5

Mortality, Senescence, and Life Span

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Little Mama," estimated to be over seventy years old, is one of the oldest chimpanzees ever recorded. Born in Africa, she is now a retiree living in a Florida theme park (Figure 5.1, left). She is a rare exception, petite and healthy after receiving excellent care in the pet industry (Segal 2012). Madame Jeanne Calment, a French supercentenarian who died at the ripe age of 122, was the oldest human ever recorded (Figure 5.1). She led a leisured life, was still riding her bicycle up until age 100, smoked cigarettes since age twenty-one, and had a good sense of humor ("I've never had but one wrinkle, and I'm sitting on it"). Although these two are hardly representative of their respective species, the gap in life span of a half century speaks to real biological differences in life span potential (Table 5.1).

The maximum human life expectancy has risen steadily by more than two years every decade over the past two centuries, a dramatic improvement that suggests new answers to old questions about species differences in programmed senescence and the existence of biologically determined maximal life-spans (Wachter and Finch 1997; Austad 1999; Oeppen and Vaupel 2003; Burger et al. 2012). Although much of the increase in life expectancy in the nineteenth century can be attributed to better sanitation,

FIGURE 5.1. Oldest recorded chimpanzee and human. (a) "Little Mama" at Florida's Lion Country Safari park is believed to be the oldest chimpanzee in captivity, between 60 and 74 years old. She has lived there since 1967. Photo courtesy of Andrew Halloran. (b) Jean Calment lived to 122 years. Here she celebrates her 121st birthday in 1996. Photo source: http://en.wikipedia.org/wiki/File:Jeanne-Calment-1996.jpg.

modern medicine, and improved diets (Riley 2001), there is strong evidence that the general pattern of a long life span is not unique to modern populations or even horticulturalists (cf. Lovejoy 1981; Washburn 1981), and that current increases in life span may be a consequence of plasticity in our evolved human life history. There is little support for Vallois's (1961: 222) infamous claim that among early humans, "few individuals passed forty years, and it is only quite exceptionally that any passed fifty," or the Hobbesian view of a "nasty, brutish and short" human life span (see also King and Jukes 1969; Weiss 1981).

Chimpanzees are the closest living genetic relatives to humans, and are likely to have a life history similar to our last common ancestor from 7–10 million years ago (Wrangham 1987, 2001). Compared to humans, our chimpanzee cousins have shorter lives, smaller brains, and bodies that grow and develop more rapidly (Isler and van Schaik 2009). Chimpanzee fertility is also lower than among human hunter-gatherers, owing primarily to longer intervals between births (Table 5.1). Closer attention to chimpanzee demography, diet, health, physiology, economics, and social behavior is therefore critical for understanding the evolution of human longevity and related life history traits. To date, however, there have been few comprehensive reviews of human hunter-gatherer life spans and survivorship that include detailed comparisons with chimpanzees.

TABLE 5.1. Comparison of life history traits among traditional humans living in natural fertility conditions and wild chimpanzees. Adapted from Gurven (2012).

Trait	*Definition*	*Units*	*Humans*	*Chimpanzees*	*Percent Increase*
Brain volume	Volume of the brain	cm^3	1,330[a,b]	330–430[a,b,c]	242%
Encephalization quotient (EQ)	Ratio of actual to predicted brain size (based on body size allometry)		7.4–7.8[c]	~2.2–2.5[c]	223%
Juvenile period	Weaning to menarche	years	12.9[d]	5[d]	158%
Adult life span	Life expectancy at age 15	years	37.7[e]	14[e]	148%
Maximum life span	Oldest observed individual	years	121[f]	54, 66[a,f,j]	83%
Fertility rate	Inverse of interbirth interval	#/yr	0.29[g]	0.21[h]	38%
Infant mortality rate	Probability of dying in first year	percent	23[e]	20[l]	15%
Juvenile survival	Probability of living to age 15	percent	57[e]	36[e]	58%
Interbirth interval	Time between successive births	months	41.3[g]	68.9[h,i]	−40%
Extrinsic mortality rate	Young adult mortality rate	percent/yr	1.1[e]	3.7[e]	−70%
Neonate mass	Mass at birth	kg	2.8[a,m]	1.7[a,m]	65%
Age at menarche	Birth to menstruation	years	15[d]	10[d]	50%
Age at first reproduction	Birth to reproduction	years	19.1[d,g]	12.8[k]	49%
Age at last reproduction	Birth to last reproduction	years	39[d,g]	27.7[k]	41%
Adolescence	Menarche to first reproduction	years	4.1[d]	3[d]	37%
Fetal growth rate	Growth rate: conception to birth	g/day	10.4[o]	7.6[o]	37%
Total fertility rate	Total number of live births	live births	6.1[g]	5[g,n]	22%
Gestation length	Conception to birth	days	269[a,p]	225[a,j]	20%
Body size	Average adult female mass	kg	47[d]	35[d]	34%

a. Not specific to natural fertility or wild populations; b. Schoenemann (2006); c. Roth and Dicke (2005); d. Walker et al. (2006); e. Gurven and Kaplan (2007); f. Finch (2007); g. Kaplan et al. (2000); h. Emery Thompson et al. (2007); I. Refers to interbirth interval when first infant survives to age four; IBI is 26.6 months otherwise when first infant dies; j. Bronikowski et al. (2011); k. Emery Thompson (2013); l. Hill et al. (2001); m. Lee et al. (1991); n. Based on completed fertility; TFR synthesized from age-specific fertility rates in Emery Thompson et al. (2007: table S1) is 6.9 live births; o. Fetal growth rate=neonate mass/gestation length; p. Wood (1994).

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In this chapter, we first compare mortality rates and life span among humans and chimpanzees in the "wild" using the most complete available data on well-studied populations. Second, we describe the plasticity in life span in both species with improved conditions. Despite some similarities in early life mortality patterns, chimpanzees have shorter potential maximal life spans than humans. Humans everywhere have a substantial postreproductive life span, whereas chimpanzees mostly do not, and some robust females are capable of reproducing until the end of their adult lives. To help contextualize these species differences in survivorship, and presumably in aging, the third section summarizes what is known about causes of death both in the wild and under more modern conditions, in both young and old. We examine species differences in senescence rates, discuss the selection pressures that may have helped shape the distinct life histories of humans and chimpanzees, and evaluate leading hypotheses that might help explain why humans are longer-lived than chimpanzees. We link life history changes with species differences in social behavior, and conclude with suggestions for future directions.

Mortality

Humans

Hunter-gatherers with minimal exposure to modern medicine, and with traditional diets and activity regimes, are an important lens for understanding how selection helped shape the evolution of the human life course. Given that quality demographic data with reasonably accurate age and mortality estimation exist only for a handful of populations (Howell 1979; Blurton Jones et al. 1992; Hill and Hurtado 1996; Early and Headland 1998), we also consider simple horticulturalists without modern amenities as an additional source of data on mortality and senescence in preindustrial societies.

The age-specific probability of survival (l_x) from birth to adulthood shows a modest amount of variation across different populations of human hunter-gatherers, forager horticulturalists, and wild chimpanzees (Figure 5.2). The within-species variation is less marked than the between-species variation, for which humans have a higher probability of survival at all stages of life, with the exception of early infancy. Infant survival rates may be lower in

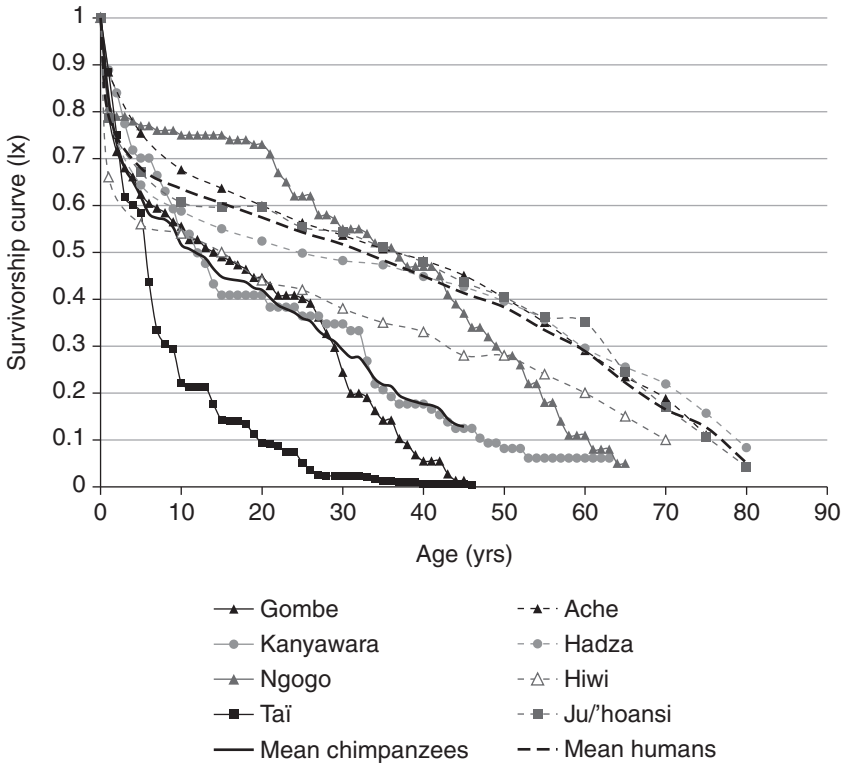

FIGURE 5.2. Survivorship curve (l_x) for four chimpanzee populations (Gombe, Kanyawara, Ngogo, and Taï), four traditional human populations (Aché, Hadza, Hiwi, and Ju/'hoansi), and the mean for chimpanzees (Bossou, Gombe, Kanyawara, Mahale, Ngogo, and Taï) and humans (Aché, Agta, Hadza, Hiwi, Ju/'hoansi, Tsimané, and Yanomamo). Both sexes are combined for each population. Sources: Gombe and Taï (Hill et al. 2001), Kanyawara (Muller and Wrangham 2014), Ngogo (Wood et al. 2017), Aché (Hill and Hurtado 1996), Agta (Early and Headland 1998), Hadza (Blurton Jones et al. 1992), Hiwi (Hill et al. 2007), Ju/'hoansi (Howell 1979), Tsimané (Gurven et al. 2007), and Yanomamo (Early and Peters 2000).

humans than chimpanzees, owing to birth complications or higher vulnerability of altricial neonates. Infant mortality rates among hunter-gatherer populations range from 14 to 40 percent, with a mean ± standard deviation of 27 ± 7 percent dying in the first year of life ($n = 15$) (Volk and Atkinson 2013). On average, 57 percent and 64 percent of children born survive to age fifteen among hunter-gatherers and forager-horticulturalists, respectively. Beyond age fifteen, adult mortality rates range from 1 to 1.5 percent per year until

about age forty, when it increases exponentially. In spite of this variation, there does appear to be a clear premodern human pattern. There is remarkable similarity in age profiles of mortality risk over the life span. By age ten, the mortality hazard has slowed to 0.01, doubled to about 0.02 by age forty, doubled yet again before age sixty, and again by age seventy. Low mortality therefore persists until about age forty, when mortality accelerations become more evident. Overall, the mortality rate after age thirty doubles every seven to ten years (Finch 1994; Gurven and Kaplan 2007).

Data from extant foragers with little to no access to medical attention or modern foods, including the Ju/'hoansi, Aché, and Hadza, show that while at birth mean life expectancies range from thirty to thirty-seven years of life, women who survive to age forty-five can expect to live an additional twenty to twenty-two years (Blurton Jones et al. 2002; Gurven and Kaplan 2007). While there is significant variation across groups in life expectancy at early ages, there is significant convergence after about age thirty. With the exception of the Hiwi, who show over ten years less remaining during early ages and over five years less remaining during adulthood, and of the Hadza, whose life expectancy at each age is about two years longer than the rest at most adult ages, all other groups are hardly distinguishable. At age forty, the expected age at death is about sixty-three to sixty-six (i.e., twenty-three to twenty-six additional expected years of life), whereas by age sixty-five, expected age at death is only about seventy to seventy-six years of age. By that age, death rates become very high.

Human life span (whether measured as maximal life span or life expectancy) is longer than predicted for a typical mammal (or primate) of human body size, but not atypical given the larger than expected brain size of humans (Allman et al. 1993). Estimates based on regressions of various primate subfamilies and extant apes suggests a major increase in longevity between *Homo habilis* (52–56 yrs) to *Homo erectus* (60–63 yrs) occurring 1.7–2 million years ago, and further increases for *Homo sapiens* (66–72 yrs) (Judge and Carey 2000). Extrapolations for early *Homo sapiens* based on comparative analyses including both brain weights and body sizes among nonhuman primates similarly suggest a maximum life span between sixty-six and seventy-eight years (Hammer and Foley 1996). Although maximum life spans can be much larger than life expectancies (the average of which is often lowered due to high infant and child mortality), it is usually reported that Paleolithic humans had life expectancies of only fifteen to twenty years. This

brief life span is believed to have persisted over thousands of generations (Cutler 1975; Weiss 1981) until less than ten thousand years ago, when early agriculture presumably caused a slight increase to about twenty-five years. Gage (2003) compiles over twelve reconstructed prehistoric life tables with similar life expectancies to form a composite life table with survivorship to age fifty (l_{50}) of about 2–9 percent and e_{45} values of about three to seven years.

There is a large paleodemographic literature concerning problematic age estimation in skeletal samples, and bias in bone preservation leading to underrepresentation of older individuals (see Buikstra and Konigsberg 1985; Walker et al. 1988; Buikstra 1997; Hoppa and Vaupel 2002). This literature is too large to discuss here, and we direct readers to recent treatments by O'Connell et al. (1999) and Kennedy (2003). Nonetheless, we point out some observations that further suggest problems with prehistoric life tables. Mortality rates in prehistoric populations are estimated to be lower than those for traditional foragers until about age two years. Estimated mortality rates then increase dramatically for prehistoric populations, so that by age forty-five they are over seven times greater than those for traditional foragers, even worse than the ratio of captive chimpanzees to foragers. Because these prehistoric populations cannot be very different genetically from the populations surveyed here, there must be systematic biases in the samples and / or in the estimation procedures at older ages, where presumably endogenous senescence should dominate as the primary cause of death. While excessive warfare could explain the shape of one or more of the prehistoric forager mortality profiles, it is improbable that these profiles represent the long-term prehistoric forager mortality profile. Such rapid mortality increase late in life would have severe consequences for our human life history evolution, particularly for senescence in humans. It is encouraging that recent treatments of prehistoric life tables show increasing similarity with those presented here based on ethnographic samples (Konigsberg and Herrmann 2006).

It is noteworthy that unlike chimpanzees (below), most hunter-gatherer and small-scale horticultural populations in the ethnographic record show positive population growth, on average 1 percent. Such growth could not have represented conditions over long stretches of our species history: in order to achieve population stationarity (i.e., zero growth), fertility would have to decline well below that ever observed in natural fertility populations (to a total fertility rate of four births per woman) or survivorship would need to decline below that ever observed (to $l_{15} = 0.41$) (Gurven and Kaplan 2007).

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Either current conditions reflected in the demographic data are not representative of the past (i.e., warfare may have been more common, or fertility lower), or population dynamics over short periods of time may be better described as a "saw-tooth" pattern characterized by periods of growth followed by rapid population crashes (Hill and Hurtado 1996). It is difficult to evaluate these two possibilities in light of current knowledge about the past. While evidence suggests that climate varied widely throughout the Pleistocene and into the Holocene Epoch (Richerson et al. 2005), the extent to which past foragers typically experienced increasing, declining, or zero growth in past environments is unknown.

Chimpanzees

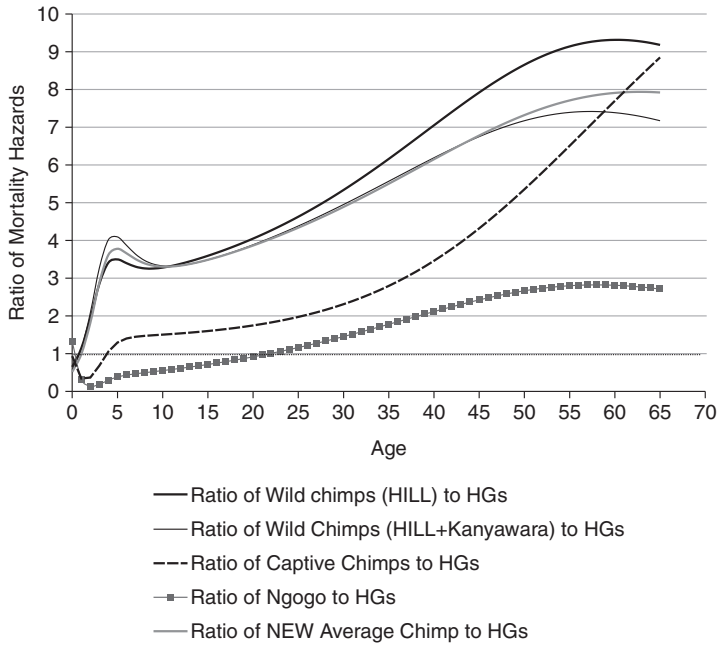
The largest dataset on mortality among free-living chimpanzees was compiled by Hill and colleagues (2001) to form a synthetic life table, based on data obtained from Gombe, Taï, Kanyawara (Kibale), Mahale, and Bossou field sites. We substituted the Kanyawara data in the former sample with an updated sample obtained by Muller and Wrangham (2014), and include a new Ngogo sample by Wood et al. (2017) from another part of the same Kibale forest. This increased the risk set by 93 percent to 7,214 risk years and deaths by 40 percent to 398. It is important to note that most wild chimpanzee populations except for Ngogo have been sampled during periods of stasis or decline, while hunter-gatherer populations are growing on average at about 1 percent (Gurven and Kaplan 2007).

Chimpanzees have a life expectancy of around fifteen years at birth, about half that of humans. Infant mortality ranges from 11 to 28 percent, with a mean ± standard deviation of 17 ± 7 percent in the first year of life, and 39 ± 11 percent until age five. The mortality rate drops to about four between ages ten and fifteen. By age fifteen, the life expectancy is another twenty years, and by age thirty, the mortality rate is about 7 percent, with twelve additional years of life left. Remaining life expectancy if surviving to age forty-five is another six years. Life expectancy at birth is higher for females than males, 14.3 years versus 10.8 years, respectively.

Chimpanzee sites vary in mortality patterns with Taï showing the lowest survivorship and Ngogo showing the highest (9.9 percent average mortality rate per year over ages 10–35 yrs in Taï vs. 1.5 percent in Ngogo). Taï may be the most affected by anthropogenic factors, whereas Kanyawara reaches

FIGURE 5.3. An elderly chimpanzee male (estimated age fifty-six years) from Kanyawara. Photo by Martin N. Muller.

levels of survivorship comparable to humans between zero and fourteen years old. Mortality rates over this early life period are even lower in Ngogo than among humans (Figure 5.4). In Ngogo, life expectancy at birth is almost thirty-three years and infant mortality is 19 percent. In Kanyawara, life expectancy at birth is almost twenty years and infant mortality is 11 percent. These low rates are likely due to the low impact of disease transmission, predation, and habitat loss at both sites (Muller and Wrangham 2014). In addition, the rich resource base at Ngogo (three fruit trees: *Ficus mucuso, Chrysophyllum albidum,* and *Pterygota mildbraedii*) can support large populations (Wood et al. 2017), making it an ideal environment for population growth with abundant foods, little anthropogenic impact, and no natural predators. It is likely, however, that some deaths are missing from Ngogo, as their life table presents the probability of surviving from ages two to fifteen to be 95.8 percent! This reflects only five reported deaths in 1,169 chimp-risk years of observation. Another caveat about the representativeness of Ngogo is that the current age composition of their population does not mirror the shape of the survivorship curve (Martin Muller, pers. comm.).



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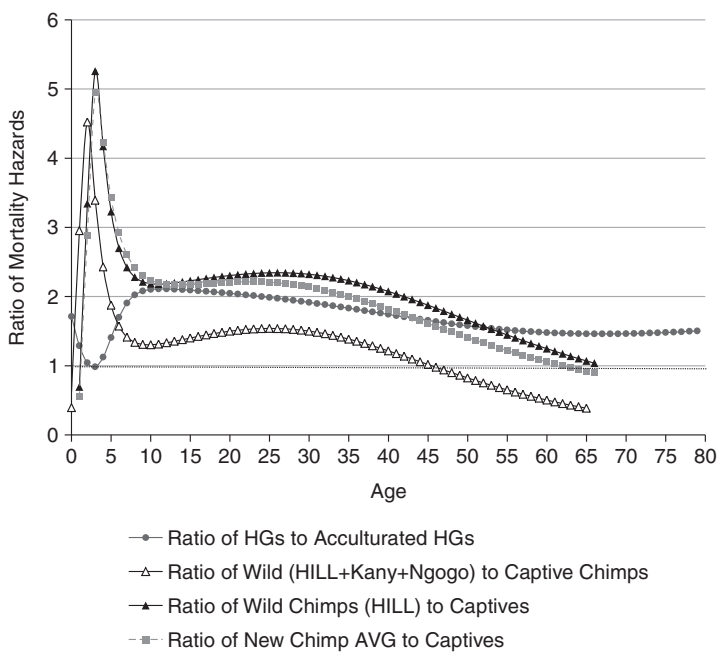

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However, even if the Kanyawara and Ngogo mortality profiles are better representatives of chimpanzee life history in the absence of recent human interactions than the other sites, adult mortality still increases at a substantially higher rate than among humans. For example, remaining life expectancies at ages thirty, forty, and fifty are about ten, seven, and six years, respectively. Mortality rates are also increasing between the ages eleven and thirty-five at Kanyawara, and between ages twenty and thirty-five at Ngogo, the period when mortality rates in hunter-gatherers are flat. Although no life table yet exists for bonobos, one report based on a small sample suggests lower infant and juvenile mortality than among most chimpanzee groups (5 percent die in first year and 27 percent die before age six) (Furuichi et al. 1998). Overall, chimpanzees show a very different life course than human hunter-gatherers, with higher mortality and lower age-specific survival, especially during adulthood.

Plasticity in Captivity and Modern Environments

Improved conditions exhibit similar within-species effects on human and chimpanzee mortality profiles (Figure 5.4). Captive chimpanzees receive medical attention, abundant food, and protection from predation, and show large increases in survival rates (Dyke et al. 1995), though still substantially lower than the Ngogo pattern described above (see Figure 5.4). Infant and juvenile survival improves dramatically, from 35 percent surviving to age fifteen to 64 percent, similar to the human averages. The effects of captivity, however, diminish with age. The probability of reaching forty-five increases

FIGURE 5.4. Ratio of mortality hazards for chimpanzees and hunter-gatherers (HGs), illustrating (a) between-species and (b) within-species differences in age-specific mortality rates. Between-species comparisons include wild and captive chimpanzees versus traditional hunter-gatherers. Additional comparison shown between Ngogo chimpanzees and hunter-gatherers. Within-species comparisons include wild versus captive chimpanzees, and more traditional versus acculturated hunter-gatherers. Source: Wild chimpanzees (Gombe, Taï, Kanyawara, Mahale, Bossou, and Ngogo), Siler estimated based on composite of Hill et al. (2001), Kanyawara from Muller and Wrangham (2014), and Ngogo from Wood et al. (2017); captive chimpanzees (Siler estimated based on Dyke et al, 1995); traditional hunter gatherers (HG, Gurven and Kaplan 2007); acculturated hunter-gatherers (ibid.). Horizontal dotted line where the mortality ratio = 1 denotes equal mortality rates among compared populations.

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from 5 percent in the wild to 20 percent in captivity, and remaining life expectancy at age forty-five is 4.6 and 7.2 years, respectively.

Among humans, the effects of improved conditions also seem to be greatest during childhood and middle adulthood, tapering off with age (Figure 5.4). Comparing mortality rates between hunter-gatherers and modern Americans, infant mortality is over thirty times greater among hunter-gatherers, and early child mortality is over one hundred times greater than encountered in the United States. Not until the late teens does the relationship flatten, with a more than tenfold difference in mortality. This difference is fivefold by age fifty, about fourfold by age sixty, and threefold by age seventy (Burger et al. 2012; Gurven and Kaplan 2007).

While captivity in chimpanzees improves survivorship at all ages, and early in life even matches or exceeds hunter-gatherer levels, differences are quite clear by age thirty-five. The twofold difference in mortality rates between captive chimps and hunter-gatherers accelerates steeply thereafter (Figure 5.4). By age forty-five, the expected future life span of chimpanzees in captivity is a third of the human expectation. Improved conditions for captive chimpanzees, though associated with lower mortality early in life than human hunter-gatherers, does not change the fundamental species differences in mortality rates in adulthood. From the age of lowest mortality rates, chimpanzee mortality rates increase, while human mortality rates remain relatively low and level for two decades before notably increasing. It appears that chimpanzees age much faster than humans and die earlier, even in protected environments.

Causes of Death

As shown above, chimpanzees, under the most favorable conditions in captivity, show much higher rates of adult mortality and a significantly shorter life span than foragers under the worst conditions (Gurven and Kaplan 2007). This is true in spite of the available evidence, which suggests that members of both species die from similar macro-causes, with the exception of predation (Table 5.2).

In traditional environments, the majority of deaths are due to infections and illness, representing 72 percent and 54 percent of all deaths in traditional humans and wild chimpanzees, respectively. Respiratory-related illnesses,

TABLE 5.2. Causes of death among humans and chimpanzees.

	Traditional Humans		*Wild Chimpanzees*		*Industrialized Human Populations*		*Captive Chimpanzees*	
Cause	*n*	*% Known*	*n*	*% Known*	*n*	*%*	*n*	*%*
All illnesses	2,333	72.4	127	53.6	1,951,920	79.1	104	45.8
Infectious disease			85	35.9	170,521	4.6	13	5.7
Respiratory[a,b]	292	22.2	35	14.8	99,948	4.0	3	1.3
Gastrointestinal[a]	239	18.1	20	8.4	13,284	0.5	6	2.6
Fever[a]	107	8.1			699	0.0	4	1.8
Other infectious			30	12.7	56,590			
Chronic disease					1,425,687	57.8	91	40.1
Heart disease					784,454	31.8	69	30.4
Renal disease					51,084	2.1	22	9.7
Cancer					590,149	23.9		
Other illnesses[a]	317	24.1	42	17.7	355,712	14.4	133	58.6
Degenerative[c]	306	9.5	28	11.8				
Accidents	166	5.2	7	3.0	120,859	4.9	11	4.8
Violence	354	11.0	36	15.2	16,268	0.7		
Homicide[d]	164	6.0			16,259	0.7		
Warfare[d]	137	5.0			9	0.0		
Predation			20	8.4				
Feline-caused			10	4.2				
Human-caused			10	4.2				
Other causes of death	62	1.9	19	8.0	379,388	15.4	112	49.3
Total	3,221	100	237	100	2,468,435	100	227	100

Human data ($n = 3,221$) come from seven groups of hunter-gatherers and forager-horticulturalists (see Gurven and Kaplan 2007 for details). Wild chimpanzee sample ($n = 237$) is based on known reported deaths from Gombe (Williams et al. 2008), Mahale (Nishida et al. 2003), and Taï (Boesch and Boesch-Achermann 2000) populations. Data on industrialized human populations ($n = 2,468,435$) are based on Table 10 of the 2010 National Vital Statistical Reports for the United States (Murphy et al. 2013). Captive chimpanzee data ($n = 227$) compiled from Varki et al. (2009).

a. Illness breakdown does not exist for all human groups. These percentages are based on a risk set of 1,644 individuals and adjusted to sum to 72.4 percent.

b. Respiratory illness accounts for 48 percent of all illnesses in Gombe, 20 percent in Mahale, and 0 percent in Taï.

c. Degenerative illnesses overlap with chronic diseases, but greater specificity is lacking among most traditional human populations (but see Gurven and Kaplan 2007 for more details).

d. Information on violence-related deaths does not exist for all human groups. These percentages are based on a risk set of 2,272 individuals and adjusted to sum to 11.0 percent.

such as bronchitis, tuberculosis, pneumonias, and other viral infections, account for a fifth or more of illness-related deaths among humans, and 15 percent among wild chimpanzees. However, most infectious diseases are absent in many newly contacted Amazonian groups, because small, mobile populations cannot support these contagious vectors (Black 1975). Gastrointestinal illnesses account for 5–18 percent of deaths in traditional human societies. Diarrhea coupled with malnutrition is and remains one of the most significant causes of infant and early childhood deaths among forager populations. People living in tropical forest environments are especially vulnerable to helminthic parasites (Dunn 1968), which, although not usually lethal, can compromise growth and immune function. Few deaths related to gastrointestinal illness have been reported among wild chimpanzees, although one case of gastrointestinal anthrax was confirmed in the Taï chimpanzee population (Leendertz et al. 2004) and four chimpanzees in the Kasekela community at Gombe were reported to have gastrointestinal symptoms prior to death (Williams et al. 2008).

There is a notable lack of data on degenerative disease in traditional humans and wild chimpanzees, but they are probably very rare in both groups. Degenerative disease accounted for about 9 percent of adult deaths in a sample of hunter-gatherers and horticulturalists, with the highest representation among Northern Territory Aborigines. Neoplasms and possible heart disease each accounted for nine of the forty-nine deaths due to degenerative illness in adults over age sixty. It should be pointed out, however, that chronic illnesses as causes of death are the most difficult to identify, since more proximate causes are likely to be mentioned in verbal autopsies. In traditional humans, cases of degenerative deaths are confined largely to perinatal problems early in infancy, late-age cerebrovascular problems, as well as attributions of "old age" in the absence of any obvious symptom or pathology. Heart attacks and strokes appear to be rare, and do not account for these old-age deaths (see Eaton et al. 1988), which often occur when sleeping. Although some evidence of degenerative joint disease has been observed in Kibale chimpanzees, these were mild and unlikely to be lethal (Carter et al. 2008). Old chimpanzees, a potential high-risk group for degenerative disease, tend to "disappear," and, as with foragers, most of these deaths are attributed to senescence (Nishida et al. 2003). Neoplasms have been observed in captive chimpanzees, but reviews suggest lower incidence than in humans (and Old World monkeys) (Lowenstine et al. 2016); the

higher proapoptotic gene expression (i.e., programmed cell death) observed in chimpanzees compared with humans is consistent with less carcinogenesis in chimpanzees (Arora et al. 2012).

Despite the expressed fear and cultural importance of dangerous predators, as represented by mythologies, stories, songs, and games, death by predation is rare among extant foragers. Grouping patterns, weapons, warning displays (e.g., fires), and other cultural means of avoiding predators may contribute to the reduced impact of predation on human survivorship (Wrangham et al. 2006). In contrast, predation could be an important cause of death among chimpanzees; however, scant data and the disappearance of predators from most chimpanzee habitats makes it difficult to determine whether the few predation reports (Boesch and Boesch-Achermann 2000; Furuichi 2000; Nakazawa et al. 2013) are rare anomalies, or are instead a typical source of mortality of past.

Intraspecies violent death appears to be a common feature of human and chimpanzee societies, accounting for 11 percent of 3,221 documented hunter-gatherer and horticultural deaths, and 15 percent of the 237 chimpanzee deaths (Table 5.2). However, Wrangham and colleagues (2006) report lower values for the latter, ranging between 1 percent and 3 percent. Infanticide is also commonly practiced in both. Those at greatest risk of being abandoned or killed in humans are the sickly, unwanted, those of questionable paternity, females, and those viewed as bad omens, such as twins (Milner 2000). Intergroup encounters are the most common context for violent deaths among chimpanzees (66 percent), with adult males the usual perpetrators (92 percent), and adult males (73 percent) and infants the most common victims (Wilson et al. 2014). Despite similar levels of violent deaths in chimpanzees and humans, the rate of intragroup violence (both lethal and nonlethal physical aggression) is remarkably higher in chimpanzees than in humans (Wrangham et al. 2006). It is likely, however, that violent deaths among humans decreased with increased state-level intervention and missionary influence in many small-scale groups around the world (e.g., Agta, Aché, Aborigines, !Kung, Yanomamo).

Finally, the composition of accidental deaths varies across groups of traditional humans, including falls, river drownings, accidental poisonings, snake bites, burns, and getting lost. Together, accidental and violent deaths account for 4–43 percent (average 19 percent) of all deaths in traditional humans. In wild chimpanzees, accidents account for 3 percent of the reported

deaths (Table 5.2), mostly due to falls from trees (Carter et al. 2008; Williams et al. 2008).

Neither hunter-gatherers nor wild chimpanzees appear to suffer from atherosclerosis or die from heart disease. It has often been remarked that few risk factors for heart disease and cardiovascular disease exist among active members of small-scale societies (Eaton et al. 1994). Obesity is rare, hypertension is low, cholesterol and triglyceride levels are low, and maximal oxygen uptake (VO_2max) is high. This is also likely to be the case among wild chimpanzees, for which evidence of the existence of atherosclerosis has never been reported. However, modern conditions have shifted the causes of death profile considerably. Lifestyle changes in industrialized human populations and captive chimpanzees compared to their traditional counterparts, which include the adoption of a pro-inflammatory diet, sedentary lifestyle, and a relatively aseptic environment, have led to a shift in morbidity from infectious to degenerative disease (Finch 2012). Infections decreased significantly with improvements in sanitation and control by vaccination and antibiotics in industrialized human societies and captive chimpanzee populations. The main cause of death among modern humans living in industrialized countries, and chimpanzees living in captivity, is now heart disease,[1] which accounts for more than 30 percent of deaths in both species (Table 5.2).

However, death by heart disease among humans is caused primarily by advanced coronary atherosclerosis, and is typically associated with high cholesterol, obesity, chronic inflammation, metabolic syndrome, and cigarette smoking. Among captive chimpanzees, heart failure is due to extensive interstitial myocardial fibrosis and arrhythmias (Varki et al. 2009). There is little evidence of arterial plaques in captive chimpanzees, despite their sedentary lifestyles, pro-atherogenic blood lipid profiles, occasional hypertension, and homozygosity for ApoE4 (a strong risk factor for atherosclerosis in humans). Finch (2012) suggests that humans are better adapted to chronic inflammation due to greater exposure to inflammation through changes in diet, technology, and pathogen exposure over evolutionary history (see below).

Senescence

The pace at which mortality rates double is a common empirical measure of demographic senescence, defined as $\ln 2 / \beta$, where β is the rate of increase

when mortality grows exponentially with age. Exponential growth, or the Gompertz model, gives a reasonable fit to adult mortality patterns in a wide range of species, including humans and chimpanzees. Finch and colleagues (1990) report mortality rate doubling times (MRDTs) of seven to eight for a variety of recent human populations with low and high mortality. Despite the overall high mortality of hunter-gatherer populations, the adult mortality rate also doubles in seven years among Aché and nine years among Ju/'hoansi (Gurven et al. 2007). Hadza MRDT is just outside the reported range of other human populations, with MRDT of six years. The Hiwi MRDT shows rapid senescence (2.8 years). Several forager-horticulturalists and acculturated foragers show a similar MRDT of eight years, including two Yanomamo samples and settled Aché. The sample of forager-horticulturalists show MRDTs within the range of six to twelve. The acculturated foragers show a range of MRDT from seven to eleven. Overall, the highest-quality data among foragers shows a range of MRDT at six to ten. Chimpanzees show MRDT values that are roughly similar to that of human foragers, ranging between seven and nine years (De Magalhães 2006; Bronikowski et al. 2011). However, as described earlier, the onset of mortality rate doubling occurs at least ten to twenty years earlier among chimpanzees.

While humans appear to senesce more slowly than chimpanzees, it is an open question whether the pace of aging has slowed down in recent human history and among captive chimpanzees. Adult mortality has declined, but it is unclear whether the rate of functional, physiological decay has fallen in tandem. Aging is often tricky to define and measure. The crudest but most available method for making inferences about past aging patterns uses historical mortality data to measure age-related changes in mortality. For example, longitudinal analysis of European mortality data suggests that senescence has slowed over the past couple of centuries, where senescence is defined in several different ways (Gurven and Fenelon 2009). This observation is consistent with the notion that reductions in "extrinsic" age-independent mortality (e.g., infectious disease, accidents, and other nondegenerative causes) should lead to greater investments in repair and maintenance, thereby resulting in longer life span, as originally hypothesized by Williams (1957). However, MRDT tends to be lower in low mortality societies, which would conversely suggest a faster rate of aging. One explanation, called the "heterogeneity hypothesis," argues that in high mortality populations, only the robust survive to late ages, thereby giving the appearance at the population level of a slower rate of adult mortality increase (Vaupel et al.

1979; Hawkes et al. 2012). According to this view, low mortality populations would show greater heterogeneity in individual frailty among adult survivors, and so population-level mortality increase might seem faster. A simpler explanation rests on the peculiarity of MRDT and the manner in which it is estimated. It is possible that if the onset of exponential growth in mortality is pushed to later adult ages in low mortality populations, and if survivorship at the latest ages has improved less than at other adult ages, then estimates of MRDT will be lower and will give the appearance of a more rapid increase at the population level.

Unraveling species differences in aging will require moving beyond actuarial measures and instead focusing on changes in physiological condition and the selective forces impinging on their function. The similar role of infections as principal causes of death in chimpanzees and humans suggests that species differences in the ability to fight against and tolerate pathogens may be critical. The proximate pathways allowing humans to delay somatic aging are, however, not well understood, and no "magic bullet" biomarker has yet been discovered. Two biomarkers proposed to promote greater somatic maintenance and longevity include the steroid hormones estrogen and dehydroepiandrosterone sulfate (DHEAS) (Lane et al. 1997; Belvins and Coxworth 2013). Estrogen affects diverse tissue and cells, and plays an important role in the maintenance of many physiological systems (Lane et al. 1997; Roth et al. 2002; Kemnitz et al. 2006). Adrenal androgens such as DHEAS may be responsible for 75 percent of estrogen in women before menopause and close to 100 percent after menopause (Labrie 1991). Total DHEAS levels in women are three times higher than in age-matched female chimpanzees, and only after their late sixties do human concentrations fall to the highest chimpanzee level (Belvins and Coxworth 2013). However, rates of decline in DHEAS are slower than in human females.

At the cellular level, slower somatic aging in humans may be produced by a reduced rate of telomere attrition (Gomes et al. 2011; Hawkes and Coxworth 2013). Telomeres, which are the noncoding sequence at the end of chromosomes, protect chromosomes from deterioration and from fusion with neighboring chromosomes. With each cell division, telomeres become shorter, which decreases the organism's capacity to regenerate tissue. Thus, if telomere shortening were responsible for somatic aging, one might hypothesize that attrition rates in chimpanzees should be twice as fast than in humans (Hawkes and Coxworth 2013). However, chimpanzees and humans appear

to show similar attrition rates, and chimpanzee telomeres are twice as long as those in humans. Indeed, telomere lengths may vary inversely with species-typical life spans (Gomes et al. 2011). Short telomeres combined with lower telomerase expression in humans may offer protection from runaway cell growth (cancer) in ways that are preferable to replicative aging. Species comparisons, even among primates, are therefore not yet clear.

Menopause and Postreproductive Life Span

Even for human populations living without health care, public sanitation, immunizations, or abundant and predictable food supply, up to one-third of the population is likely to live to age fifty, with an expected fifteen to twenty years remaining (Figure 5.5). With an average age of first reproduction of eighteen years, up to 40 percent of hunter-gatherer women could expect to reach the age at which a first grandchild would be born (36 yrs). For hunter-gatherers who survive to the age of reproduction, the average modal adult life span is about seventy-two years of age (range: 68–78; Gurven and Kaplan 2007). Existing paleontological evidence suggests that a postreproductive life span existed anywhere from 150,000–1.6 million years ago (Bogin and Smith 1996; Caspari and Lee 2004). Chimpanzees, on the other hand, have somatic aging rates similar to humans, and rarely survive their reproductive years (Goodall 1986; Emery Thompson et al. 2007; Jones et al. 2007). New evidence suggests that the rate of decline in ovarian follicular stock may even be faster in humans than chimpanzees (Cloutier et al. 2015), which further suggests that menopause is not a characteristic of chimpanzee life history; fertility decline is the ancestral trait common to humans and chimpanzees, whereas human postreproductive longevity is the derived trait. Indeed, attempts to model the evolution of menopause fail to show that the selective benefits of helping descendants would ever be sufficient to favor fertility cessation over extending reproduction to later ages (Hill and Hurtado 1991; Rogers 1993).

A more recent approach suggests that menopause would be favored when there are resource conflicts among women and their daughters-in-law; asymmetries in kinship—where women would be unrelated to their mothers-in-law's future offspring, but mothers-in-law are related to their grandchildren—give daughters-in-law the upper hand, and presumably priority reproduction (Cant and Johnstone 2008). This model, however, has fairly rigid assumptions

FIGURE 5.5. A Hadza grandmother cares for her grandson. Photo by Brian Wood.

(e.g., female dispersal and male philopatry, no coercion nor synergies in production or childcare, other kin relationships are ignored), and mixed empirical support (Lahdenperä et al. 2012; Mace and Alvergne 2012).

It is likely that extension of the female reproductive life span is not feasible due to trade-offs associated with the mammalian pattern of restricted oocyte production, where the complete, fixed supply of follicles is established in the second trimester of fetal development, and later subject to processes of gamete selection and decay (atresia) that seems oriented toward preserving embryo quality (Ellison 2001). Several theories have therefore been proposed to explain the extension of the human life span, rather than the evolution of menopause. The classic theory of senescence in evolutionary biology was first proposed by Medawar (1952), developed further by Williams (1957), and then formalized by Hamilton (1966). It proposes that as individuals age, they contribute less to biological fitness because less of their expected lifetime fertility remains. Consequently, natural selection acts more weakly to reduce mortality at older ages. The existence of substantial postreproductive life among humans therefore suggests that older individuals have "reproductive value" by increasing fitness through nonreproductive means.

George Williams was the first to suggest that beginning at ages forty-five to fifty, mothers may benefit more from investing their energy and resources in existing children rather than from producing new ones (1957). This idea became known thirty years later as the "Grandmother Hypothesis." One version of the Grandmother Hypothesis proposed by Kristen Hawkes and colleagues (Hawkes et al. 1998; Hawkes 2003) focuses on intergenerational transfers by older women. It proposes that older women can increase their inclusive fitness by enhancing offspring fertility and survivorship of grandchildren through provisioning or providing support to younger generations. Among foragers, the resources acquired by women are strength-intensive, disadvantaging young children and thereby increasing the value of older women's labor contributions. According to this view, extensions in the human life span are driven by selection on women, and the value of resource transfers from grandmothers to grandchildren. The initial inspiration for the Grandmother Hypothesis came from fieldwork done with Hadza foragers in Tanzania, where "hardworking" older women were observed to produce substantial quantities of food.

Peccei (2001) amends this view by pointing out that the long-term juvenile dependence among humans implies that adults who cease reproducing

in their forties will not finish parenting until they are sixty or older (see also Lancaster and King 1985). The notion that most of the benefits to longevity derive from helping offspring rather than grandchildren has been called the "Mother Hypothesis."

An alternative view focuses on men. Marlowe argues that the extension of the life span is driven by selection on men, stressing the fact that men do not experience menopause and can have children into the seventh and eighth decades of life (2000). His argument, called the "Patriarch Hypothesis," is that as men age they accrue status and power that they use to obtain reproductive benefits. These benefits and the lack of a physiological menopause select for their greater longevity. Formal demographic models of life history evolution typically focus only on females, but two-sex demographic models where men tend to be older than their spouses may also lead to a pattern of delayed senescence after the age of fifty (Tuljapurkar et al. 2007). In the two-sex model, selection can favor survivorship for as long as men reproduce, lending additional support to the Patriarch Hypothesis. This model, however, requires extensive late-age male fertility more characteristic of polygynous societies, and/or mating patterns where fertile women mate with older men. Another model that does not require kin assistance (and that has been applied so far only to the arthropod *F. candida*) proposes that postreproductive life span can evolve as insurance against "life span indeterminacy," whereby greater variance in somatic and/or reproductive life spans selects for longer postreproductive life spans (Tully and Lambert 2011). The logic is that longer postreproductive life span reduces the risk of dying by chance before the cessation of reproduction. Both of these models are noteworthy in that neither requires extended parental or grandparental care.

The "Embodied Capital Model" suggests that timing of life events is best understood as an "embodied capital" investment process (Kaplan et al. 2000; Kaplan and Robson 2002; Gurven et al. 2006). Embodied capital is organized somatic tissue such as muscles, immune system components, and brains. In a functional sense, embodied capital includes strength, skill, knowledge, and other abilities. Humans are specialists in brain-based capital. High levels of knowledge and skill are required to exploit the suite of high-quality, difficult-to-acquire resources human foragers consume (Walker et al. 2002; Gurven et al. 2006). Those abilities require a large brain and a longtime commitment to development. This extended learning phase, during which productivity is

low, is compensated for by higher productivity during the adult period. 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. Thus, the long human life span coevolved with the lengthening of the juvenile period, increased brain capacities for information processing and storage, and intergenerational resource flows. Similarly, the "Reserve Capacity" hypothesis proposes that a supportive social system allowed mothers to wean their children earlier and to delay maturity, which allowed for a longer period of somatic investment. The larger reserve capacity resulting from a longer parental investment could result in prolonged longevity (Larke and Crews 2006; Bogin 2009).

Finally, the control-of-fire hypothesis complements these models by arguing that human use of fire for cooking helps increase the efficiency of provisioning by promoting food digestibility and energy, and by allowing early weaning through increased availability of weaning foods (Wrangham 2009; Wrangham and Carmody 2010). It also further reduces extrinsic mortality by detoxifying certain foods, helping to eliminate food-borne pathogens, and deterring predators.

Many of these evolutionary models and hypotheses are not mutually exclusive. They differ in their focus on women (Grandmothering, Mothering), men (Patriarch), or both sexes (Embodied Capital), their reliance on resource transfers as primary (Grandmothering, Mothering, Embodied Capital) or secondary (Patriarch) to life span extension, and whether slow development early in life and life span extension are coupled with economic surplus midlife and the skills-intensive nature of the human foraging niche requiring learning and instruction (Embodied Capital versus Grandmothering/Mothering).

All models except the Patriarch and Life Span Indeterminacy Hypotheses posit that future remaining (caloric) productivity, or productive value (V_x), can impact fitness even when reproductive value (R_x) (*sensu* Hamilton) is low or zero. In one study, V_x has been estimated as the sum of all future net caloric production, discounted by future mortality (Gurven et al. 2012). When comparing R_x and V_x in humans and chimpanzees, it is clear that they show similar profiles across the life course (Figure 5.6). Humans, however, show a huge surplus of caloric production in midlife, with declines occurring well beyond the reproductive years in both males and females. Given the ubiquity of food transfers among humans within and across generations,

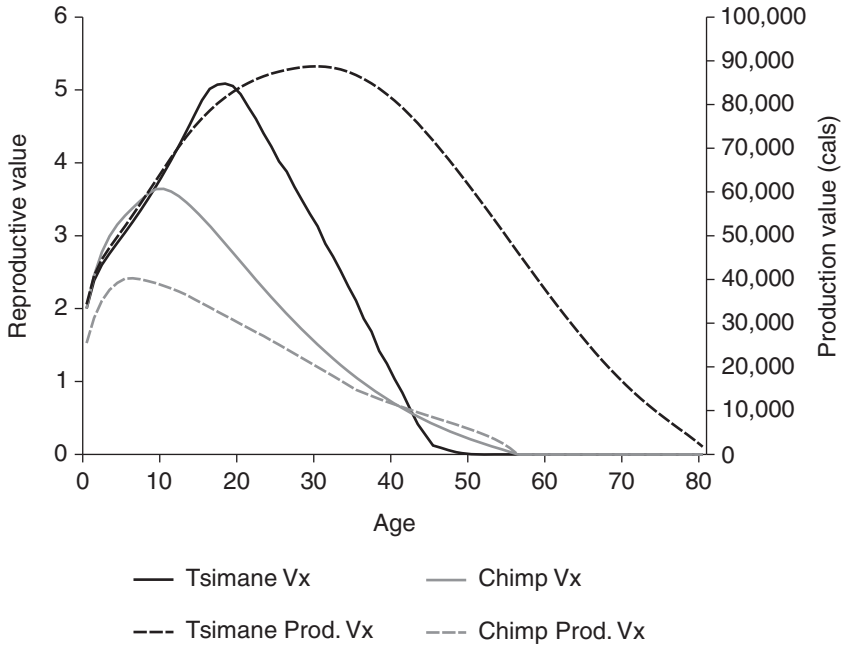

FIGURE 5.6. Reproductive and productive value among Tsimané and wild chimpanzees. Reproductive value (R_x) at age x reflects expected future remaining reproduction (i.e., $\sum_{i=x+1}^{\infty} l_i m_i$, where l_i is survival from birth to age i and m_i is annual fertility at age i). Productive value (V_x) is similar to reproductive value but replaces m_i with age-specific caloric production. Chimpanzee reproductive and productive values show similar trajectories with age; among humans, productive value peaks much later than reproductive value and remains substantial throughout much of adulthood, even after reproductive value is zero. Chimpanzee mortality and fertility data come from Hill et al. (2001) and Emery Thompson et al. (2007: table S1), respectively. Tsimané mortality data are from Gurven et al. (2007) and fertility from Gurven (unpublished data).

particularly to close kin, the high production value in late adulthood can greatly increase fitness impacts of older adults. Older adults make "transfers" of aid, advice, instruction, mediation, and other nonfood contributions that can also have important fitness consequences for kin (Gurven et al. 2012). Mortality rates in late adulthood coincide with rapid declines in V_x, and with lower numbers of potential recipients who are dependent kin (Gurven and Kaplan 2007).

These models were developed to explain the extended life span in the hominid lineage, relative to chimpanzees and other primates, but none di-

rectly address more recent changes in the human life span or the proximate mechanisms by which life span is extended. Since the early 1800s, human life expectancy has increased worldwide due to rapid declines in infant and child mortality; however, late-age mortality has continued to decline as well, and the modal age of adult death has increased by at least a decade (Vaupel 1997). Finch and colleagues have argued that genetic changes responding to alterations in infectious exposure, nutrition, and inflammatory immune responses over the course of hominin evolution are responsible for the lengthier life spans of humans, and the possibility for improved environmental conditions to continue lowering mortality rates (Finch 2012). A gradual reliance on scavenging, hunting, and cooking could alter the selective environment among hominins relative to forest-dwelling apes. Greater meat and fat consumption, pathogen exposure from scavenged meat, and noninfectious inflammagens (or harmful compounds called advanced glycation end products that speed up oxidative damage to cells and are implicated in worsening of many degenerative diseases) from cooked food would have selected for "meat-adaptive" genes. One of the more important of these includes apolipoprotein E alleles that are pro-inflammatory to heighten immune responses. While advantageous in high infection contexts, as in ancestral populations, pro-inflammatory genotypes unique to humans, in the low infection environments of the past century, have had adverse consequences on cardiovascular disease and brain aging (Finch and Sapolsky 1999; Finch and Stanford 2004). These genetic changes might explain why captive chimpanzees do not experience the same degenerative diseases as industrialized populations of humans (Varki et al. 2009).

Social Buffering and Extrinsic Mortality Reduction

While evidence and support are limited to pit the models above against each other, it is likely that low extrinsic mortality is a critical factor underlying the life history of long-lived species such as humans (Table 5.1). Among early humans, low juvenile-adult mortality was likely a prerequisite for further reductions in adult mortality and the further slowing of the life course. Whereas other long-lived species with low extrinsic mortality often inhabit microbe-free and predator-free microenvironments, the lower extrinsic mortality of early humans may have come from effective group defense against

predation and from the nurturing of sick and injured individuals (Gurven et al. 2000; Sugiyama 2004).

The average forager female has about six births (Table 5.1), which places substantial burden on household feeding requirements. Allowances usually made for pregnant and lactating women, who reduce foraging efforts but nonetheless receive ample food, enable such a high level of human fertility with short interbirth intervals. However, the risk of food shortfalls occurs among males and females at all ages, as even adults in their peak production years cannot consistently meet the daily caloric needs of their large families. The human foraging niche leads to the possibility of greater risk of food shortfalls over the life course, but also includes a variety of other important risks that can impact fitness. Illness left untreated can lead to cascading morbidity and possibly death, and often impairs the ability to produce food or perform other important daily tasks. Death or divorce renders dependent children vulnerable to food shortage, disease, and lack of protection, and renders adults vulnerable to labor shortage. Conflict left unresolved, especially among kin, can result in fractured social and sharing networks, migration, fighting, and homicide. Theft and breakage of important tools, possessions, or other resources can potentially disrupt production and often incurs substantial costs to replace.

Human cooperation and sociality likely evolved to reduce risk in these fitness-relevant domains. Managing risk in the short term (e.g., daily food shortfalls) and in the long term (e.g., handling illness, feeding extra dependents, defense against predators and enemies) would result in lower baseline or "extrinsic" mortality. Baseline mortality in chimpanzees is about 70 percent higher than in humans (Table 5.1), due primarily to differences in predation and illness rates. Risk reduction was necessary for sustaining a foraging way of life, and is a central component of the evolved human life history. Although wild chimpanzees share food and other resources (Mitani and Watts 2001; Gomes and Boesch 2011), most food transfers are passive (Jaeggi and van Schaik 2011). Chimpanzees rarely live beyond age forty-five, the age when humans reach peak net economic productivity, and chimpanzee grandparents are rarely observed helping anyone (Goodall 1986). Chimpanzees also rarely help or care for sick individuals. Thus, humans may be unique in the breadth and volume of transfers and help given across different domains. But the question remains as to how *Homo sapiens* diverged from other hominins and why others did not follow the same path. As mentioned

above, the control-of-fire hypothesis suggests that the strategic use of fire could have protected early hominins from predators. Additionally, early bipedalism, longer day ranges, and more efficient terrestrial locomotion among ancestral hominins likely pushed for greater hunting and gathering specialization, and a reliance on large packaged but relatively high-variance foods (Kaplan et al. 2000). During the drying of the Pleistocene and the expansion of African savannahs, these hominins would have been "pre-adapted" to better reap the gains from increased specialization on the mammal species, roots, and nuts that proliferated during this period.

Thus, we can speculate that the use of fire, economic gains from improved foraging behavior, and greater sociality may have helped lower extrinsic mortality risks in ancestral hominins. This initial lowering of extrinsic mortality could then select for further investment in reducing adult mortality rates. With a greater probability of reaching adulthood, selection would then have favored further specialization toward skill-intensive foraging subsistence strategies with delayed returns, and a higher rate of intergenerational transfers, as argued by the Embodied Capital Model (Kaplan and Robson 2002). Regardless of the original benefits of sociality, the mortality-lowering effect of social support should thus have pushed early hominins toward greater foraging specialization, extended development, and survivorship to produce and support kin at higher levels later in life.

Conclusion

There appears to be a characteristic life span for the human species, in which mortality decreases sharply from infancy through childhood, followed by a long period in which mortality rates remain essentially constant to about age forty, at which point mortality rises exponentially. Despite the potential for similar mortality levels in infancy and the juvenile period, chimpanzee mortality (except for Ngogo) tends instead to increase exponentially from its trough at age ten. There is a modal age of adult death of about seven decades for humans, whereas the mode does not exceed three decades in chimpanzees. Productivity and helping behavior can positively impact kin fitness before this time, after which senescence occurs rapidly and people die. We hypothesize that human bodies are designed to function well for about seven decades in the environment in which our species evolved.

There are differences in mortality rates among populations and among periods, especially in risks of violent death. However, those differences are small in a comparative cross-species perspective, and the similarity in mortality profiles of traditional peoples living in varying environments is impressive.

The evolved human life span is a core life history trait whose explanation frequently includes other derived or exaggerated traits. Compared to our chimpanzee cousins, humans ~~have~~ not only have long lives, but large brains and bodies that grow and develop slowly (Isler and van Schaik 2009). Human diets are made up of high-quality, nutrient-dense foods that come in large packages, while cooperation, sociality, pair-bonds, divisions of labor and multigenerational resource transfers help underpin subsistence, parenting, and risk-management strategies. Whether these traits coevolved as a bundle or in sequence, and how they evolved, remain to be determined, but the answer will no doubt require greater attention to chimpanzee behavior and ecology. The Embodied Capital Model is the most comprehensive explanation for species differences to date, in that it ties together in one coherent framework the coevolution of long life, slow growth, encephalized brains, and high sociality—all as an outcrop of the shift to a more uniquely human foraging niche. However, it may not be the most parsimonious model. One comparative study involving fifty-seven bird and mammal species, however, provides broader support for the Embodied Capital Model by demonstrating positive relationships between cooperative foraging, greater sociality, and delayed foraging competency (Schuppli et al. 2012).

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 appears along with a complex of long-term child dependence during which learning trumps productivity, and high productivity of adults, especially in middle age. Together, the costs of slowing senescence, preventing mortality, and the benefits of extended investment in descendants produced selection for a characteristic human life span, with some variance around the central tendency. The similar mortality profiles of eighteenth-century Sweden to hunting and gathering populations suggest that comparable age distribu-

tions of adult deaths occur under a relatively broad range of environmental conditions. The chimpanzee-human comparison does reveal, however, that species differences overwhelm differences in environmental conditions in determining mortality hazards throughout adulthood. This might suggest 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.

Chimpanzees are likely to be a good ancestral model for testing hypotheses about the evolution of long life spans in humans. Future studies could benefit from controlled comparisons of biomarkers of aging among wild and captive chimpanzees, and among subsistence populations varying in diet, activity, and pathogen risk. Species comparisons of metabolism, immune function, repair mechanisms, and their genetic underpinnings will be especially instructive, and may provide insight into how and why the greater (or at least more specialized) investment in somatic maintenance that occurred over the course of hominin evolution resulted in human life span surpassing that of all other primates.

Endnotes

1. In the Yerkes captive chimpanzee colony, infectious disease was replaced by heart disease as the main cause of death when vaccination was introduced and sanitation improved in the early 1990s (Varki et al. 2009).

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Similarly wide variations in life expectancy and maximum life spans are observed in animals and plants, within populations as well as between closely related species. The preceding chapters review the details of these increases and some of the mechanisms that may be involved. However, major unknowns remain about the nature of the aging process in humans and other organisms. The general information that is available from all sources gives a very limited basis for predicting further changes in human aging schedules and ultimate life spans.Â A scale of senescence is shown that approximates the rates of mortality acceleration in adults, ranging from rapid to gradual to negligible (Finch, 1990).