendants who could benefit from assistance, the number of offspring and grandoffspring, especially dependents less than 18 years old, drops considerably after about 65 years. This is the point when mortality begins to rise precipitously. The late age decline in dependents is similar to the modal age at death from Figure 3.4. We have tabulated actual flows of food and observed that men and women invest in children and grandchildren after reproduction has ceased, with a shifting emphasis from mostly children to mostly grandchildren as they age.

3.6

Age-specific dependency and adult mortality.

Number of children and one-half the number of grandchildren by age of a Tsimane woman (top panel) compared against age-specific mortality rate for Tsimane (bottom panel). Adapted from Gurven and Kaplan (2007).



As the number of closely related dependent kin eligible to receive investment decreases after age 65, the fitness benefits of longer life decrease, and there is less evolutionary incentive to pay increasing maintenance and repair costs to remain alive and functional beyond this period. Similar results are obtained when the same exercise is done with other populations. Data on males would also show a similar pattern, except that the male peak is 3 to 5 years later because of their later age at marriage. This is potentially why few people lived beyond the seventh decade of life.

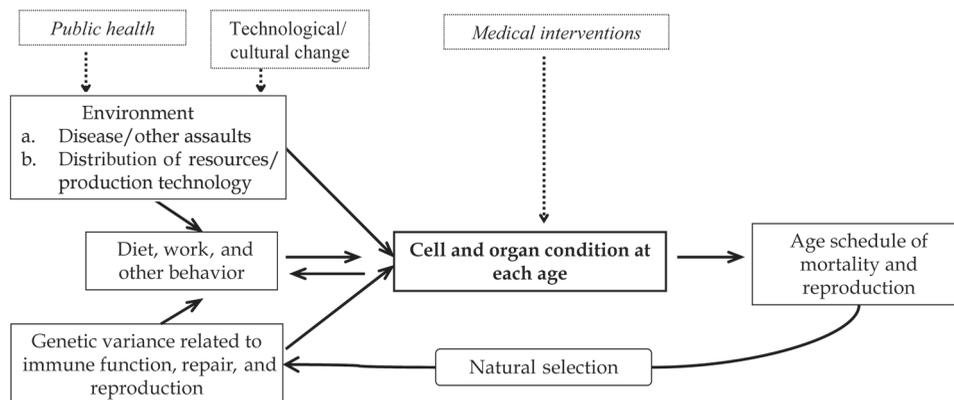
Conclusions: The Present and the Future

In response to modern conditions, the same genome results in a different life history profile. We began this chapter by considering the debate on the future of human longevity. While average life expectancy has changed significantly over recent history, it is an open question whether gains will continue linearly and whether maximum life span itself will still increase (Vaupel, 1997; Wilmoth, 1997). The model we propose in Figure 3.1 can be expanded to shed light on this issue. In Figure 3.7, we add changed features to human environments to the model. **[AuQ2]**

If we imagine the environments in which our ancestors evolved, environmental assaults and access to energy to combat those assaults are likely to have varied across time and locale. Such variation is likely to select for some phenotypic plasticity in allocations to defense and repair. At the same time, the hunting-and-gathering adaptation practiced by evolving humans was built on a complex of long-term child dependence during which learning trumps productivity and the extremely high productivity of adults, especially in middle age. Together, the costs of slowing senescence and mortality prevention and the benefits of extended investment in descendants produced selection for a

3.7

Modernization effects on life span.



characteristic human life span, with some variance around the central tendency. The comparison of the data from 18th-century Sweden to the hunting-and-gathering populations suggests that relatively similar age distributions of adult deaths occur under a relatively broad range of environmental conditions.

The reductions in infectious diseases and improvements in food supply dramatically lower the assault rate on people's bodies as modernization occurs. Aging individuals are increasingly insulated from assaults as well. The same set of defenses that evolved to be phenotypically plastic (at least to some degree) in relation to ancestral environmental variation produces a very different distribution of deaths under modern conditions. In that sense, when one considers the evolved human life span, it is perhaps best conceived as a population-level distribution of deaths that corresponds to the characteristic range of environments in which our ancestors lived.

In this light, it is likely that neither of the two extreme views (a fixed upper limit to human life span or unlimited flexibility in relation to environmental change) is correct. However, we suspect that environmental change will ultimately have decelerating effects on life expectancy improvement. With relatively constant selection over much of human history, the different somatic subsystems of an organism should tend toward a shared rate of senescence (i.e., stochastically at the population level). In response to a radically changed environment, however, such coordination is not necessarily to be expected, and there may be much carryover from the selective environment, thereby limiting life span extension. The chimpanzee–human comparison also suggests that species differences tend to overwhelm differences in environmental conditions in determining mortality hazards as individuals age. This implies that some differences in our respective genomes have resulted in basic differences in rates of repair and tissue maintenance that manifest themselves in physiological deterioration at older ages.

On the other hand, we differ from those who expect few gains in life expectancy because our view suggests that a reduction in the assault rate, coupled with medical interventions and increases in energy balance, should increase longevity (Abrams, 1993). In addition to lowered assault rates slowing physiological damage, we can expect on theoretical grounds that the evolved human repair and defense system will respond to improved environmental conditions, leading to greater longevity.

We do not yet understand the mechanisms underlying the effects of modernization. Do members of industrialized countries senesce more slowly, in a physiological sense, than people exposed to higher assault environments? Alternatively, are most of the mortality improvements due to reductions in cause-specific mortality at specific ages through prevention of assaults or medical treatment of illnesses? Is a 50-year-old Hadza as robust and functional as a 50-year-old American? It has been argued that aging and the onset of chronic disease is accelerated in response to poor nutrition, infectious disease, and chronic inflammatory processes in general (Bengtsson & Lindstrom, 2000; Blackwell, Hayward, & Crimmins, 2001; Elo & Preston, 1992). For example, there is increasing evidence that chronic diseases, such as diabetes, occurred at earlier ages in the 19th century in the United States than occur today (Fogel & Costa, 1997). In contrast to the United States, the Tsimane show higher levels of C-reactive protein across all ages (Gurven, Kaplan, Crimmins, Finch, & Winking, 2008). This protein is an

acute-phase marker that promotes inflammation and among Tsimane associates with disease load and presence of parasites. There is also increasing evidence that malnutrition and health insults during fetal and perinatal development produce a set of cascading effects leading to a greater risk of coronary heart disease later in life (Barker & Osmond, 1986; Cameron & Demerath, 2002). Together, these results suggest that aging and old-age mortality are modulated through energy allocation decisions made early in life in a particular disease ecology. Nevertheless, definitive answers to these questions await further research.

References

- Abrams, P. A. (1993). Does increased mortality favor the evolution of more rapid senescence? *Evolution*, *47*, 877–887.
- Aiello, L., & Wheeler, P. (1995). The expensive-tissue hypothesis: The brain and the digestive system in human and primate evolution. *Current Anthropology*, *36*, 199–221.
- Allman, J., McLaughlin, T., & Hakeem, A. (1993). Brain weight and life-span in primate species. *Proceedings of the National Academy of Sciences of the United States of America*, *90*(1), 118–122.
- Armstrong, E. (1982). A look at relative brain size in mammals. *Neuroscience Letters*, *34*, 101–104.
- Arrow, K. J., & Kurz, M. (1970). *Public investment, the rate of return, and optimal fiscal policy*. Baltimore: Johns Hopkins University Press.
- Barker, D. J. P., & Osmond, C. (1986). Infant mortality, childhood nutrition, and ischaemic heart disease in England and Wales 1986. *Lancet*, *i*, 1077–1081. **[AuQ3]**
- Becker, G. S. (1975). *Human capital*. New York: Columbia University Press.
- Bengtsson, T., & Lindstrom, M. (2000). Childhood misery and disease in later life: The effects on mortality in old age of hazards experienced in early life, southern Sweden, 1760–1894. *Population Studies*, *54*, 263–277.
- Black, F. L. (1975). Infectious disease in primitive societies. *Science*, *187*, 515–518.
- Black, F. L., Woodall, J. P., Evans, A. S., Liebhaver, H., & Henle, G. (1970). Prevalence of antibody against viruses in the Tiriyo, an isolated Amazonian tribe. *American Journal of Epidemiology*, *91*, 430–438.
- Blackwell, D. L., Hayward, M. D., & Crimmins, E. M. (2001). Does childhood health affect chronic morbidity in later life? *Social Science & Medicine*, *52*, 1269–1284.
- Blurton Jones, N. G., Hawkes, K., & Draper, P. (1994). Differences between Hadza and !Kung children's work: Original affluence or practical reason? In E. S. Burch & L. Ellana (Eds.), *Key issues in hunter gatherer research* (pp. 189–215). Oxford: Berg.
- Blurton Jones, N. G., Hawkes, K., & O'Connell, J. (1989). Modeling and measuring the costs of children in two foraging societies. In V. Standen & R. A. Foley (Eds.), *Comparative socioecology of humans and other mammals* (pp. 367–390). London: Basil Blackwell.
- Blurton Jones, N. G., Hawkes, K., & O'Connell, J. (1997). Why do Hadza children forage? In N. L. Segal, G. E. Weisfeld, & C. C. Weisfield (Eds.), *Uniting psychology and biology: Integrative perspectives on human development* (pp. 297–331). New York: American Psychological Association.
- Bock, J. (1995). *The determinants of variation in children's activities in a Southern African community*. Unpublished manuscript.
- Boesch, C., & Boesch, H. (1999). *The chimpanzees of the Tai Forest: Behavioural ecology and evolution*. Oxford: Oxford University Press.
- Buikstra, J. E. (Ed.). (1981). *Prehistoric tuberculosis in the Americas*. Evanston, IL: Northwestern University Archaeological Program.
- Bunn, H. T. (2001). Hunting, power scavenging, and butchering by Hadza foragers and by Plio-Pleistocene *Homo*. In C. B. Stanford & H. T. Bunn (Eds.), *Meat-eating and human evolution* (pp. 199–218). Oxford: Oxford University Press.
- Cameron, N., & Demerath, E. W. (2002). Critical periods in human growth and their relationship to diseases of aging. *American Journal of Physical Anthropology*, *35*(Suppl.), 159–184.
- Carey, J. R. (2001). Insect biodemography. *Annual Review of Entomology*, *46*, 79–110.

- Charnov, E., & Berrigan, D. (1993). Why do female primates have such long lifespans and so few babies? *Evolutionary Anthropology*, *1*, 191–194.
- Cheung, S. L. K., Robine, J.-M., Robine, J. M., Jow-Ching, E., & Caselli, G. (2005). Three dimensions of the survival curve: Horizontalization, verticalization and longevity extension. *Demography*, *42*, 243–258.
- Clark, G. A., Kelley, M. A., Grange, J. M., & Hill, M. C. (1987). The evolution of mycobacterial disease in human populations: A reevaluation. *Current Anthropology*, *28*, 45–62.
- Clutton-Brock, T. H. (1991). *The evolution of parental care*. Princeton, NJ: Princeton University Press.
- Coimbra, C. E. A., Jr. (1988). Human settlements, demographic pattern, and epidemiology in lowland Amazonia: The case of Chagas' disease. *American Anthropologist*, *90*, 82–97.
- Cordain, L., Brand Miller, J., Eaton, B., Mann, N., Holt, S. H. A., & Speth, J. D. (2000). Plant-animal subsistence ratios and macronutrient energy estimations in hunter-gatherer diets. *American Journal of Clinical Nutrition*, *71*, 682–692.
- Crimmins, E., Alley, D., Reynolds, S., Johnston, M., Karlamangla, A., & Seeman, T. (2005). Changes in biological markers of health: Older Americans in the 1990s. *Journal of Gerontology: Medical Sciences*, *60*, 1409–1413.
- Donovan, B. (2000). The repertoire of human efforts to avoid sexually transmissible diseases: Past and present. Part 1. Strategies used before or instead of sex. *Sexually Transmitted Infections*, *76*, 88–93.
- Dunn, F. L. (1968). Epidemiological factors: Health and disease in hunter-gatherers. In R. B. Lee & I. DeVore (Eds.), *Man the hunter* (pp. 221–228). Chicago: Aldine.
- Elo, I. T., & Preston, S. H. (1992). Effects of early-life conditions on adult mortality: A Review. *Population Index*, *58*, 186–212.
- Fiennes, R. (1978). *Zoonoses and the origins and ecology of human disease*. London: Academic Press.
- Finch, C. E., Pike, M. C., & Whitten, M. (1990). Slow mortality rate accelerations during aging in animals approximate that of humans. *Science*, *249*, 902–905.
- Fleagle, J. G. (1999). *Primate adaptation and evolution*. New York: Academic Press.
- Fogel, R. W., & Costa, D. L. (1997). A theory of technophysio evolution, with some implications for forecasting population, health care costs and pension costs. *Demography*, *34*, 49–66.
- Fries, J. F. (1989). The compression of morbidity: Near or far? *Milbank Quarterly*, *67*, 208–232.
- Gadgil, M., & Bossert, W. H. (1970). Life historical consequences of natural selection. *American Naturalist*, *104*, 1–24.
- Gurven, M., Hill, K., Kaplan, H., & Hurtado, A. M. (2000). Food transfers among Hiwi foragers of Venezuela: Tests of reciprocity. *Human Ecology*, *28*, 171–218.
- Gurven, M., & Kaplan, H. (2006). Determinants of time allocation to production across the lifespan among the Machiguenga and Piro Indians of Peru. *Human Nature*, *17*(1), 1–49.
- Gurven, M., & Kaplan, H. (2007). Longevity among hunter-gatherers: A cross-cultural comparison. *Population and Development Review*, *33*, 321–365.
- Gurven, M., Kaplan, H., Crimmins, E., Finch, C., & Winking, J. (2008). Lifetime inflammation in two epidemiological worlds: The Tsimane of Bolivia and the United States. *Journal of Gerontology: Biological Sciences*, *63A*(2), 196–199.
- Gurven, M., Kaplan, H., & Gutierrez, M. (2006). How long does it take to become a proficient hunter? Implications for the evolution of delayed growth. *Journal of Human Evolution*, *51*, 454–470.
- Gurven, M., & Walker, R. (2006). Energetic demand of multiple dependents and the evolution of slow human growth. *Proceedings of the Royal Society of London, Series B: Biological Sciences*, *273*, 835–841.
- Hakeem, A., Sandoval, G. R., Jones, M., & Allman, J. (1996). Brain and life span in primates. In R. P. Abeles, M. Catz, & T. T. Salthouse (Eds.), *Handbook of the psychology of aging* (pp. 78–104). San Diego: Academic Press.
- Hamilton, W. D. (1966). The molding of senescence by natural selection. *Journal of Theoretical Biology*, *12*, 12–45.
- Hayflick, L. (2007). Biological aging is no longer an unsolved problem. *Annals of the New York Academy of Sciences*, *1100*, 1–13.
- Hill, K. (1988). Macronutrient modifications of optimal foraging theory: An approach using indifference curves applied to some modern foragers. *Human Ecology*, *16*, 157–197.

- Hiraiwa-Hasegawa, M. (1990). The role of food sharing between mother and infant in the ontogeny of feeding behavior. In T. Nishida (Ed.), *The chimpanzees of the Mahale Mountains: Sexual and life history strategies* (pp. 267–276). Tokyo: Tokyo University Press.
- Holliday, M. A. (1978). Body composition and energy needs during growth. In F. Falcker & J. M. Tanner (Eds.), *Human growth* (Vol. 2, pp. 117–139). New York, Plenum Press.
- Hossain, P., Kavar, B., & El Nahas, M. (2007). Obesity and diabetes in the developing world—A growing challenge. *New England Journal of Medicine*, *356*, 213–215.
- Intriligator, M. D. (1971). *Mathematical optimization and economic theory*. Englewood Cliffs, NJ: Prentice Hall.
- Jerison, H. (1973). *Evolution of the brain and intelligence*. New York: Academic Press.
- Jerison, H. J. (1976). Paleoneurology and the evolution of mind. *Scientific American*, *234*, 90–101.
- Judge, D. S., & Carey, J. R. (2000). Postreproductive life predicted by primate patterns. *Journal of Gerontology: Biological Sciences*, *55*, B201–B209.
- Kannisto, V. (2001). Mode and dispersion of the length of life. *Population: An English Selection*, *13*, 159–171.
- Kaplan, H. (1997). The evolution of the human life course. In K. Wachter & C. Finch (Eds.), *Between Zeus and Salmon: The biodemography of aging* (pp. 175–211). Washington, DC: National Academies Press.
- Kaplan, H., & Gurven, M. (2005). The natural history of human food sharing and cooperation: A review and a new multi-individual approach to the negotiation of norms. In H. Gintis, S. Bowles, R. Boyd, & E. Fehr (Eds.), *Moral sentiments and material interests: The foundations of cooperation in economic life* (pp. 75–113). MIT Press.
- Kaplan, H., Hill, K., Lancaster, J., & Hurtado, A. M. (2000). A theory of human life history evolution: Diet, intelligence, and longevity. *Evolutionary Anthropology*, *9*, 156–185.
- Kaplan, H., Hill, K. R., Lancaster, J. B., & Hurtado, A. M. (2001). The embodied capital theory of human evolution. In P. T. Ellison (Ed), *Reproductive ecology and human evolution* (pp. XX–XX). Hawthorne, NY: Aldine de Gruyter.
- Kaplan, H., Mueller, T., Gangestad, S. & Lancaster, J. (2003). Neural capital and lifespan evolution among primates and humans. In C. E. Finch, J.-M. Robine & Y. Christen (Eds.), *The brain and longevity* (pp. 69–98). Berlin: Springer Verlag.
- Kaplan, H. S., & Robson, A. (2002). The emergence of humans: The coevolution of intelligence and longevity with intergenerational transfers. *Proceedings of the National Academy of Sciences of the United States of America*, *99*, 10221–10226.
- Kaplan, H., & Robson, A. (n.d.). *Why aging is optimal: Senescence and size-dependent costs of somatic repair*. Unpublished manuscript. Department of Anthropology, University of New Mexico.
- Kozlowski, J., & Wiegert, R. G. (1986). Optimal allocation to growth and reproduction. *Theoretical Population*, *29*, 16–37.
- Lack, D. (1954). *The natural regulation of animal numbers*. Oxford: Oxford University Press.
- Lexis, W. (1878). Sur la Durée Normale de la Vie Humaine sur la Théorie de la Stabilité des Rapports Statistiques. *Annales de Démographie Internationale*, *2*, 447–460.
- Lloyd, D. G. (1987). Selection of offspring size at independence and other size-versus-number strategies. *American Naturalist*, *129*, 800–817.
- McNeil, W. H. (1989). *Plagues and peoples* (2nd ed.). New York: Anchor Books.
- Mincer, J. (1974). *Schooling, experience, and earnings*. Chicago: National Bureau of Economic Research.
- Murdock, G. P. (1967). Ethnographic Atlas: A summary. *Ethnology*, *6*, 109–236.
- Nunn, C. L., Altizer, S., Sechrester, W., Jones, K., Barton, R., & Gittleman, J. (2004). Parasites and the evolutionary diversity of primate clades. *American Naturalist*, *164*(Suppl.), S90–S103.
- Parker, S. T., & McKinney, M. L. (1999). *Origins of intelligence: The evolution of cognitive development in monkeys, apes and humans*. Baltimore: Johns Hopkins University Press.
- Pearce-Duvet, J. M. C. (2006). The origin of human pathogens: Evaluating the role of agriculture and domestic animals in the evolution of human disease. *Biological Reviews*, *81*, 369–382.
- Promislow, D. E. L. (1991). Senescence in natural populations of mammals: A comparative study. *Evolution*, *45*, 1869–1887.
- Richards, M. P., & Hedges, R. M. (2000). Focus: Gough's Cave and Sun Hole Cave human stable isotope values indicated a high animal protein diet in the British Upper Paleolithic. *Journal of Archeological Science*, *27*, 1–3.

[AuQ4]

[AuQ5]

- Robson, A., & Kaplan, H. (2003). The evolution of human life expectancy and intelligence in hunter-gatherer economies. *American Economic Review*, *93*(1), 150–169.
- Rose, M. R. (1991). *Evolutionary biology of aging*. New York: Oxford University Press.
- Sacher, G. A. (1959). Relation of lifespan to brain weight and body weight in mammals. In G. E. W. Wolstenhome & M. O'Connor (Eds.), *Ciba Foundation Colloquia on Ageing* (pp. 115–133). London: Churchill.
- Salzano, F. M., & Callegari-Jacques, S. (1988). *South American Indians: A case study in evolution*. (Research monographs on human population biology, No. 6.). Oxford: Oxford University Press.
- Schoeninger, M., Bunn, H. T., Murray, S., Pickering, T., & Moore, J. (2001). Meat-eating by the fourth African ape. In C. B. Stanford & H. T. Bunn (Eds.), *Meat-eating and human evolution* (pp. 179–195). Oxford: Oxford University Press.
- Seidell, J. C. (2000). Obesity, insulin resistance and diabetes—A worldwide epidemic. *British Journal of Nutrition*, *83*(Suppl. 1), S5–S8.
- Shanley, D. P., & Kirkwood, T. B. L. (2000). Calorie restriction and aging: A life-history analysis. *Evolution*, *54*, 740–750.
- Shock, N. W. (1981). Indices of functional age. In D. Danon, N. W. Shock, & M. Marois (Eds.), *Aging: A challenge to science and society* (pp. 302–319). Oxford: Oxford University Press.
- Silk, J. B. (1978). Patterns of food-sharing among mother and infant chimpanzees at Gombe National Park, Tanzania. *Folia Primatologica*, *29*, 129–141.
- Smith, C. C., & Fretwell, S. D. (1974). The optimal balance between size and number of offspring. *American Naturalist*, *108*, 499–506.
- Stanford, C. B. (1998). *Chimpanzee and red colobus: The ecology of predator and prey*. Cambridge, MA: Harvard University Press.
- Teleki, G. (1973). *The predatory behavior of wild chimpanzees*. Lewisburg, PA: Bucknell University Press.
- Vaupel, J. W. (1997). The remarkable improvements in survival at older ages. *Philosophical Transactions of the Royal Society of London. Series B, Biological Sciences*, 1799–1804.
- Weale, R. A. (2004). Biorepair mechanisms and longevity. *Journal of Gerontology: Biological Sciences*, *59*, 449–454.
- Wrangham, R. W., Jones, J. H., Laden, G., Pilbeam, D., & Conklin-Brittain, N. (1999). The raw and the stolen. *Current Anthropology*, *40*, 567–594.

- [AuQ1]** Please add Finch & Stanford, 2004, to the reference list or delete this citation.
- [AuQ2]** Please add Wilmoth, 1997, to the reference list or delete this citation.
- [AuQ3]** Barker & Osmond (1986): please supply volume number in place of “i”
- [AuQ4]** Kaplan & Gurven (2005): please provide location (city, state) of publisher.
- [AuQ5]** In Kaplan, H. Hill, K. R., Lancaster, J. B., & Hurtado, A. M. (2001), please provide the page range of the chapter.