Longevity Among Hunter-Gatherers: A Cross-Cultural Examination

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AVERAGE WORLDWIDE HUMAN life expectancy reached 66 years in the first quinquennium of the twenty-first century, with extremes at the country level ranging from 39 years in Zambia to 82 years in Japan (United Nations 2007). Average life expectancy has increased linearly at almost three months per year over the past 160 years, with improvements in sanitation, nutrition, and public health accounting for much of this change (Riley 2001; Oeppen and Vaupel 2003). As a consequence of longevity in the developed world, women currently live more than a third of their lives in a post-reproductive state following menopause.

Such high survival rates almost surely had never occurred before in human history. Agriculture and pastoralism have been practiced for only about 10,000 years, and most extensively in the past 5,000 years. The genus *Homo* has existed for about 2 million years, and humans have lived as hunter-gatherers for the vast majority of their evolutionary history. While some important genetic changes may have occurred in populations after the advent of agriculture, the major distinctive features of our species (Wang et al. 2006), such as large brains, long lives, marriage and male investment in offspring, long child dependency on parents, and grandparental support of grandchildren, appear to have evolved during our preagricultural history (see Kaplan 1997 for reviews; Kaplan et al. 2000, 2001). Despite recent improvements in human survivorship, it is likely that the age-specific mortality pattern and the timing and pace of development and senescence evolved during our hunter-gatherer past as well.

The purpose of this article is to assess the evolved human mortality profile and particularly the pattern of senescent mortality change with age. We address five questions:

1) Is there a characteristic shape to the human mortality profile, as it decreases first during childhood and then increases with aging?

2) How robust is the occurrence of a post-reproductive life span, and how likely is it that older adults were alive and available in human populations?

3) Is there a characteristic modal age at death for adults, and what can this mode tell us about aging and the evolution of the human life span?

4) How variable is this mortality profile among populations, and what factors shape any variation?

5) How do the mortality patterns of modern hunter-gatherers compare with estimates of the mortality profiles of both chimpanzees, our closest living primate relative, and prehistoric populations derived from funerary samples, and what implications can be derived from those comparisons?

Our approach is to assess and analyze available demographic data on extant hunter-gatherers and forager-horticulturalists (i.e., peoples who mix hunting and gathering with swidden agriculture). To understand the processes that shaped the evolution of our life course, it would be useful to have data on mortality and fertility across populations and over evolutionary time. Because these data do not exist, we critically evaluate data on modern groups as one lens into our past, however imperfect.

Our conclusion is that there is a characteristic life span for our species, in which mortality decreases sharply from infancy through childhood, followed by a period in which mortality rates remain essentially constant to about age 40 years, after which mortality rises steadily in Gompertz fashion. The modal age of adult death is about seven decades, before which time humans remain vigorous producers, and after which senescence rapidly occurs 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. Mortality rates differ 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.

After publishing their life table for Yanomamo Amerindians of Venezuela 30 years ago, Neel and Weiss (1975) made an "appeal to anthropologists…to produce comparable bodies of [demographic] data." This is our attempt to synthesize the best information about mortality in relatively isolated, small-scale foraging-based populations. The sample of groups used in this analysis is larger than in any other previous study (e.g., Kaplan et al. 2000; Kennedy 2003; Pennington 2001; Gage 1998). This is our best opportunity to examine the mortality structure of humans living a lifestyle most similar to the lifestyles of our foraging ancestors. These populations are acculturating at a rapid rate, thus future data are unlikely to be forthcoming. For example, 33 of 105 isolated indigenous groups in Brazil became extinct between 1900 and 1957, and only 33 groups remain isolated (Ribeiro 1967:

92). There are few extant groups of human hunter-gatherers, and probably no large group for which detailed demographic information on people of all ages is available. The few groups that exist are relatively small, disrupted by infectious disease and interactions with miners, colonists, or other nationals. It is therefore unlikely that more than a few new societies will be added to the sample presented in this article.

Methodology

Our approach is to assess the mortality profiles of all extant hunter-gatherers for which sufficient high-quality demographic data exist. Table 1 provides the study years, time period covered, sample sizes of individuals, total person-years and person-years for individuals aged 50 and older, and sources of the data. The societies in the sample are described in Appendix 1. We include in our data set small-scale populations that also engage in slash and burn horticulture because these groups share similar characteristics with modern foraging populations. Whereas *forager-horticulturalists* have engaged in horticulture for many generations, groups in our *acculturated hunter-gatherer* category have either recently started horticulture and/or have been exposed to medicines, markets, and other modern amenities. All groups in our ethnographic sample of *hunter-gatherers* have had minimal or no exposure to modern medicine, and minimal or no inclusion of products of horticulture or market-derived foods in their diet. Other traits commonly associated with a foraging lifestyle vary among hunter-gatherer groups, such as mobility, an egalitarian ethic, widespread sharing, minimal storage, and other social traits (Kelly 1995). The designation of high-quality data means that age estimation is reasonably accurate and there is no systematic bias in the underreporting of deaths. Most importantly, survivorship and mortality profiles for these populations are based on actual deaths from prospective or retrospective studies, and not on model life tables fitted to scanty data or census data. These profiles therefore make no assumptions about stable or stationary populations, which can bias estimates of adult mortality. Stable population theory requires that mortality and fertility schedules remain constant over long periods of time, while stationary distributions additionally require zero population growth. If a population is actually growing, and stationarity is instead assumed, the death rate is usually overestimated (see Pennington 1996). Additional criteria, borrowed from Early and Peters (2000: 71), require that demographic data collection was an objective of research and that data were subject to rigorous error-checking.

Although this sample is the most complete set of preindustrial populations available, data limitations make it necessary to rely upon subsets of the populations for specific analyses. For example, the Agta and Warao demographies lack sufficient age resolution at later ages for us to trust mortality profiles after the age of 45 or 50 years. Age estimates for older adults among the Gainj are unreliable, with no age categories beyond age 60. Data on older

TABLE 1 Sample of study populations

Group	Type	Years studied	Years covered	No. of individuals	Person-years	No. of deaths	Person-years over age 50	Age intervals	Data collection	Data source
Hunter-gatherers										
!Kung	HG	1963–74	1963–74	500	1,767	96	<17	5,10	retro	Howell 1979: Table 4.1
Ache	HG	1980–94	<1971	971	16,105	353	1,147	1	retro	Hill and Hurtado 1996
Agta	HG	1962–86	1950–64	176	2,635	117	184	5,10	prosp/retro	Early and Headland 1998: Table 8.1
Hadza	HG	1985–95	1985–95	706	6,893	125	1,224	1	prosp census	Blurton Jones et al. 2002
Hiwi	HG	1988–90	<1960	375	3,565	107	341	1	retro	Hill et al. 2007
Forager-horticulturalists										
Yanomamo Xilixana	FH	1959–95	1930–56	120	2,843	64	180	5,10	retro	Early and Peters 2000
Yanomamo Xilixana "Brazilian"	FH	1959–86	1982–95	328	4,420	124	279	5,10	prosp/retro	Early and Peters 2000
Yanomamo	FH	1960s	1960s	2,513	N/A	N/A	N/A	5	cens model fit	Neel and Weiss 1975
Tsimane	FH	2002–03	1950–89	1,702	55,990	916	2,785	1	retro	Gurven et al. 2007
Machiguenga	FH	1988–89	<1988	287	5,520	150	278	5	retro	Kaplan, unpublished
Gainj	H	1970–78	1970–78	1,500	9,102	287	N/A	5	vital regist	Wood 1980, 1987
Acculturated foragers and others										
!Kung	A-HG	1963–74	1963–74	418	4,511	75	818	5	prosp	Howell 1979: Table 4.6
!Kung	A-HG	1963–74	1963–74	94	3,527	94	792	5	prosp	Howell 1979: Table 4.4
Agta	TRANS HG	1962–86	1965–79	200	2,995	100	210	5,10	prosp	Early and Headland 1998: Table 8.1
Agta	A-HG	1962–94	1980–94	212	3,185	147	223	5,10	prosp	Early and Headland 1998: Table 8.1
Warao	A-HG	1954, 66, 1972–73	1954, 66, 1972–73	1,629	18,170	269	1,136	5,10	cens/retro	Layrisse et al. 1980: Table 4.8
Northern Territory Aborigines	A-HG	1958–60	1958–60	17,469	52,407	285	7,968	5	prosp vital regist	Lancaster Jones 1961; Crotty and Webb 1960
Tiwi	A-HG	1952–61	1952–61	N/A	7,243	150	N/A	5,10,15	prosp vital regist	Lancaster Jones 1963
Hiwi	A-HG	1988–90	1961–89	537	4,370	131	614	1	retro	Hill et al. 2007
Ache (settled)	A-HG	1980–94	1978–93	1,112	12,412	113	780	1	prosp	Hill and Hurtado 1996
Sweden 1751–59	HIST	1906	1751–59	1,839,256	16,944,534	457,589	2,857,057	1	parish record	Human Mortality Database: www.mortality.org

A = Acculturated; HG = hunter-gatherer; FH = forager-horticulturalist; H = horticulturalist; TRANS = transitional; HIST = historical.

adults are sparse among Machiguenga. The Aborigine data may suffer from underreporting of infant deaths. The highest-quality data across the entire life span exist for Hadza, Ache, !Kung, Hiwi, Tsimane, and Yanomamo Xilixana. These populations are used in focused analyses of mortality at older ages.

To illustrate patterns of mortality, we present age-specific survivor-ship curves, $l(x)$, and log mortality hazard rates, $\ln h_x$. We model each group's mortality using a Siler competing hazard model, chosen for its simplicity, robustness, and the interpretability of its parameters (Siler 1979; Gage 1989, 1991; but see Wood et al. 2002). The Siler model includes three components of mortality: declining mortality from birth through childhood, a constant mortality hazard across the life span, and an increasing component in older ages. Infant and child mortality are thus modeled with a negative Gompertz function. The final component is the familiar Gompertz exponential, and the constant term is the Makeham (1860) addition of age-independent mortality. Our use of the Gompertz function is based on convenience and tradition. Gompertz is not derived from first biological principles, although different explanations have been proposed to produce Gompertz-like mortality (Olshansky and Carnes 1997; Gavrilov and Gavrilova 2001). Other survival models that show monotonically increasing mortality rates over time, such as Weibull and Gamma, often explain data as accurately as Gompertz (Ricklefs and Scheuerlein 2002; Wood et al. 1994).

The Siler hazard has the following functional form:

$$h(x) = a_1 \exp(-b_1 x) + a_2 + a_3 \exp(b_3 x) \tag{1}$$

This is a five-parameter model. The parameter a_1 describes the initial infant mortality rate, and b_1 describes the rate of mortality decline. The proportion of deaths due to juvenile mortality is captured by the first component as $\exp(-a_1/b_1)$. The parameter a_2 describes age-independent mortality, which is usually interpreted as exogenous mortality due to environmental conditions. The parameter a_3 is the initial adult mortality rate, and b_3 describes the rate of mortality increase. Because the survivorship, $l(x)$, is equal to the product of the three survivorship components, the formula for survivorship at age x is:

$$l(x) = \exp\left(-\frac{a_1}{b_1}(1 - \exp(b_1 x))\right) \cdot \exp(-a_2 x) \cdot \exp\left(\frac{a_3}{b_3}(1 - \exp(b_3 x))\right) \tag{2}$$

We estimate Siler parameters based on fits of (1) to the $h(x)$ values. In view of the discrete nature of the age intervals, we assign the ages for the Siler model to the midpoint between two successive age intervals. All estimation is done using weighted nonlinear regression (PROC NONLIN) with SAS version 9.1, where weights are assigned according to the number of age-specific risk years for each population. Regression of log mortality hazards on age by population is done using the generalized linear model (GLM) procedure in SAS.

Results

Survivorship, mortality hazards, and post-reproductive life

Table 2 presents the results of modeling mortality hazards with the Siler model, and Figures 1 and 2 show the age-specific survivorship (l_x) and the log mortality hazard ($\ln h_x$) curves, based on the life tables derived from the raw data. The results are presented for five hunter-gatherer, four forager-horticulturalist, and five acculturated hunter-gatherer populations. In the table and in panel D of both figures, we compare the averages across groups with data from chimpanzees (Hill et al. 2001) and from eighteenth-century Sweden. The estimated survival curves from the Siler models (not shown) are very close to those generated from the life tables in Figure 1.

In Table 2, we see that on average 57 percent, 64 percent, and 67 percent of children born survive to age 15 years among hunter-gatherers, forager-horticulturalists, and acculturated hunter-gatherers. Of those who reach age 15, 64 percent of traditional hunter-gatherers and 61 percent of forager-horticulturalists reach age 45. The acculturated hunter-gatherers show lower young adult mortality rates, with 79 percent surviving to age 45, conditional on reaching age 15.

All groups show evidence of significant post-reproductive life among women. Mean number of expected years of life, conditional on reaching age 45, is about two decades (20.7, 19.8, and 24.6 for hunter-gatherers, forager-horticulturalists, and acculturated hunter-gatherers). Traditional hunter-gatherers and forager-horticulturalists are almost identical in the adult life course, and, on average, acculturation improves adult life expectancy.

There is some variability among groups. Among traditional hunter-gatherers, the average life expectancy at birth (e_0) varies from 21 to 37 years, the proportion surviving to age 45 varies between 26 percent and 43 percent, and life expectancy at age 45 varies from 14 to 24 years (Figure 1; Table 2 and Figure 3). Ache show higher infant and child survivorship than the other groups, and Agta mortality is high at all ages. These patterns are verified in the parameter estimates of the Siler model (Table 2). Initial immature mortality (a_1) for the Ache is about half that of other foragers, while for the Agta it is two to three times greater.[1]

Forager-horticulturalists also vary significantly in infant mortality, with a threefold difference between Neel and Weiss's Yanomamo sample and the Tsimane. Survival to age 45 varies between 19 and 54 percent, and those aged 45 live an average of 12–24 additional years. The Tsimane show earlier accelerations in adult mortality than the Yanomamo and the forager populations. The raw and simulated Gainj population shows earlier mortality accelerations, although the raw data do not permit a strong inference about ages greater than 55.

TABLE 2 Parameter estimates of the Siler mortality model

Population	a_1	b_1	a_2	a_3	b_3	95 percent C.I. Lower	Upper	MRDT	MRDT 95 percent C.I. Lower	Upper	l_{15}	l_{45}	e_{15}	e_{45}
Hadza (e_0=34)	0.351	0.895	0.011	6.70E-06	0.125	0.078	0.173	5.5	4.0	8.9	0.57	0.40	42.5	24.2
Ache forest (e_0=37)	0.157	0.721	0.013	4.80E-05	0.103	0.048	0.158	6.7	4.4	14.6	0.66	0.43	38.5	21.1
Hiwi (e_0=27)	0.458	1.390	0.020	6.32E-09	0.251	0.127	0.374	2.8	1.9	5.4	0.53	0.29	32.2	17.9
!Kung (e_0=36)	0.340	0.913	0.010	3.31E-04	0.077	0.045	0.110	9.0	6.3	15.6	0.59	0.39	38.1	19.7
Agta (e_0=21)	0.961	1.506	N/A	7.57E-03	0.040	0.016	0.063	17.5	10.9	44.1	0.45	0.26	28.6	13.7
Average hunter-gatherer	**0.422**	**1.131**	**0.013**	**1.47E-04**	**0.086**	**0.051**	**0.174**	**8.1**	**4.0**	**13.6**	**0.57**	**0.36**	**37.7**	**20.7**
Yanomamo Mucaj (e_0=39)	0.624	3.058	0.011	9.60E-05	0.086	0.039	0.134	8.0	5.2	17.9	0.69	0.47	41.3	23.5
Yanomamo (e_0=21)	0.752	1.663	0.024	1.82E-04	0.086	0.056	0.117	8.0	5.9	12.4	0.44	0.19	28.3	16.8
Tsimane (e_0=42)	0.221	1.193	0.009	2.30E-05	0.119	0.084	0.155	5.8	4.5	8.3	0.73	0.54	41.6	20.6
Machiguenga	0.367	1.471	0.016	N/A	N/A	N/A	N/A	N/A	N/A	N/A	0.62	0.39	N/A	N/A
Gainj (e_0=30)	0.277	0.917	0.002	3.18E-03	0.059	0.045	0.073	11.8	9.5	15.5	0.67	0.33	29.6	11.9
Average forager-horticulturalist	**0.418**	**1.657**	**0.012**	**3.65E-04**	**0.074**	**0.024**	**0.181**	**9.4**	**3.8**	**28.9**	**0.64**	**0.39**	**36.4**	**19.8**
!Kung 1963–74 (e_0=50)	0.282	1.089	0.001	3.06E-04	0.073	0.034	0.111	9.5	6.3	20.1	0.76	0.67	50.9	25.2
Ache reservation (e_0=50)	0.248	0.985	0.003	6.20E-05	0.092	0.003	0.180	7.6	3.9	219.4	0.74	0.64	52.0	27.2
Northern Territory Aborigines (e_0=49)	0.242	1.031	0.000	7.13E-04	0.063	0.056	0.070	11.0	9.9	12.4	0.77	0.65	47.6	23.1
Hiwi post-1960 (e_0=28)	0.478	0.828	0.011	4.30E-05	0.098	–0.075	0.272	7.1	2.6	N/A	0.48	0.33	41.9	23.8
Agta transition (e_0=28)	0.404	0.848	0.012	1.01E-07	0.230	0.112	0.349	3.0	2.0	6.2	0.52	0.37	35.7	14.8
Agta peasant (e_0=21)	0.405	0.587	0.004	2.98E-03	0.056	0.010	0.102	12.4	6.8	72.1	0.44	0.25	30.6	13.4
Average acculturated HG	**0.248**	**0.816**	**0.006**	**1.78E-04**	**0.079**	**0.048**	**0.104**	**8.8**	**6.7**	**14.4**	**0.67**	**0.53**	**46.4**	**24.6**
Sweden 1751–59 (e_0=34)	0.400	1.074	0.008	2.01E-04	0.083	0.078	0.087	8.3	7.9	8.9	0.60	0.43	40.0	20.0
Overall human average	**0.309**	**1.138**	**0.009**	**1.31E-04**	**0.090**	**0.052**	**0.128**	**7.7**	**5.4**	**13.4**	**0.66**	**0.47**	**40.3**	**20.8**
Wild chimpanzees (e_0=13)	0.248	0.608	0.028	7.53E-03	0.063	–0.037	0.162	11.0	4.3	N/A	0.37	0.03	14.0	5.0
Captive chimpanzees (e_0=26)	0.389	2.465	0.017	9.96E-04	0.094	0.086	0.102	7.4	6.8	8.1	0.64	0.20	22.3	7.2

MRDT=mortality rate doubling time in years, defined as $\ln2/b_3$. Groups in italics refer to data of questionable quality (see text for details).

FIGURE 1 Survivorship (l_x) for (a) hunter-gatherers, (b) forager-horiculturalists, and (c) acculturated hunter-gatherers using the Siler competing hazards model to estimate mortality. Panel (d) illustrates average l_x for each of these group sets and compares l_x from wild chimpanzees and from Sweden, 1751–59
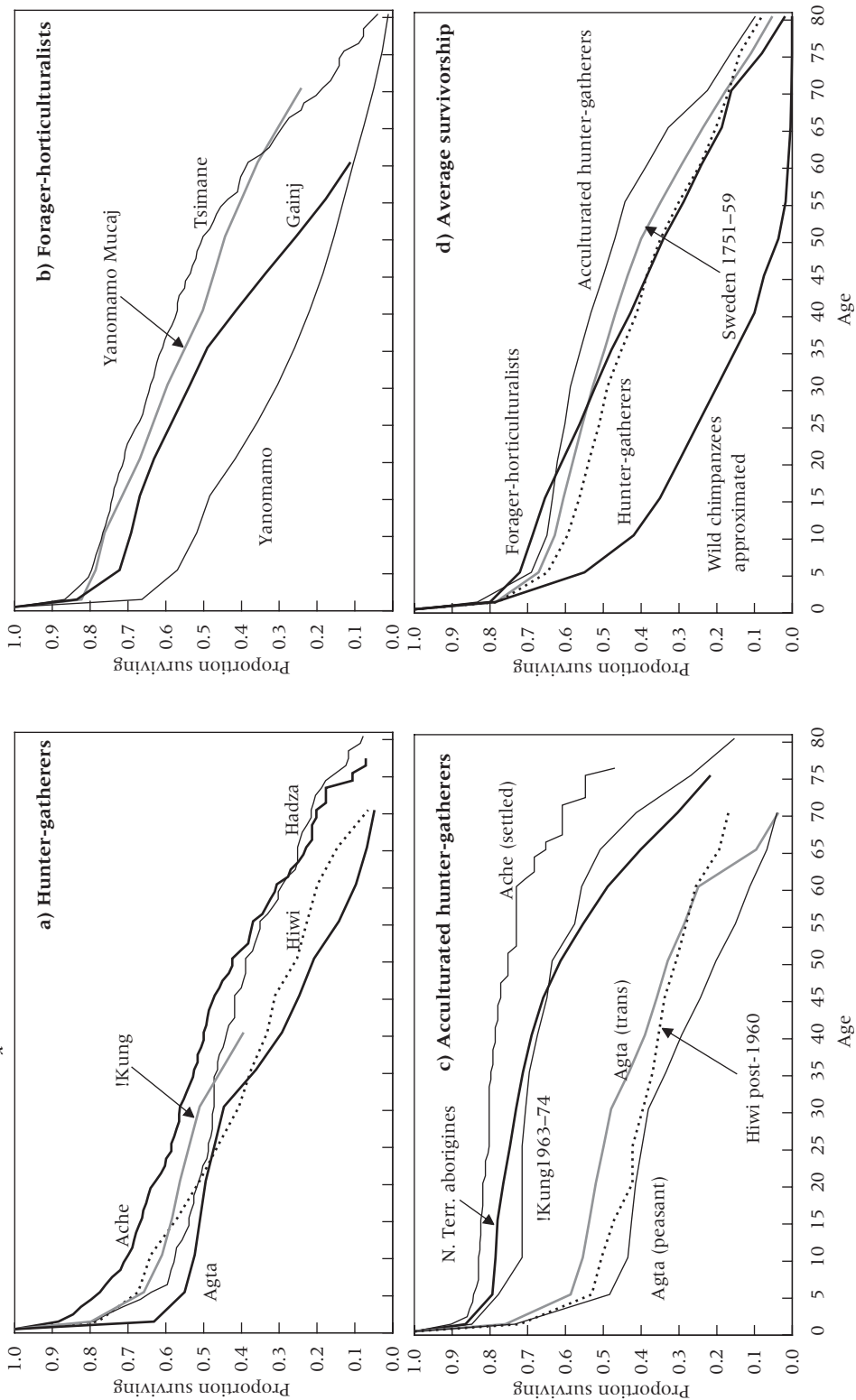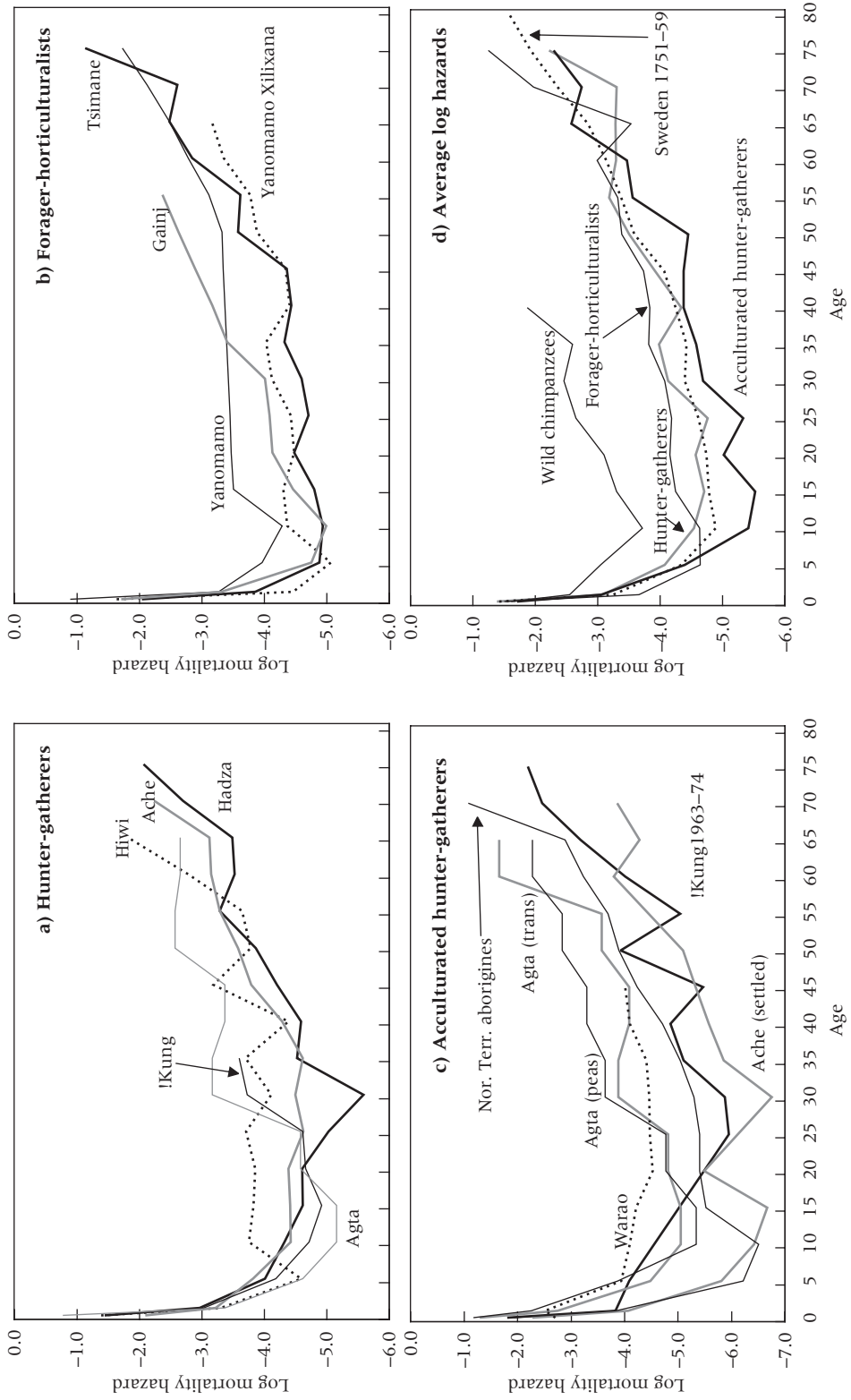
FIGURE 2 Log mortality hazards (ln(h_x)) for (a) hunter-gatherers, (b) forager-horiculturalists, and (c) acculturated hunter-gatherers , and (d) same group averages from Figure 1.

NOTE: All estimates are based on raw data clumped into 5-year age groups.

Acculturated foragers vary most in their likelihood of reaching age 45 (ranging from 26 percent among the peasant Agta population to about 67 percent among sedentary !Kung, Aborigines, and Ache), but show a range of 13–27 additional years of life upon reaching age 45, similar to the range for less acculturated foragers and forager-horticulturalists. Adult mortality is also highly variable. For example, life expectancy at age 15 is 48 years for Aborigines, 52 and 51 for settled Ache and !Kung, yet 31 and 36 for peasant and transitional Agta. Hiwi show similarly low life expectancy. The acculturated category shows a range of mortality experiences associated with acculturation.

The rate at which mortality rates double is a measure of the senescence rate. Finch et al. (1990) report mortality rate doubling times (MRDTs) of 7–8 years for a variety of recent human populations with a wide range in overall mortality.[2] Despite the overall high mortality of hunter-gatherer populations, we find that the adult mortality rate also doubles in 7 years among Ache and 9 years among !Kung (Table 2). Hadza MRDT of 6 years is just outside the reported range of other human populations. The Hiwi MRDT shows rapid senescence (2.8 years). Among the Agta, where high adult mortality is placed in the a_3 rather than a_2, senescence appears very slow for the forager and peasant samples (MRDT=18, 12).

Several forager-horticulturalists and acculturated foragers show a similar MRDT of 8 years, including two Yanomamo samples and settled Ache. The sample of forager-horticulturalists shows MRDTs within the range of 6–12 years. The acculturated foragers show a MRDT range of 7–11 years. Only the two acculturated Agta samples deviate from this range. The highest-quality data mostly show a MRDT range of 6–10 years for our study groups (Table 2, groups not in italics). Combining all groups within each of the three subcategories in the same Siler model, we find a more restricted range for MRDT of 8–9 years (Table 2).

Among groups, differences in early juvenile mortality largely explain the differences in overall mortality. Infant and child mortality varies widely among small-scale traditional populations. Indeed, a linear regression of e_1 on the infant mortality rate using the full sample of populations shows that 56 percent of the variation in e_1 is explained by differences in IMR. IMR also predicts 52 percent of the variation in age-independent mortality, a_2. The largest departure from mortality hazards at later ages appears among the Agta, whose mortality data we earlier described as being circumspect at later ages.

In spite of this variation, a clear premodern human pattern emerges. Age profiles of mortality risk over the life span are remarkably similar. The mortality hazard has slowed to 0.01 by age 10, doubled to about 0.02 by age 40, doubled yet again before age 60, and again by age 70. Low mortality therefore persists until about age 40, when mortality acceleration becomes evident.

The adult mortality rate doubles every 7–10 years. The results obtained from these groups are similar to those from Sweden in 1751 (see panel d in Figures 1 and 2), where mean life expectancy was 34 years and e_{45} was an additional 20 years (Berkeley Mortality Database). For groups living without access to modern health care, public sanitation, immunizations, or adequate and predictable food supply, it seems that still at least one-fourth of the population is likely to live as grandparents for 15–20 years.

Figure 3 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 human groups in life expectancy at early ages, there is significant convergence after about age 30. With the exception of the Hiwi, who have over 10 fewer years remaining during early ages and over 5 fewer years 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 eighteenth-century Sweden, are hardly distinguishable from one another. Figure 3 also shows that at age 40, the expected age at death is about 63–66 (i.e., 23–26 additional expected years of life), whereas by age 65, expected age at death is only about 70–76 years of age. By age 65, death rates become very high.

Chimpanzees show a very different life course, with higher mortality and lower age-specific survival, especially during adulthood. While chimpanzee MRDT values are similar to those of human foragers, the onset of mortality rate doubling occurs at least ten years earlier among chimpanzees.

It is of interest to note how the protected environment of captivity affects chimpanzee mortality profiles (Dyke et al. 1995). Captivity raises infant and juvenile survival greatly, from 37 percent surviving to age 15 in the wild to 64 percent in captivity, with the latter being similar to the human averages. However, while the proportion surviving to age 45 increases sevenfold, from 3 percent in the wild to 20 percent with captivity, it is still just half as high as for humans living in premodern conditions. The difference between chimpanzees and humans after age 45 is even greater, with an expected additional life span for chimpanzees in captivity of only 7 years, about a third of the human expectation. It appears that chimpanzees age much faster than humans and die earlier, even in protected environments.

Nonlinearity in rates of senescence

Senescence is usually defined as an increase in the endogenous rate of mortality (Finch, Pike, and Whitten 1990; Rose 1991). In many populations mortality reaches its minimum at reproductive maturity and 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).

FIGURE 3   Age-specific life expectancy: Expected number of years remaining for six sample populations with sufficient data quality and Sweden, 1751–59
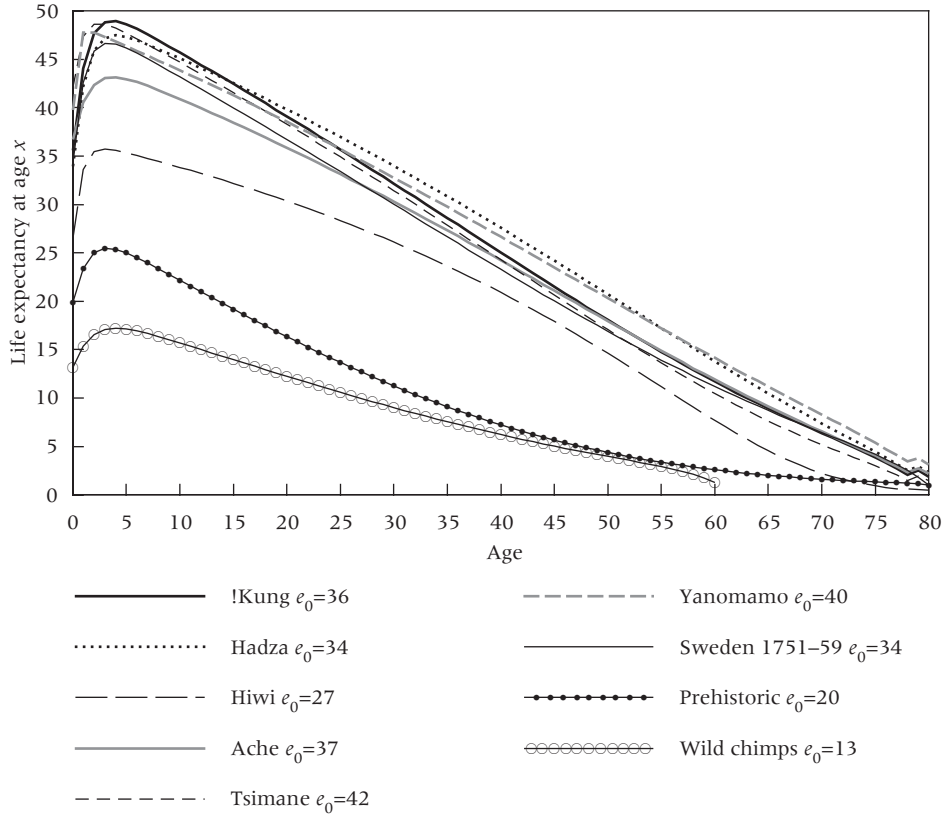
NOTE: Curves are based on life-table estimtes using the Siler model.

The demographic literature often varies on the starting point for expected mortality increases in the Gompertz relationship (for examples see Olshansky and Carnes 1997). Here we examine whether traditional human populations reveal a constant proportional increase in mortality rates. Our goal is to determine whether an extended period of non-senescence during adulthood plays a role in the exceptional longevity of our species.

We do so in two ways. First, we disaggregate the regression of log mortality rate on age into two components, age 15–40 and age 40+. The results are shown in Table 3. For most populations, especially among those with the most reliable data (marked in bold), we find strong evidence of departure from linearity. The slope of mortality increase is greater after age 40 than before that age. Among the Hiwi and Hadza, mortality rates actually decline from age 15 until about 35. The remaining groups show very little increase in mortality rates, ranging from about 1 percent a year among the Ache,

TABLE 3   Model of log mortality hazard ln$h(x)$ **by age**

Group	Age 15–40					Age 40+				
	ά	p	exp(β)	R^2	MRDT	ά	p	exp(β)	R^2	MRDT
Ache	0.0006		0.0113	0.00	1,216.0	0.0564	**	0.0150	0.91	12.3
Hiwi	−0.0157		0.0238	0.30		0.0812	*	0.0137	0.72	8.5
Hadza	−0.0010		0.0081	0.00		0.0621	**	0.0105	0.89	11.2
!Kung	0.0423	*	0.0076	0.85	16.4	0.0696	**	0.0106	0.68	10.0
Agta‡	0.0834	*	0.0064	0.80	5.4	0.0330	#	0.0376	0.61	21.0
Average hunter-gatherer	0.0219		0.0115		31.7	0.0604		0.0175		11.5
Yanomamo-Mucaj	0.0065		0.0125	0.12	106.0	0.0535	***	0.0112	0.96	13.0
Tsimane	0.0136		0.0088	0.50	51.1	0.0856	***	0.0097	0.92	8.1
Yanomamo‡	0.0052		0.0301	0.99	134.1	0.0487	***	0.0263	0.92	14.2
Gainj‡	0.0499	**	0.0111	0.91	13.9	0.0535	***	0.0422	0.99	13.0
Average forager-horticulturalist	0.0188		0.0156		36.9	0.0603		0.0224		11.5
!Kung 1963–74	0.0805	+	0.0011	0.91	8.6	0.0882	**	0.0044	0.80	7.9
Ache reservation	0.0212		0.0018	0.13	32.7	0.0672	***	0.0098	0.99	10.3
Northern Territory Aborigines	0.0301	*	0.0037	0.86	23.1	0.0610	**	0.0038	0.87	11.4
Agta transition	0.0490	*	0.0065	0.75	14.1	0.1110	*	0.0112	0.82	6.2
Agta peasant‡	0.0844	*	0.0050	0.86	8.2	0.0463	**	0.0340	0.91	15.0
Warao	0.0055		0.0119	0.09	126.3	N/A				
Average acculturated HG	0.0451		0.0050		15.4	0.0747		0.0126		9.3
Overall average	0.0304		0.0100		22.8	0.0655		0.0172		10.6

***p<0.0001; **p<0.001; *p<0.01;+p<0.05, #p<0.10
MRDT is mortality rate doubling time, defined here as ln2/ά
In the Gompertz model, ln[$h(x)$]= β + ά* x
‡Parameter estimates for 40+ regression are based on small samples or are affected by age estimation problems (see text).

Yanomamo, and Tsimane to about 4 percent a year among the !Kung. Only the Agta, whose data contain less reliable age control, show greater increases over the 15–40 age range. This may be artifactual, or real and due to high adult mortality. Under normal circumstances, however, traditional humans experience about two decades of prime adulthood in which mortality rates hardly change, then experience a sharply increasing mortality hazard in middle and old age.[3] A distinctive feature of human mortality profiles is a lengthy period of prime adulthood, delayed senescent decline, and extended life span.

Modal ages of adult death

The effective end of the human life course under traditional conditions seems to be just after age 70 years. Following the lead of Kannisto (2001) and Lexis (1878), we evaluate 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 4 and Table 4). Figure

FIGURE 4   Modal ages of adult death
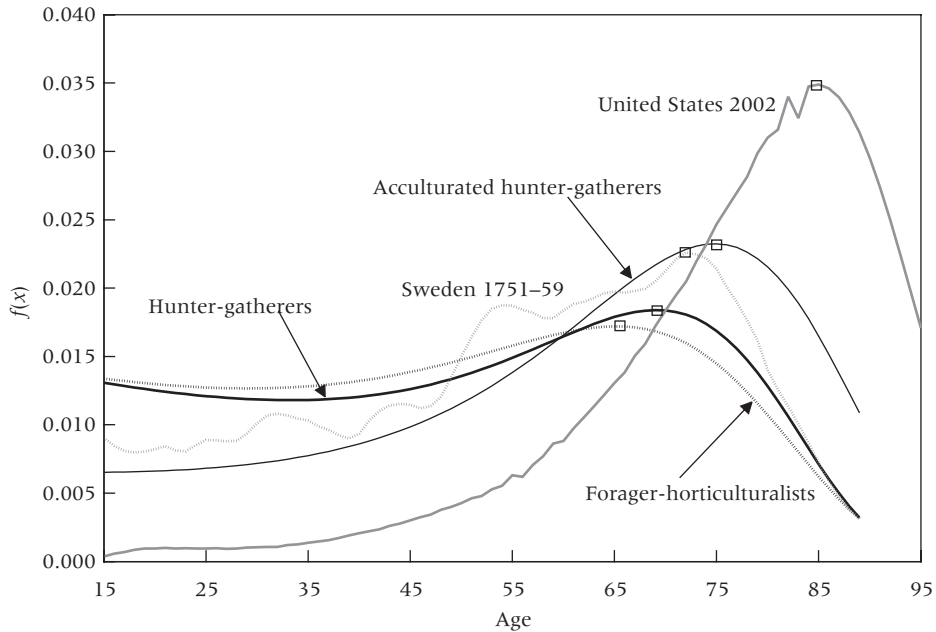
NOTE: Frequency distribution of ages at death *f*(*x*) for individuals over age 15 shows strong peaks for hunter-gatherers, forager-horticulturalists, acculturated hunter-gatherers, Sweden 1751–79, and the United States 2002 (both sexes). All curves except for Sweden and the United States are smoothed using Siler estimates.

4 shows the frequency distribution, *f*(*x*), of deaths at age *x*, conditional upon surviving to age 15, for our composite categories of hunter-gatherers, forager-horticulturalists, and acculturated hunter-gatherers using all populations with high data quality and age specificity. All curves (except those for Sweden and the United States) are based on Siler models. Data from historic Sweden and contemporary United States are shown for comparative purposes. The sample of premodern populations shows an average modal adult life span of about 72 years, with a range of 68–78 years (Table 4).

While modal age at death is not the same as the effective end of the life span, because modal age refers to a peak in the distribution of 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 physical decline such that if they do not die from one cause, they soon die from another. This is consistent with our anecdotal impressions of frailty and work in these 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.

Settled Ache show a modal age at adult death that is 7 years greater than that of pre-contact Ache, consistent with their improved conditions after

TABLE 4   Modal ages at death

Population	Modal age at death	Standard deviation	Percent of adult deaths at mode year	Percent of 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 1963–74	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 1751–59	72	7.4	2.3	24.3
United States 2002	85	1.7	3.5	35.3

NOTE: The extent of variation around the mode is usually defined as four standard deviation units around the mode (Cheung et al. 2005).

settlement. There is much greater variability in the ages of adult death within each of these populations than typically found in modern industrial populations (Cheung et al. 2005), in part because of the higher age-independent mortality (a_2) among the Ache. Lexis (1878) considered such mortality to be different from the "normal" course of aging. For example, re-estimating Hadza mortality without an age-independent mortality component ($a_2=0$) increases the mode from 76 to 78, and decreases the standard deviation around the mode from 6.0 to 5.1. The modes derived from the Siler model are therefore less peaked, accounting for less than 3 percent of adult deaths within the year marking the mode. Of the three study population groups, acculturated hunter-gatherers show the greatest proportion of deaths after age 55, possibly indicating that as some causes of death (such as violence) are reduced, age-related causes of death become more substantial, leading to a greater share of deaths around the mode (Figure 4).

Variation in mortality rates and the effects of changing conditions

The relationship between juvenile and adult mortality rates across populations. Some historical cohort evidence indicates that high infant and child mortality have substantial impact on survivorship later in the life span (Costa 2000; Wilmoth, Vallin, and Caselli 1990). Finch and Crimmins (2004) show that the level of child mortality explains 85 percent of the variance in old-age mortality in cohorts in Sweden and the United States, and that early mortality has a threefold greater predictive effect on old-age mortality for those cohorts than

period measures of early mortality. Figure 5 presents the results of regressing life expectancy at 45 (e_{45}) on survival to age 15 (l_{15}), and compares those results with historical cohort evidence from Denmark (1835–2002) and Sweden (1751–2003) (Berkeley Human Mortality Database). In the range of survival probabilities in the traditional foraging sample, the relationship between juvenile survival and later adult mortality rates is almost the same among foragers as it is for northern Europeans. An additional 15–20 years of life are expected upon reaching age 45.

   The effects of contact and acculturation.   One of the best ways to examine the effects of acculturation on traditional small-scale populations is to compare mortality profiles of the same groups at different time periods. These diachronic comparisons can be made for the Yanomamo, Ache, Agta, Hiwi, and !Kung. As was noted above, demographic data exist on these groups before and after some critical period, be it contact (Yanomamo, Ache) or acculturation and transition to peasant status (Agta and !Kung). Because similar data are available for the Herero, a group of agro-pastoralists in Botswana and Namibia (Pennington and Harpending 1993), we include them in this comparison. Figure 6 shows the ratio of age-specific mortality hazards from more and less acculturated time periods. Figure 6a displays populations whose survivorship has improved with acculturation (!Kung, Ache, Herero, Hiwi) and Figure 6b populations whose condition has worsened (Agta and Yanomamo).

FIGURE 5   Juvenile survivorship and life expectancy at age 45
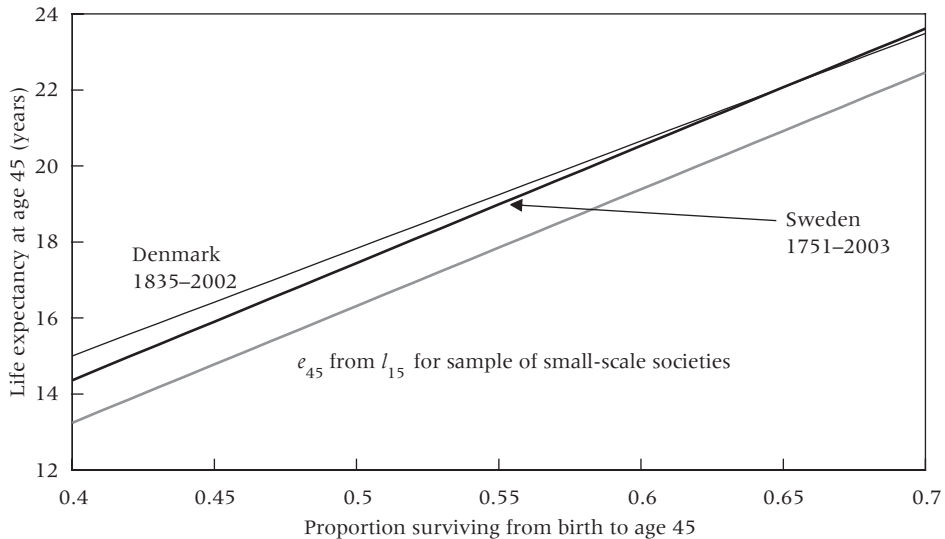
NOTE: Linear regression estimates of life expectancy at age 45 (e_{45}) from survivorship to age 15 (l_{15}).

Relationship shown for our sample of small-scale populations, and for Sweden (using longitudinal cohort data from 1751 to 2003) and Denmark (from 1835 to 2002).

FIGURE 6   Diachronic changes in age-specific mortality patterns within populations

a) improved conditions

b) worse conditions
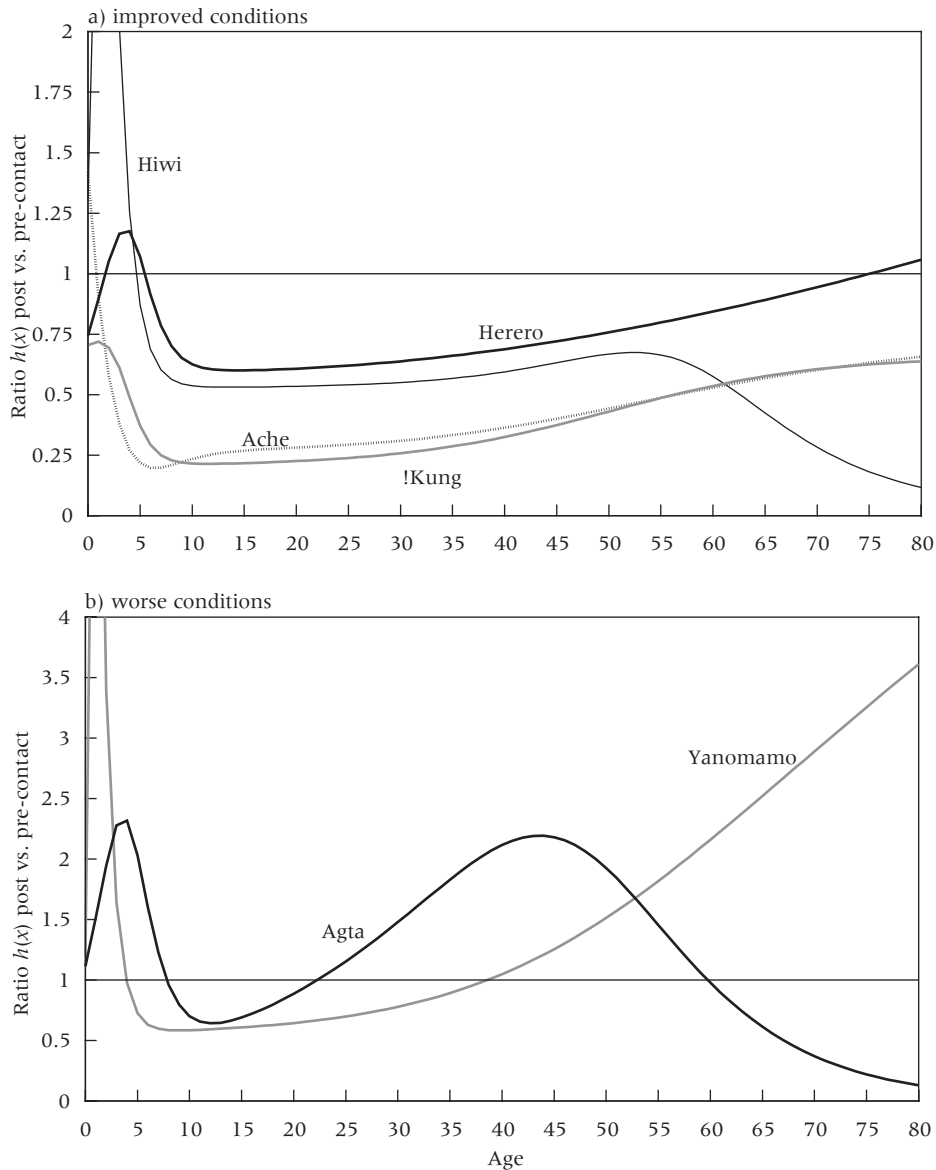
NOTE: The ratio of estimated mortality hazards from post-contact or acculturated time perids and pre-contact or relatively unacculturated time periods from the same populations. Panel (a) shows mortality hazard ratio for populations with improvements in survivorship after contact for much of the life span, while (b) shows those with higher survivorship before contact and interaction with outsiders.

Contact and acculturation had large effects on mortality rates in some groups. Among the Ache, Hill and Hurtado (1996) divide the demography into three periods: pre-contact, contact, and post-contact. The period of contact brought catastrophic diseases to the Ache, about 40 percent of whom

died less than a decade after contact. Here we compare the post-contact with the pre-contact period. After a period of contact, mortality increased during infancy but diminished to about one-fourth to one-third of pre-contact levels for late childhood and adulthood. The effects of improved conditions are greater at younger ages and gradually decline with age. Reduced mortality among settled Ache is largely due to reductions in homicide and forest-related accidents attendant on missionary influence and state intervention (Hill and Hurtado 1996). Medical attention has also helped lower mortality among Ache.

A similar pattern is found for the !Kung, with the exception that survival improves at all ages. Although settled !Kung frequently complain about meat scarcity and changing norms of resource distribution, they also benefit from increased access to milk, protein-rich weaning foods, and a more predictable diet through greater association with cattleposts and receipt of government rations (Harpending and Wandsnider 1982). It is possible, however, that we have overestimated the effects of acculturation on !Kung survivorship because of gaps in the prospective life table created by Howell (1979: Table 4.6).[4]

Among the Hiwi, contact has greatly increased infant mortality, but decreased mortality after infancy by about a half; data on the very old are insufficient to ascertain what happens after age 60. Among the Herero, there is a small increase in childhood mortality, but an improvement at older ages. The initially higher level of survival among the Herero probably accounts for the smaller effect of acculturation on mortality rates.

Two groups appear to show deleterious effects of contact. Early child and adult mortality are much higher among "acculturated peasant" Agta and Xilixana Yanomamo. Infant mortality may be buffered by protective effects of breastfeeding, and so post-weaning mortality seems to worsen more in acculturated settings among both Agta and Yanomamo. Peasant Agta are landless agriculturalists living in more populated and degraded environments with few foraging options, and they no longer maintain close trading relationships with nearby horticulturalists (Early and Headland 1998). Early and Headland suggest that cumulative effects of malnutrition and infectious diseases such as measles have increased child mortality during the peasant phase, and they verify this through comparison of postneonatal and neonatal mortality rates. Additionally, malaria, tuberculosis, and other infectious diseases were believed to be largely absent in the forager phase among both Agta and Yanomamo but have reached epidemic proportions in the past 30 years. Lower adult mortality among Yanomamo may be due to reduced warfare and homicide in recent years (Early and Peters 2000). It is unclear why the negative impact of infectious disease appears to affect adults over age 40 among Yanomamo whereas it affects reproductive-age adults among Agta. One possibility is that susceptibility attributable to differential prior exposure may vary by age groups.

Studies that have focused on the effects of sedentism on mortality show a general decrease in child mortality, consistent with our description of the experience of !Kung, Herero, and Ache. These include the Ghanzi !Kung (Harpending and Wandsnider 1982), Kutchin Athapaskans (Roth 1981), Turkana pastoralists (Brainard 1986), and Nunamiut (Binford and Chasko 1976). On the other hand, a slight increase in child mortality was observed among the Adavasi Juang agriculturalists of India (Roth and Ray 1985). Fertility increases among members of all of these populations, and often does so before a noticeable decline in mortality.

Acculturation in the past several decades is likely to be very different from the transitions to sedentary agricultural or peasant life in the more remote past. Even when foragers become the new underclass of national society and foraging behavior becomes rarer, recent post-contact recovery periods are often accompanied by immunization campaigns and public health and sanitation measures that can substantially improve survivorship. It is likely that a worsening of physical health, or at best a lack of improvement, occurs when these benefits are absent or unavailable.

To put these changes in perspective, Figure 7 shows the ratio of the average hunter-gatherer mortality hazards to US hazards in 2002. Infant mortality is over 30 times greater among hunter-gatherers, and early child mortality is over 100 times greater than encountered in the United States. Even late childhood mortality is about 80 times greater among hunter-gath-

FIGURE 7   Two extremes in human mortality hazards: Ratio of Siler-estimated mortality hazards (h_x) for hunter-gatherer composite sample and the United States 2002
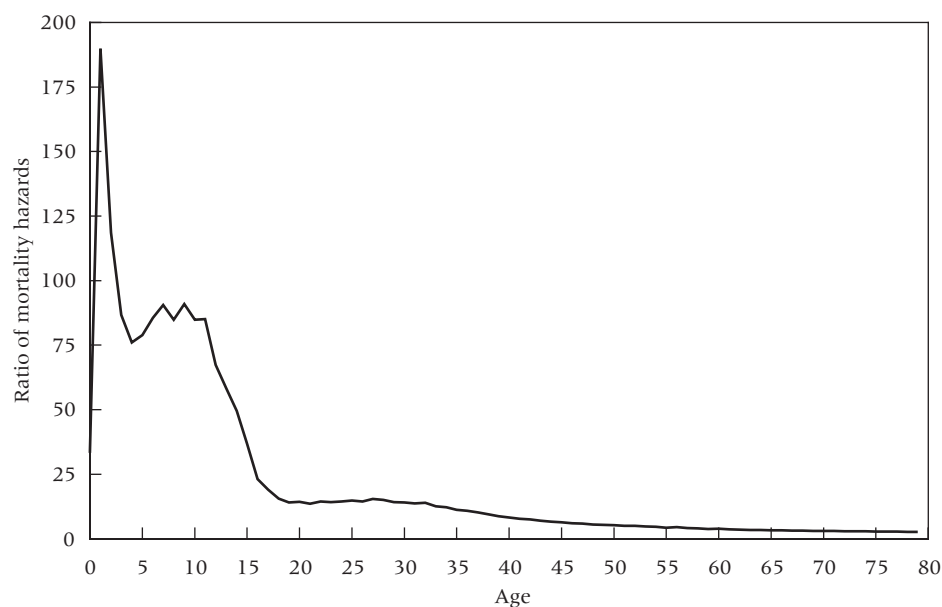
erers. Not until the late teens does the relationship flatten, with over a tenfold difference in mortality. This difference is only fivefold by age 50, fourfold by age 60, and threefold by age 70.

Causes of mortality. It is important to investigate causes of death among groups and at different ages to ascertain whether mortality patterns are the result of unique circumstances and history, ecological conditions, or genetic predispositions. We have seen that several of the study groups showed erratic mortality profiles that highlight the need for further examination, even after taking into account discrepancies in methodology. Many causes of death are difficult to assess in traditional populations without physicians or autopsies, and especially when causes of death are elicited during retrospective interviews. Even in modern hospitals, determining cause of death is sometimes elusive or ambiguous because of multiple levels of causation. Malnutrition, infections, and disease are common in our study populations, making designation of the primary cause of death problematic, if not inevitably incomplete. Accidents and homicides are usually easier to identify, and estimates of these causes should be less subject to error. Table 5 presents causes of death among the populations for which such data exist. It also includes published causes of death for Aka Pygmies of the Congo (Hewlett, van de Koppel, and van de Koppel 1986) and Bakairi of Brazil (Picchi 1994). The table shows the overall percentage of deaths attributed to illness, degenerative disease, and accidents or violence. Our sample includes 3,328 deaths. Where age-specific data exist, cause of death is listed for juveniles (<15), adults (15–59), and older adults (60+). Illness accounts for over half of the deaths in all groups except among pre-contact Ache. Among juveniles and adults, illness accounts for the majority of all deaths, again except among Ache, where rates of female-biased infanticide and adult homicide are fairly high. Among older adults, degenerative disease and accidents compete with illness as major contributors of death.

We subdivide illness into respiratory, gastrointestinal, fever, and other illnesses (sensu Hill and Hurtado 1996; Howell 1979). Gastrointestinal illnesses account for 5–18 percent of deaths. Such deaths are higher among Tsimane than Ache, Yanomamo, and Bakairi, even though the latter three groups show high rates of parasitism in tropical environments. This higher rate is likely explained by the younger age structure of the Tsimane. Diarrhea coupled with malnutrition remains one of the most significant causes of infant and early child deaths among foragers and peasant populations. People living in tropical forests are especially vulnerable to helminthic parasites (Dunn 1968), which, although not usually lethal, can compromise growth and immune function.

Twenty percent or more of illness-related deaths among these groups are due to such respiratory-related illnesses as bronchitis, tuberculosis, pneumonias, and other viral infections. Among many South American Amazo-

TABLE 5 Causes of death among study populations (in percent)

	Hadza	Yano-mamo	Ache forest	Ache settled	!Kung	Tsi-mane	Aka	Agta	Hiwi	Machi-guenga	Northern Territory Aborigines	Bakairi	Gainj	Total Number	Total Percent
a) <15 yrs old		(111)	(230)	(84)	(164)	(423)		(112)	(94)	(82)	(74)				
all illness		21.6	22.2	65.5	87.8	79.9		95.5	44.8	63.8	67.6			825	65.3
degenerative		5.4	8.3	20.2	3.7	10.4			10.4	9.6	24.3			120	9.5
accidents		6.3	6.1	3.6	8.5	9.7		1.8	15.6	11.7	6.8			102	8.1
violence		40.5	63.5	10.7		7.4		2.7	27.1	2.1	1.4			216	17.1
b) 15–59 yrs old			(125)	(22)	(127)	(192)		(77)	(31)	(19)	(68)				
all illness			28.0	86.4	79.5	74.7		69.5	35.3	33.3	61.8			400	60.5
degenerative			3.2		3.1	16.5		4.9	2.9	14.3	25.0			61	9.2
accidents			23.2	13.6		8.8		4.9	8.8	42.9	0.0			85	12.9
violence			45.6		17.3	12.9		14.6	44.1	0.0	13.2			115	17.4
c) 60+ yrs old			(27)		(52)	(60)				(2)	(33)				
all illness			18.5		51.9	66.1					72.7			95	54.6
degenerative			22.2		40.4	25.4					21.2			49	28.2
accidents			25.9			8.5					6.1			18	10.3
violence			33.3		7.7	1.7				100.0	0.0			12	6.9
d) All ages	(125)		(382)	(104)	(343)	(690)	(669)	(364)	(139)	(117)	(175)	(65)	(44)		
respiratory			0.8	31.1		19.9				6.8	28.6	56.9		292	23.7[a]
gastrointestinal			5.5	13.2		18.2				34.2	17.1	3.1		239	13.8[a]
fever			8.1	21.7		5.7				1.7	0.0	7.7		107	7.3[a]
other			9.4	1.9		25.9				14.5	20.6	3.1		317	16.6[a]
All illness	**66.7**	**73.9**	**23.8**	**67.9**	**79.3**	**69.6**	**92.2**	**86.7**	**41.0**	**57.3**	**66.3**	**70.8**	**79.0**	**2,333**	**70.1**
Degenerative	**12.0**	**6.3**	**7.6**	**16.0**	**9.0**	**12.2**	**2.5**	**7.6**	**7.9**	**10.3**	**24.0**	**16.9**	**7.0**	**306**	**9.2**
Accidental	0.8	7.2	13.1	2.8		8.4		2.7	12.9	17.1	4.0	12.3		166	8.1[a]
Homicide	3.2	4.5	22.0	4.2		7.5				3.4	5.7	0.0		164	6.3[a]
Warfare	0.0	8.1	33.5	0.0		0.0				0.0	0.0	0.0		137	5.2[a]
All violence	3.2	12.6	55.5	4.2		7.5		3.0	30.2	3.4	5.7	0.0		354	12.5[a]
All violence/ accidental	**4.0**	**19.8**	**68.6**	**7.1**	**11.7**	**15.9**	**5.4**	**5.8**	**43.2**	**20.5**	**9.7**	**12.3**	**14.0**	**626**	**18.8**
Other causes	17.3	0.0	0.0	0.0	0.0	2.2	0.0	0.0	7.9	12.0	0.0	0.0	0.0	62	1.9

NOTE: Numbers in parentheses are number of persons in relevant age group.
[a]Category average includes only groups for which good quality data are available; unknown deaths for Hadza, Agta, and Aka are distributed among illness categories. Unknown deaths for Bakairi and Tsimane are omitted from the analyses.
SOURCES: See Table 1.

nian groups, Black (1975) reports that most infectious diseases are absent in newly contacted groups, because small, mobile populations cannot support these contagious vectors. Post-contact prevalence of infectious disease among Ache is similar to rates among Yanomamo (Hill and Hurtado 1996). Tsimane show a similar importance of respiratory disease at 20 percent of illness-related deaths. Bakairi have suffered repeated epidemics of respiratory illness, especially tuberculosis and whooping cough (Picchi 1994). Pneumonia and tuberculosis are claimed to be the top killers of adults and older children among !Kung (Howell 1979: 63). Gainj mortality and more recent mortality among Agta also show high rates of infectious disease.

Degenerative deaths are relatively few, confined largely to problems early in infancy and late-age cerebrovascular problems, as well as attributions of "old age" in the absence of obvious symptoms or pathology. Heart attacks and strokes appear rare and do not account for these old-age deaths (see Eaton, Konner, and Shostak 1988), which tend to occur when sleeping. It has often been remarked that few risk factors for cardiovascular disease exist among active members of small-scale societies (Eaton et al. 1994), although compromised lung or kidney functioning can interact with cardiac fibrosis or moderate arterial stenosis to cause cardiac arrest. Obesity is rare, hypertension is low, cholesterol and triglyceride levels are low, and maximal oxygen uptake is high. Overall, degenerative disease accounts for 6–24 percent (average 9 percent) of deaths, with the highest representation among Northern Territory Aborigines. Neoplasms and heart disease each accounted for 9 of the 42 deaths due to degenerative illness. 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. To our knowledge there have been no focused studies or mention of Alzheimer's, Parkinson's, or other forms of dementia.

Violence and warfare are variable across groups. Agta, Ache, Yanomamo, and Hiwi suffer from high levels of homicide, affecting adult males disproportionately. Homicide is low among Hadza, Tsimane, and Northern Territory Aborigines. Ache display a very high level of homicide, although much of this is infanticide, child homicide, and a result of skirmishes with rural Paraguayans. Infanticide is fairly high among Ache and Yanomamo, occasional among !Kung and Tsimane, and rare among Hadza. Infants most susceptible to infanticide include those born with obvious defects, those perceived as weak, twins, and those of questionable paternity. It seems likely that violent deaths decrease with increased state-level intervention and missionary influence in many small-scale groups around the world (e.g., Agta, Ache, Aborigines, !Kung, Yanomamo). The composition of accidental deaths varies across groups, including falls, river drownings, animal predation, accidental poisonings, burns, and getting lost in the environment. Only one Hadza death (1 percent) was accidental, with remaining groups showing

4–13 percent of deaths as unintentional or accidental. Together, accidental and violent deaths account for 4–69 percent (average 19 percent) of all deaths. The high proportion of Ache deaths due to violence is a consequence of few illness-related deaths in the pre-contact sample. The post-contact Ache sample shows only 14 percent violent and accidental deaths, with 68 percent of deaths due to illness.

The sample of deaths for Agta, pre-contact Xilixana Yanomamo, and Gainj is small. Early and Headland (1998) and Early and Peters (2000) surmise that a large percentage of unknown deaths among Agta is due to infectious disease. Among Gainj, most adult deaths are due to infectious disease. The very high adult mortality among Agta results from a combination of maternal mortality, homicide, and infectious disease. The rates of Agta death for maternal mortality and homicide are the highest reported for any population in the world.

Traditional humans, chimpanzees, and paleodemography

Figure 8 examines five mortality ratios as a function of age: 1) traditional to acculturated hunter-gatherers, 2) wild to captive chimpanzees, 3) wild chimpanzees to traditional foragers, 4) captive chimpanzees to traditional foragers, and 5) estimates for prehistoric populations to traditional foragers. The first two comparisons reflect the effects of changed conditions. The next two comparisons allow for the assessment of the interaction of changed conditions and species differences. The last combines the effects of errors in estimation and changes in mortality rates over time.

The effects of improved conditions have similar within-species effects on humans and chimpanzees. Captivity among chimpanzees, which provides medical attention, abundant feeding, and protection from predation, exerts a very large effect on infant and juvenile survival rates. The effects of captivity diminish with age. Among traditional humans, the effects of improved conditions seem to be greatest during childhood and middle adulthood, tapering off with age.

The age-specific differences between humans and chimpanzees are revealing. The difference between foragers and wild chimpanzees increases dramatically with age. The ratio of mortality hazards is over three times as high among chimpanzees during childhood, and then increases to about eight times as high at age 45. Improved conditions for captive chimpanzees actually generate lower mortality during infancy and early childhood than among some foragers. However, the species differences then overcome the effects of conditions, with captive chimpanzee mortality rates being over three times as high as that of foragers by age 40.

FIGURE 8   Ratio of mortality hazards for humans and chimpanzees
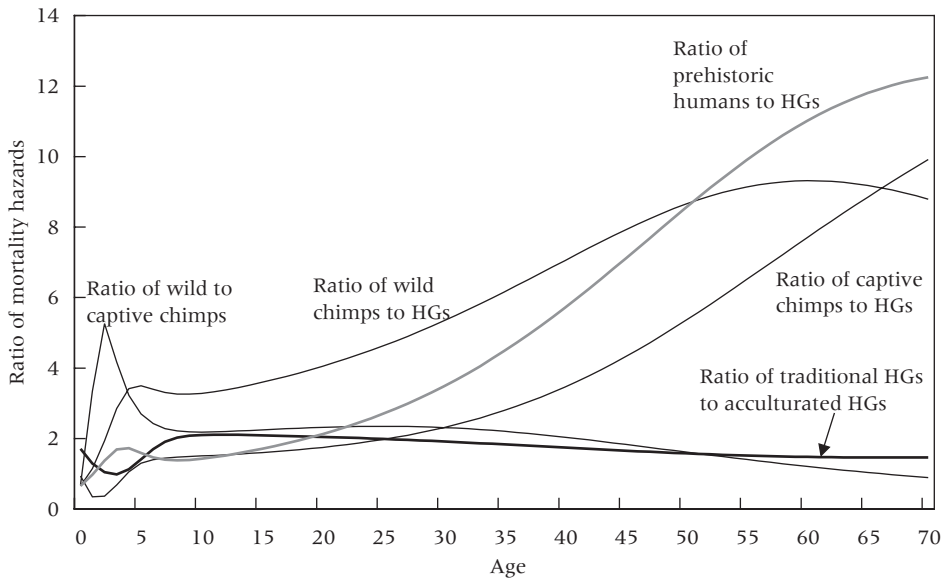
NOTE: Comparisons in mortality based on hazard ratios for unacculturated and acculturated hunter-gatherers, wild and captive chimpanzees, wild chimps and hunter-gatherers, captive chimps and hunter-gatherers, and prehistoric humans and hunter-gatherers. See text for description of samples.
SOURCES: Wild chimpanzees (Siler estimated based on Hill et al. 2001); captive chimpanzees (Siler estimated based on Dyke et al. 1995); prehistoric humans (Gage 2002).

It is usually reported that Paleolithic humans had life expectancies of 15–20 years and that this brief life span persisted over thousands of generations (Cutler 1975; Weiss 1981) until early agriculture less than 10,000 years ago caused appreciable increases to about 25 years. Several prehistoric life tables support this trend, such as those for the Libben site in Ohio (Lovejoy et al. 1977), Indian Knoll in Kentucky (Herrmann and Konigsberg 2002), and Carlston Annis in Kentucky (Mensforth 1990). Gage (1998) has compiled a set of reconstructed prehistoric life tables with similar life expectancies and computed Siler estimates for a composite prehistoric mortality profile. This and most other prehistoric profiles show l_{50} of 2–9 percent and e_{45} values of 3–7 years. There is a large paleodemographic literature concerning problems of age estimation in skeletal samples and bias in bone preservation among older individuals (see Buikstra and Konigsberg 1985; Buikstra 1997; Walker, Johnson, and Lambert 1988; Hoppa and Vaupel 2002). Howell (1976) has identified many problems with prehistoric life tables. This literature is too large to discuss here and we direct readers to recent treatments by O'Connell et al. (1999), Kennedy (2003), and Konigsberg and Herrmann (2006).

The comparison of Siler estimates of prehistoric humans with those for traditional modern foragers poses a further challenge to the historical data (consistent with the criticisms discussed above). Mortality rates in prehistoric

populations are estimated to be lower than those for traditional foragers until about age 2 years. Estimated mortality rates then increase dramatically for prehistoric populations, so that by age 45 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 primary cause of death. While excessive warfare could explain the shape of one or more of these typical 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 may be possible to use the data from modern foragers to adjust those estimates for prehistoric foragers. Longitudinal analyses and cross-cultural comparisons have shown that in the Gompertz–Makeham model, the overall rate of mortality is negatively associated with the rate of mortality increase with age. Strehler and Mildvan (1960) referred to this relationship as the *compensation law of mortality*, where high initial adult mortality is offset by lower values of the mortality slope. Their argument assumes a decline in "vitality" with age and an inverse relationship between the frequency of environmental stresses and the intensity of those stresses on the body. Mortality compensation implies that differences in mortality rates among populations should decrease with age and converge on a common species-typical pattern of senescence (Gavrilov and Gavrilova 1991; Yashin et al. 2001). While the theory underlying mortality compensation is controversial, the robust inverse relationship between the two Gompertz parameters is not. In the Siler formulation, the intercept and slope parameters are a_3 and b_3, respectively. In the forager dataset, the best-fit equation for the relationship between $\ln a_3$ and b_3 is:

$$\ln a_3 = -64.983 b_3 - 2.9888 \tag{3}$$

This relationship is highly significant ($r=0.994$, $p<0.001$). Inserting (3) into the nonimmature components of (1) yields

$$h(x) = a_2 + 0.050 \cdot e^{b_3(x-64.983)} \tag{4}$$

Equation (3) describes the set of mortality curves that intersect by about age 65 and explains 89 percent of the variation in these parameters ($p<0.0001$). Thus in our sample, mortality is estimated to converge at around 65 years of age, some 20 years earlier than in modern industrialized populations (e.g., Riggs 1990; Riggs and Millecchia 1992; Riggs, Hobbs, and Gerald 1998).

The Siler estimates of a_3 and b_3 for the prehistoric composite life table provided by Gage (1998) are extreme outliers from the regression equation (3). The average mortality profile for our forager populations is similar to that for Sweden in 1751, and early European populations and foraging populations show similar negative relationships between a_3 and b_3. If we fix a_3 and solve for the appropriate b_3 based on equation (3), we find that b_3 should be 0.038, or one-half of the estimate by Gage. Calculation of the life table with this revised value of b_3 changes l_{50} from 4 percent to 24 percent and e_{45} from 6 years to 19 years. Overall life expectancy only changes from 20 to 28 years. Alternatively, if we fix b_3 and instead recalculate a_3, we find that an a_3 consistent with the linear relationship of equation (4) is 1/12 of Gage's estimate. A life table with this revised a_3 estimate shifts l_{50} to 36 percent, e_{45} to 18 years, and mean life expectancy to 33 years.

Critics may argue that the uniformitarian mortality assumption does not apply to those prehistoric populations that tend to show relatively low infant and child mortality but excessively high adult mortality. If not based on erroneous assumptions about population growth and model life tables, such adult mortality would presumably be due to violence and warfare and not to widespread infectious disease or intrinsic senescence. Infectious disease is unlikely to reach epidemic proportions in small, isolated populations (Black 1975). In our sample, widespread lethal infectious disease was most common in groups that had been interacting with large populations of outsiders. Intrinsic senescence is also unlikely to have been much higher in the past because evidence also suggests that early and late-age mortality are closely linked among cohorts as a result of the cascading effects of early infections and undernutrition (Kuh and Ben-Shlomo 1997; Costa 2000; Finch and Crimmins 2004).

Making inferences about population growth in the past by reference to modern foragers

One of the criticisms of making inferences about ancestral populations from the study of ethnographic foragers is that the high population growth rates of some modern groups could not possibly be representative of ancestral foragers for long periods of time. Figure 9 displays "iso-growth" curves, which are lines of equal population growth as a function of different levels of the total fertility rate (TFR) and survivorship to age 15 (l_{15}).[5] Defining curves in terms of these two variables is useful because TFR and l_{15} exist for many more groups than our sample of societies used here. Other foraging groups included here are the Chenchu, Mbuti, Aka, Batak, and Greenland Inuit. Additional horticulturalists include the Bari, Dusun, Kapauku, Ngbaka, Semai, Talensi, Tamang, and 23 South American Amazonian groups. Additional pastoralists include the Datoga, Kipsigis, and Sebei. These groups were largely compiled from Hewlett (1991) and Salzano and Callegari-Jacques (1988). Although

FIGURE 9   Iso-growth curves: Iso-clines of equal population growth rates are shown as a function of total fertility rate (TFR) and survivorship to age 15 (l_{15})
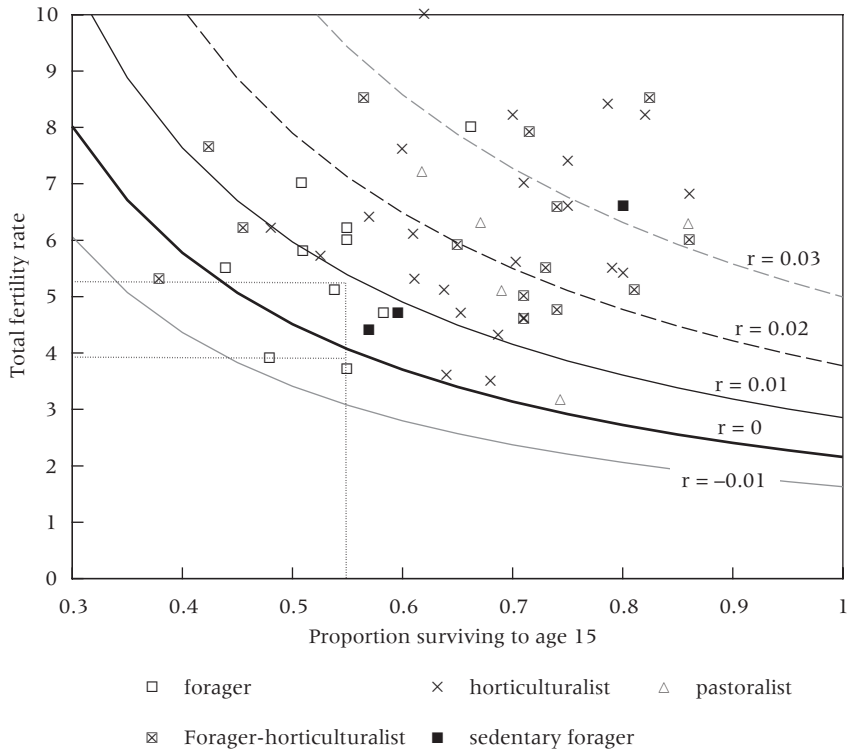
NOTE: Each data point refers to a single population.

forager mortality is narrowly confined, fertility ranges widely from below 4 to as high as 8 children per woman. At the forager average l_{15} of 0.55, population growth is zero at a TFR of about 4. At the forager average TFR of 5.7, population growth is zero at an l_{15} of 0.41. The average observed forager population shows a growth rate of about 1 percent per year. Although several groups have fertility and mortality levels implying zero or negative population growth, the majority of small-scale foraging populations show positive growth. Forager-horticultural and horticultural populations both have an average l_{15} of about 0.67 and TFR of 6.2. Their implied average growth rate thus exceeds 2 percent. Pastoralists have an average l_{15} of 0.72 and TFR of 5.6, indicating an average growth rate also above 2 percent.

Of the foraging populations, just under half show population growth rates near zero (two are just below replacement and three just above), and the remainder show higher population growth rates. At least two explanations of these results are possible. One possibility is that past population growth rates were close to zero and that current positive growth rates in some groups

are unrepresentative of past populations. Levels of homicide and intergroup warfare may have been greater in the past. Also, some groups may have had greater access to food during the sample period than in the past. For example, the northern Ache experienced high growth rates throughout the twentieth century, and the depopulation of Guarani horticultural groups in their area, caused by slave trading and warfare, may have been a factor. The southern Ache, in contrast, were on the verge of extinction because of increasing encroachment on their traditional range by outsiders. As illustrated in Figure 9, zero population growth requires either a significant decrease in fertility below that observed in almost all foraging groups, or a decrease in survivorship below that ever observed among modern or prehistoric foraging populations. Even with the inclusion of an additional nine values of l_{15} from other foraging populations, mean survivorship drops only to 0.53 (s.d.=0.07).[6] This probably means that the fertility of sample populations would have to have been closer to 4.5 in order for population growth to remain at zero.

A second possibility is that although long-term population growth must have been nearly zero over long periods of time, population dynamics over shorter time periods resembled a "saw-tooth" pattern (Hill and Hurtado 1996), characterized by rapid population increases and subsequent crashes. Most time is therefore spent in a growing state, consistent with the high growth rates seen in many ethnographic reports on hunter-gatherers. Binford (personal communication) recounts the case of an Nunamiut Eskimo group that perished in its entirety, having been snowed in without sufficient food supplies to survive through the winter.

It is difficult to evaluate these two hypotheses in light of current knowledge about past conditions. While evidence suggests that climate varied widely throughout the Pleistocene and into the Holocene epoch (see Richerson, Bettinger, and Boyd 2005), the extent to which past foragers typically experienced increasing, declining, or zero growth in past environments is unknown. Perhaps some combination of the two hypotheses is the correct answer. Mortality experiences of infants and children and adult mortality due to violence and trauma probably varied significantly over time and place.

Summary of results

We summarize our main findings to this point:

Post-reproductive longevity is a robust feature of hunter-gatherers and of the life cycle of *Homo sapiens*. Survivorship to grandparental age is achieved by over two-thirds of people who reach sexual maturity and can last an average of 20 years.

Adult mortality appears to be characterized by two stages. Mortality rates remain stable and fairly low at around 1 percent per year from the age of maturity until around age 40. After age 40, the rate of mortality increase is exponential (Gompertz) with a mortality rate doubling time of about 6–9

years. The two decades without detectable senescence in early and mid-adult-hood appear to be an important component of human life span extension.

The average modal age of adult death for hunter-gatherers is 72 with a range of 68–78 years. This range appears to be the closest functional equivalent of an "adaptive" human life span.

Departures from this general pattern in published estimates of life expectancy in past populations (e.g., low child and high adult mortality) are most likely due to a combination of high levels of contact-related infectious disease, excessive violence or homicide, and methodological problems that lead to poor age estimates of older individuals and inappropriate use of model life tables for deriving demographic estimates.

Illnesses account for 70 percent, violence and accidents for 20 percent, and degenerative diseases for 9 percent of all deaths in our sample. Illnesses largely include infectious and gastrointestinal disease, although less than half of all deaths in our sample are from contact-related disease.

Comparisons among hunter-gatherers, acculturated hunter-gatherers, wild chimpanzees, and captive chimpanzees illustrate the interaction of improved conditions and species differences. Within species, improved conditions tend to decrease mortality rates at all ages, with a diminishing effect at older ages. Human and chimpanzee mortality diverge dramatically at older ages, revealing selection for a longer adult period in humans.

Discussion and conclusions: The evolved human life span

A fundamental conclusion we draw from this analysis is that extensive longevity appears to be a novel feature of *Homo sapiens*. Our results contradict Vallois's (1961: 222) claim that among early humans, "few individuals passed forty years, and it is only quite exceptionally that any passed fifty," and the more traditional Hobbesian view of a nasty, brutish, and short human life (see also King and Jukes 1969; Weiss 1981). The data show that modal adult life span is 68–78 years, and that it was not uncommon for individuals to reach these ages, suggesting that inferences based on paleodemographic reconstruction are unreliable. One recent study that avoids several common problems of skeletal aging used dental-wear seriation and relative macro-age categories (ratio of old to young) to demonstrate an increase in the relative presence of older adults from australopithecines to early *Homo* and, more strikingly, among Upper Paleolithic humans (Caspari and Lee 2004; but see Hawkes and O'Connell 2005). More compellingly, a recent re-estimation of several common paleo-mortality curves based on hazard analysis and maximum likelihood methods shows a life course pattern similar to that of our ethnographic sample (Konigsberg and Herrmann 2006).

We conclude the article with a brief discussion of life span evolution of the hunter-gatherers in our sample. Two issues are addressed. The first

is why modal life spans are about seven decades, not shorter or longer. The second issue is how best to conceive of life span evolution, especially in relation to the gene–environment interactions that determine age distributions of death at the population level.

Why seven decades?

The classic theory of senescence in evolutionary biology argues that individuals contribute less to reproductive fitness as they age because less of their expected lifetime fertility remains (Haldane 1942; Medawar 1952; Williams 1957; Hamilton 1966). Consequently, natural selection acts more weakly to counteract deleterious mutations affecting mortality at older ages. The existence of substantial post-reproductive life among humans therefore suggests that older individuals maintain "reproductive value" by increasing their fitness through non-reproductive means, a critical component of all evolutionary models of human longevity.

George Williams (1957) was first to propose that beginning at ages 45–50 years, mothers may benefit more from investing their energy and resources in existing descendants than from producing new ones. This idea later became known as the "grandmother hypothesis" (Hill and Hurtado 1991). A specific version of the grandmother hypothesis proposed by Hawkes et al. (1998) focuses on intergenerational transfers among women and proposes that older women can increase their inclusive fitness by raising offspring fertility and grandoffspring survivorship through provisioning. The resources acquired by women foragers are strength-intensive, disadvantaging young children while increasing the value of older women's 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. Peccei (2001) proposes an amendment to this view. She points out that long-term juvenile dependence among humans implies that adults who cease reproducing in their 40s will not finish parenting until they are 60 or older (see also Lancaster and King 1985). The notion that most of the benefits to longevity derive from helping offspring rather than grandoffspring has been called the "mother hypothesis" (see Packer et al. 1998 for a similar argument based on baboons).

The embodied capital model (Kaplan et al. 2000; Kaplan and Robson 2002; Gurven and Kaplan 2006) extends these ideas to both sexes in a formal life-history model, relating intergenerational transfers to the ecology of the hunting and gathering lifestyle. It proposes that timing of life events is best understood as an "embodied capital" investment process. Embodied capital is organized somatic tissue, which functionally increases lifetime adult income and 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 humans consume. Those abilities require a large brain and a long time-commitment to development. This extended learning phase, during which productivity is low, is compensated for by higher productivity during adulthood. 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. According to this model, the long human life span co-evolved with the lengthening of the juvenile period, increased brain capacities for information processing and storage, and intergenerational resource flows. It is a two-sex model, as it proposes that both men and women engage in learning-intensive food production tasks; these activities result in delayed productivity until older ages, selecting for life span extension in both sexes.

Each of these models relates the benefits of longevity to fitness effects achieved through transfers to dependent descendants (see Lee 2003). Indeed, an increasing number of studies focusing on the contributions of older individuals have found significant fitness effects via increased child and grandchild survivorship and fertility (e.g., Beise and Voland 2002; Jamison et al. 2002; Sear, Mace, and McGregor 2000; Sear et al. 2002; Hawkes et al. 1998; Lahdenpera et al. 2004). The demographic data derived from foragers and forager-horticulturalists allow us to assess how those contributions can change with age. The two panels of Figure 10, 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 lower panel shows age-specific mortality rates. Even though a woman still has descendants who could benefit from her assistance, the number of offspring and grandoffspring, especially dependents less than 15 years old, drops considerably after about age 65. This is also the point when mortality begins to rise steeply. The late age at which decline in contributions to dependents begins is similar to the modal age at death from Figure 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. Those results provide the greatest support for the embodied capital hypothesis.

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. Data on males would show a similar pattern, except that the male peak in dependency is 3 to 5 years later, due to their later age at marriage. This decline in the poten-

FIGURE 10   **Age-specific dependency and adult mortality**
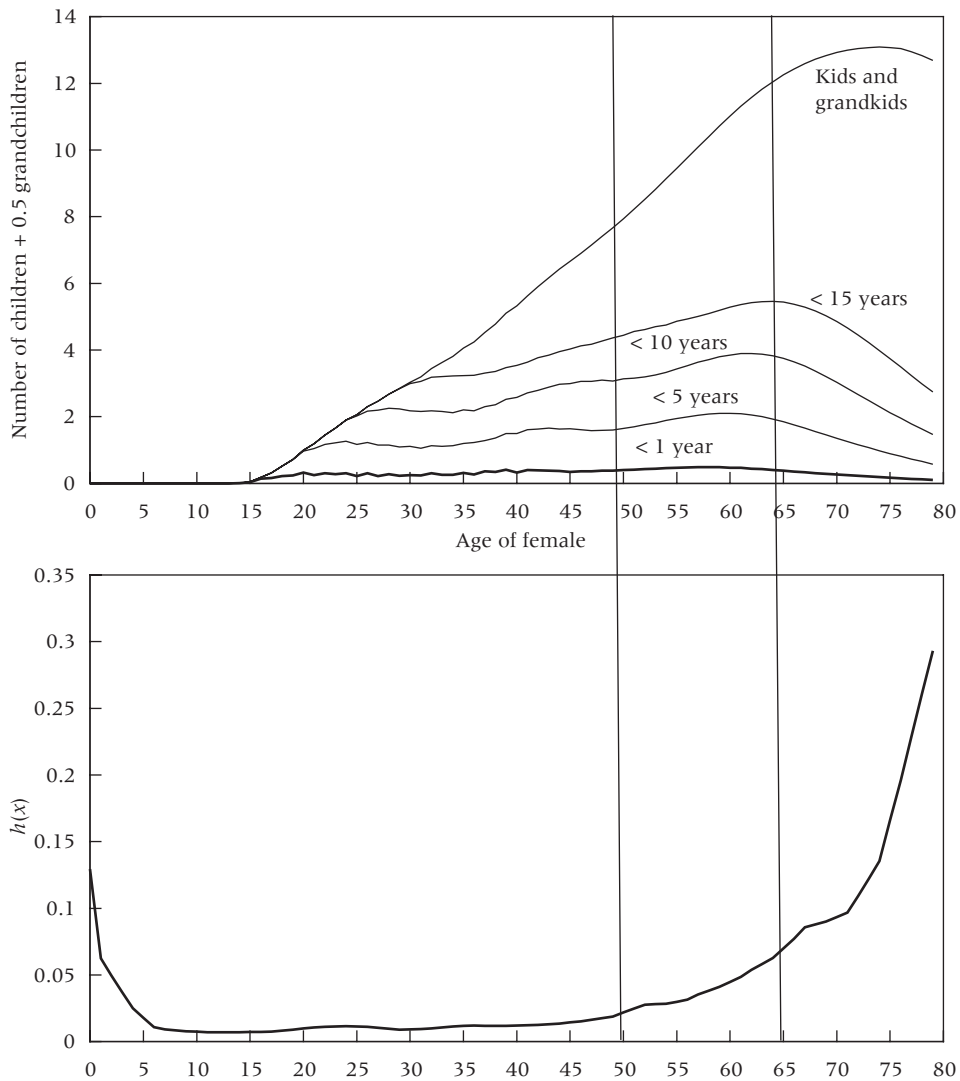
NOTE: Number of children and 1/2 number of grandchildren by age of a Tsimane woman (top panel) compared with age-specific mortality rate for Tsimane (bottom panel).

tial pool of genetic beneficiaries may explain why few people lived beyond the seventh decade of life.

Gene–environment interactions and life span evolution

How can we reconcile the idea of an evolved life span with the evidence that mortality distributions are influenced by environmental conditions? Our answer may shed light on the recent debate over modern changes in hu-

man life span. While average life expectancy has changed significantly over recent history, it is an open question whether gains will continue linearly and whether the maximum life span itself will continue to increase (Vaupel 1997; Wilmoth 1998).

One view is that there is a fixed upper limit to the human life span at about 85±6 years (Wood et al. 1994), where the distribution of deaths becomes compressed or "rectangularized" over time as improved medical care and public health increasingly reduce early-age death (Fries 1989; Weiss 1989). An alternative view posits that there is no set limit to the human life span and that improvements in medical care, treatments, and living conditions will continue to produce increases in longevity (Wilmoth 1998). Wood et al. (1994) characterize these two views as gerontological versus epidemiological. The gerontological view treats the various outcomes of aging as resulting from a single unitary process, leading to programmed death; the epidemiological view treats aging and its implications for mortality as resulting from a competing set of risks due to a set of independent or quasi-independent processes (see also Gage 1989). For example, Wood et al. argue against the notion of a unifying species-typical mortality rate doubling time (cf. Finch, Pike, and Whitten 1990), citing MRDT estimates varying from 3 to 33 years for different causes of senescent death, such as atherosclerosis, emphysema, and cirrhosis.

We suggest that neither view is fully correct and that a hybrid of the two approaches is more productive. It is necessary to consider not only the benefits of life span extension, but also the costs. Aging can be thought of as resulting from the combined effects of environmental assaults and the deleterious byproducts of metabolism. Natural selection favors optimal effort at repairing the damage produced by both processes. In each physiological subsystem, repair and damage prevention should evolve in response to their marginal and partially separable effects on age-specific fitness. 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 upon a complex of long-term child dependence, during which learning outweighed productivity, and compensating high productivity of adults, especially in middle age. Together, the costs of slowing senescence and 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 comparison of data from eighteenth-century Sweden to the hunting and gathering populations in our study suggests that similar age distributions of adult deaths occur under a broad range of environmental conditions.

Reductions in infectious diseases and improvements in food supply sharply lower the assault rate on people's bodies as modernization occurs.

Aging individuals are increasingly insulated from assaults as well. The defenses that evolved to be phenotypically plastic in relation to ancestral environmental variation produce a very different distribution of deaths under modern conditions. In that sense, the evolved human life span is perhaps best conceived as a population-level distribution of deaths that corresponds to the characteristic range of environments in which our ancestors lived.

We do not fully understand the mechanisms underlying the effects of modernization. Do members of industrialized countries age more slowly, in a physiological sense, than people exposed to greater environmental assaults? Alternatively, are most of the mortality improvements attributable 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 are accelerated in response to poor nutrition, infectious disease, and chronic inflammatory processes in general (Elo and Preston 1992; Blackwell, Hayward, and Crimmins 2001; Bengtsson and Lindström 2000). For example, there is increasing evidence that chronic diseases, such as diabetes, occurred at earlier ages in the nineteenth century in the United States than they do today (Fogel and Costa 1997). In contrast, the Tsimane show higher levels of C-reactive protein (CRP) across all ages. CRP is an acute-phase protein that acts as a marker and instigator of inflammation, and among Tsimane it is associated with disease load and presence of parasites. Increasing evidence indicates that malnutrition and health insults during fetal and perinatal development produce cascading effects leading to a greater risk of coronary heart disease later in life (Barker and Osmond 1986; Cameron and 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. Definitive answers on this score await further research.

The comparison between humans and chimpanzees suggests, however, that species differences overwhelm differences in environmental conditions in determining mortality hazards as individuals age. This finding suggests 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. When those differences are understood and then subjected to manipulation, the result may be changes in human mortality distributions at old ages that greatly exceed those attributable to disease treatment and assault prevention.

Appendix: The sample of societies

The Appendix describes each group in our sample, including the environmental and historical conditions during the time period to which the data correspond, the

methodology used to collect the data, and the methodological strengths and weaknesses of each data set. Contemporary hunter-gatherers have been affected by global socioeconomic forces and are not living replicas of our Stone Age ancestors. Each group has been exposed to a particular set of historical, ecological, and political conditions, and extant groups occupy only a small subset of the environments that foragers occupied in the past. Thus, even without the variable impact of infectious diseases and modernization, no single group can accurately represent all modern foragers or pristine foragers typical of our ancestral past (see Solway and Lee 1990).

Isolation from outsiders, small-scale social structure, and absence of amenities also characterize many incipient horticulturalist populations, many of whom also engage in foraging. Remote populations of forager-horticulturalists therefore merit attention. It is debatable whether transitions to agriculture always result in better nutrition and uniform increases in survivorship among all age classes (Armelagos, Goodman, and Jacobs 1991; Cockburn 1971; Cohen and Armelagos 1984). Incipient agriculturalists without modern amenities are an additional source of preindustrial societies to supplement our foraging sample. Horticulture-based populations are typically larger than foraging populations, which provides greater confidence in mortality estimates at late ages.

Hunter-gatherers

The ethnographic record of hunter-gatherers includes hundreds of cultures, but only about 50 groups have been studied. The sample of foraging societies presented here does not adequately cover all geographical areas. Only five foraging societies have been explicitly studied using demographic techniques—Hadza of Tanzania (Blurton Jones, Hawkes, and O'Connell 2002; Blurton Jones et al. 1992), Dobe !Kung (Howell 1979), Ache of Paraguay (Hill and Hurtado 1996), Agta of Philippines (Early and Headland 1998), and Hiwi of Venezuela (Hill et al. 2007).

Nancy Howell's Dobe !Kung study in the Kalahari desert of Botswana and Namibia is one of the first and most impressive demographic accounts of a foraging society. The majority of !Kung have been settled during the last 50 years and have been rapidly acculturating in close association with nearby Herero and Tswana herders. At the time of her study in the 1960s, many of the adults had spent most of their lives foraging, despite ethnohistorical evidence showing interactions with mercantile interests in the nineteenth century and archaeological evidence suggesting trade with pastoral and agricultural populations (see Solway and Lee 1990). An early !Kung sample refers to the period before the 1950s when the Bantu influence in the Dobe area was minimal. Later !Kung samples refer to the prospective time of study when the lifestyles of the !Kung were rapidly changing. In the 1970s, 454 people were living in the study site. Two life tables are used from this period. One is based on the 94 deaths during the 11-year study period (Howell 1979: Table 4.4), and the other uses the referent study population with a smaller number of deaths during the same time period (ibid.: Table 4.6). Because the early !Kung sample clumps all adults aged 45 years and older into one category, we construct a composite early !Kung sample by using mortality estimates for ages 40 and higher from Howell's Table 4.4 and combine this with mortality estimates for ages less than 30 from the "early" sample.

Ages 30–39 years are modeled as an average of the mortality rates from both early and prospective samples. Howell's Table 4.6 is placed in the acculturated hunter-gatherer category. Ages were determined through a combination of relative age lists, known ages of children and young adults, and application of stable age distribution from a "West" Coale and Demeny (1966) model, an approach similar to that initiated by Rose (1960).[7]

The Ache were full-time, mobile tropical forest hunter-gatherers until the 1970s. Kim Hill and Magdalena Hurtado (1996) separate Ache history into three periods—a pre-contact "forest" period of pure foraging with no permanent peaceful interactions with neighboring groups (before 1970), a "contact" period (1971–77) in which epidemics had a profound influence on the population, and a recent "reservation" period in which Ache live as forager-horticulturalists in relatively permanent settlements (1978–93). During this last period, the Ache have had some exposure to modern health care. The pre-contact period shows marked population increase, resulting in part from the open niche that was a direct result of high adult mortality among Paraguayan nationals during the Chaco War with Bolivia in the 1930s. No life table is published for the high-mortality contact period, during which many old and young died. Hill and Hurtado improve on Howell's methods of age estimation by using averaged informant-ranking of age, informant estimates of absolute age differences between people, and polynomial regression of estimated year of birth on age rank. Apart from living individuals, reproductive histories of a large sample of adults produced the samples used for mortality analysis. At the time of study, there were roughly 570 northern Ache.

The Hadza in the eastern rift valley of Tanzania were studied in the mid-1980s by Nicholas Blurton Jones and colleagues. Trading with herders and horticulturalists has been sporadic among Hadza over the past century, and the overall quantity of food coming from horticulturalists varies from 5 to 10 percent (Blurton Jones, Hawkes, and O'Connell 2002). The Hadza have been exposed to settlement schemes over the past 50 years, but none of these has proven very successful. The 1990s saw a novel type of outsider intervention in the form of further habitat degradation and "ethno-tourism" (ibid.). Although some Hadza have spent considerable time living in a settlement with access to maize and other agricultural foods, most continue to forage and rely on wild foods. Age estimation of the population was achieved using relative age lists, a group of individuals of known ages, and polynomial regression. Two censuses done about 15 years apart, with an accounting of all deaths during the interim, allowed Blurton Jones to construct a life table and to show that sporadic access to horticultural foods and other amenities cannot account for the mortality profile. There were roughly 750 Hadza in the study population.

The Hiwi are neotropical savanna foragers of Venezuela studied by Hill and Hurtado in the late 1980s (Hurtado and Hill 1987, 1990). They were contacted in 1959 when cattle ranchers began encroaching on their territory. Although living in semi-permanent settlements, Hiwi continue to engage in violent conflict with other Hiwi groups. At the time of study, almost their entire diet was wild foods, with 68 percent of calories coming from meat and 27 percent from roots, fruits, and an arboreal legume. The study population contains 781 individuals. Nearby Guahibo-speaking peoples practiced agriculture, but the Hiwi inhabited an area poorly suited

for agriculture. As among the Hadza, repeated attempts at agriculture by missionaries or government schemes have failed among this group. Mortality information comes from Hill et al. (2007).

The Casiguran Agta of the Philippines are Negrito foragers studied by Tom Headland during 1962–86. They live on a peninsula close to mountainous river areas and the ocean. There are 9,000 Agta in eastern Luzon territory, and demographic study was focused on the San Ildefonso group of about 200 people (Early and Headland 1998). Although Luzon itself is isolated, Agta have maintained trading relationships with lowlander horticulturalists for at least several centuries (Headland 1997). The twentieth century introduced schooling and brief skirmishes during American and Japanese occupation. Age estimation was achieved through reference to known ages of living people and calendars of dated events. As in the Ache study, the Agta demography is divided into a "forager" period (1950–65), a transitional period of population decline (1966–80), and a "peasant" phase (1981–93). These last two phases are marked by guerilla warfare, and subjugation by loggers, miners, and colonists. Abridged life tables are available for all three time periods. The age groups given are broad (10 years for adult ages), and the last age category is 60+.

Forager-horticulturalists

The above five populations comprise the foraging sample because the typology "hunter-gatherer" defines their mode of subsistence and its lack of reliance on domesticated foods. To the forager sample described above, we add the Yanomamo of Venezuela and Brazil, Tsimane of Bolivia, and Gainj of Papua New Guinea.

Yanomamo, Tsimane, and Machiguenga are forager-horticulturalist populations in Amazonian South America. Several Yanomamo studies have been carried out over the past 30 years. Although often construed as hunter-gatherers, Yanomamo have practiced slash and burn horticulture of plantains for many generations (Chagnon 1968). They live mostly in small villages of less than 50 people. The effects on Yanomamo of the rubber boom and slave trade before the eighteenth century were minimal (Ferguson 1995). They remained mostly isolated until missionary contact in the late 1950s. The most complete demography comes from Early and Peters (2000) based on prospective studies of eight villages in the Parima Highlands of Brazil. Births and deaths were recorded by missionaries and FUNAI personnel since 1959. The pre-contact period (1930–56) predates missionary and other outside influence. The contact period (1957–60), "linkage" period (1961–81), and Brazilian period (1982–96) saw increased interaction with miners and Brazilian nationals and increased incidence of infectious disease. Ages for Xilixana (Mucaj) during this period were estimated using a chain of average interbirth intervals for people with at least one sibling of known age, and relative age lists in combination with estimated interbirth intervals. Because it set a historical precedent, we include the Neel and Weiss (1975) life table for Yanomamo based on 29 villages in Venezuela even though it does not meet our inclusion criteria. It applies a best-fit model life table using census data, a growth rate based on repeated censuses, and age-specific fertility. These censuses were taken during the 1960s, and ages were obtained by averaging different researchers' independent estimates.

The Tsimane inhabit tropical forest areas of the Bolivian lowlands, congregating in small villages near large rivers and tributaries. Roughly 8,000 Tsimane live in dispersed settlements in the Beni region. Tsimane have had sporadic contact with Jesuit missionaries since before the eighteenth century, although they were never successfully converted or settled. Evangelical and Catholic missionaries set up missions in the early 1950s and later trained some Tsimane to become teachers in the more accessible villages. However, the daily influence of missionaries is minimal. Market integration is increasing, as are interactions with loggers, merchants, and colonists. Most Tsimane continue to fish, practice horticulture, and hunt and gather for the bulk of their subsistence. The demographic sample used here is based on reproductive histories collected by Gurven of 348 adults in 12 remote communities during 2002–03. Changes in mortality are evident in the 1990s, hence mortality data used here are restricted to the years 1950–89. Age estimation of older individuals was done by a combination of written records of missionaries, relative age rankings, and photo and verbal comparison with individuals of known ages.

The Gainj are swidden horticulturalists of sweet potato, yams, and taro in the central highland forests of northern Papua New Guinea. Meat is fairly rare (Johnson 1981). At the time of study by Patricia Johnson and James Wood in 1978–79 and 1982–83, 1,318 Gainj were living in 20 communities. Contact was fairly recent, in 1953, with formal pacification in 1963, and there is genetic and linguistic evidence of their relative isolation (Wood et al. 1982). Prior to contact, population growth had been zero for at least four generations (Wood and Smouse 1982). An A2 Hong Kong influenza epidemic reduced the population by some 7 percent in 1969–70 and probably accounts for the dearth of older people in this population. Data were obtained from government censuses from 1970 to 1977; they include non-Gainj Kalam speakers, and it is likely that age estimates are fraught with error for older adults (see Wood and Smouse 1982). Additionally, published mortality estimates were already fitted with a Brass two-parameter logit model.

Acculturated hunter-gatherers

In addition to the Ache reservation sample, Agta transitional and peasant samples, and recent !Kung and Yanomamo samples, we include the Warao of Venezuela and Northern Territory Aborigines into the acculturated hunter-gatherer category.

The Warao were traditional swamp foragers living in the Orinoco Delta in eastern Venezuela. They remained isolated in the bogland of the Orinoco until the last century. Apart from the fauna common to most of tropical South America, the Warao diet included products of the carbohydrate-rich sago palm. The Warao are included here even though they were once foragers because at the time of study they were practicing intensive agriculture. Oil exploration, colonist intrusion, and missionary influence all affected Warao from the 1920s on and pushed them to live near more accessible riverbanks. Increased sedentism and some access to modern health care have probably improved infant and child mortality. Preliminary demographic data were collected in 1954 by Johannes Wilbert (Wilbert and Layrisse 1980), but most information was collected by H. Dieter Heinen from 1966 to 1976 (Layrisse, Heinen, and Salas 1977). The sample covered 1,360 individuals. Age groups for adult ages are ten years, and the last category is 50+.

The Northern Territory Australian Aborigine mortality data come from analysis of vital registration from 1958 to 1960 by Frank Lancaster Jones (1963, 1965). At that time, few Aborigines in the region remained full-time foragers. There was substantial age-clumping at five-year intervals, so a smoothing procedure was performed on the population age distribution. It is likely that infant deaths and more remote-living individuals were underenumerated, and Lancaster Jones made adjustments to impute missing deaths. We view these data with caution but include them because no other reliable data exist for Australia, apart from the Tiwi sample, which was culled from the same author and is also included here although it suffers from the same limitations. We also include data by Crotty and Webb (1960) on causes of death based on autopsy for 175 Northern Territory Aborigines during the same time period.

Notes

1 Age-independent mortality (a_2) is not statistically significant for the Agta, but initial adult mortality (a_3) is about 150 times greater than in other foragers. Part of this high effect is compensation due to the lack of age-independent mortality among Agta (Gage and Dyke 1986). Correspondingly, the senescence rate (b_3) is less than one-half that of other foragers.

2 These analyses, however, did not control for age-independent mortality (a_2), a factor which could bias Gompertz estimates (Gavrilov and Gavrilova 2001).

3 A second approach to examining departures from linear increases in mortality hazards is to introduce a second-order age term in the Gompertz model: $\ln h(x) = a_3 + b_3 x + b_4 x^2$. Of the most reliable cases, positive statistically significant values for b_4 are found for Hadza, Ache, Hiwi, !Kung, Yanomamo, and Tsimane, indicating that the hazard increases at an increasing rate with age. Only the settled Ache and recent !Kung did not show a significant positive second-order term.

4 Indeed, Howell (1979) shows that the estimated life expectancy of acculturated !Kung based on this life table is about 50 years, which is ten years higher than the national estimate of Botswana during the same time period.

5 In a stable age distribution, $R_0 = \exp(rT)$, where R_0 is the net reproductive rate, r is population growth rate, and T is the average generation length, set here as 28 years. We define $R_0 \approx R * l_{25} = \text{TFR}/2.06 * l_{25}$, where TFR is the total fertility rate. Based on a regression of l_{25} on l_{15}, we estimate l_{25} as $0.9973 * l_{15} - 0.0422$ ($R^2 = 0.98$, $p < 0.0001$). These equations allow the construction of approximate iso-growth curves as a function of TFR and l_{15}.

6 These include the Ngamiland Ju'/hoansi ($l_{15} = 0.66$) (Harpending and Wandsnider 1982), Greenland Inuit (0.55), Chenchu (0.51) (cited in Hewlett 1991), Aka Pygmies (0.55) (Bahuchet 1979), Bofi Pygmies (0.59) (Fouts, Hewlett, and Lamb 2005), Mbuti Pygmies (0.44) (Harako 1981), Batak (0.48) (Eder 1987), Australian Aborigines (0.55), and Seri (0.39) (cited in Weiss 1973).

7 There is not a very large difference between our composite "early" !Kung sample and the early sample employed by Howell that includes model life table estimates for mortality rates for ages over 40. For example, $l_{45} = 0.37$ (vs. 0.40 for the composite), $e_{45} = 23.2$ (vs. 21.2), $l_{65} = 0.21$ (vs 0.21), $e_{65} = 13.2$ (vs. 9.1).

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