

Longevity among hunter-gatherers: a cross-cultural examination

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Abstract

Post-reproductive longevity is a robust feature of human life and not only a recent phenomenon caused by improvements in sanitation, public health and medical advances. We argue for an adaptive lifespan of 65-75 years for modern *Homo sapiens* based on our analysis of mortality profiles obtained from small-scale hunter-gatherer and horticultural populations from around the world. We compare patterns of survivorship across the lifespan, rates of senescence, modal ages at adult death and causes of death. We attempt to reconcile our results with those derived from paleodemographic studies that characterize prehistoric human lives as “nasty, brutish and short”, and with observations of recent acculturation among contemporary subsistence populations. We integrate information on age-specific dependency and resource production to help explain the adaptive utility of longevity in humans from an evolutionary perspective.

1. Introduction

Average worldwide human life expectancy reached 63 years in 1998 (World Factbook 2004), with extremes at the national level ranging from 37 in Sierra Leone and Zambia to 81 years in Japan and San Marino. Average life expectancy has increased linearly at 4 months per year over the past 160 years, with improvements in sanitation, nutrition, and public health initiatives accounting for much of this change (Oeppen and Vaupel 2003, Riley 2001). 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 are novel, almost surely never having occurred before in human history. The genus *Homo* has existed for about two million years; humans have therefore lived as hunter-gatherers for the vast majority of their evolutionary history. Agriculture and pastoralism have only been practiced for about 10,000 years, and most extensively in the past 5,000 years. While there may have been some important genetic changes 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 pre-agricultural history (see Kaplan et al. 2001, Kaplan et al. 2000, see Kaplan 1997 for reviews). 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 paper is to assess our evolved human mortality profile and particularly the pattern of senescent mortality change with age. We will address the following

questions: 1) is there a characteristic shape to the human mortality profile, as it decreases first during childhood and then increases in old age; 2) how variable is this mortality profile among populations, and what factors shape any variation; 3) how robust is the occurrence of a post-reproductive lifespan and how likely is it that older adults were alive and available; and 4) 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 lifespan?

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). In order to understand the processes that shaped the evolution of our life course, it would be useful to have data on mortality and fertility profiles across populations and over evolutionary time. Given that these data do not exist, we utilize and critically evaluate data on modern groups, as one ‘imperfect lens’ into our past.

Present day hunter-gatherers have all been affected by global socioeconomic forces and are not living replicas of our stone age ancestors. Each group has been exposed to a unique set of historical, ecological and political conditions, and extant groups only occupy a small subset of the environments that were occupied by foragers in the past. Thus, even without the variable impact of infectious disease and modernization, no single group has or can ever accurately represent all modern foragers, or pristine foragers typical of our ancestral past (see Solway and Lee 1990).

The isolation from outsiders, small-scale social structure and absence of amenities are also characteristic of many small-scale incipient horticulturalist populations, many of whom also engage in foraging activities. Remote populations of forager-horticulturalists therefore merit special attention. It is debatable whether transitions to agriculture always result in better nutrition

and uniform increases in survivorship among all age classes (Armélagos, Goodman, and Jacobs 1991, Cockburn 1971, Cohen and Armélagos 1984). Incipient agriculturalists without modern amenities are an additional source of pre-industrial 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. In addition to general population variability, demographic data collected among the study populations vary in the methodologies used to obtain them and in the quality of information regarding ages and especially, adult mortality hazards. For this reason, the next section provides a description of each group in our sample, including both the environmental and historical conditions during the time period to which the data correspond and the methodology used to collect the data. The methodological strengths and weaknesses of each data set are also addressed.

The third section then models log mortality hazards as a function of age for each of these populations. We first focus on both the estimations of the likelihood of being alive at the age of reproductive termination, and the expectation of life upon reaching this age. We argue that post-reproductive longevity is a robust feature of hunter-gatherers, and hence of the life-cycle of *Homo sapiens*. “Old age” is not merely a novel artifact of improved conditions (cf. King and Jukes 1969, Pavelka and Fedigan 1991, Smith 1993, Vallois 1961, Washburn 1981, Weiss 1981). We then take two approaches for modeling mortality hazards. First, we use the Siler competing hazards model (Gage and Dyke 1986, Siler 1979) to assess the shape of the mortality function over the whole life course. This model yields three biologically interpretable components of mortality. We find that there is much variability in infant and juvenile mortality rates across populations, and that mortality rates tend to converge in adulthood. All of the populations with reliable data for older ages show that life expectancy is almost another two decades after age 45,

when most women have ceased reproducing. A fourth section focusing on the distributions of deaths in adulthood reveals that each of the populations shows evidence of a clear mode in the death distribution, with an inter-population mean of 72 years of age.

The second approach to modeling mortality hazards is explored in the fifth section by fitting Makeham-Gompertz models to different phases of the life course. We examine the rate of senescence in these populations, and test the extent to which the Gompertz (1825) relationship, found in many biological species, including mice, fruit flies, and most human populations under normal conditions (Finch 1990, Gavrilov and Gavrilova 1991), applies to our ethnographic sample of foraging societies. The Gompertz model of mortality after reproductive maturity assumes an exponential increase in the number of deaths, or a linearly increasing log mortality hazard with age. Parameterization of this model therefore permits estimation of ‘senescence’ as the rate of mortality increase, and of the mortality rate doubling time (MRDT) (Finch, Pike, and Whitten 1990, Stearns 1992). All modern populations appear to exhibit a Gompertz-like mortality increase over much of the adult years, with a leveling out or slight decline occurring only after age 85 (Vaupel 1997). We find that mortality rates do not rise significantly between the ages of 15 and 40, and then rise exponentially thereafter.

The populations discussed in this article live under different ecological conditions, varying from wet to dry, more or less access to food, disease exposure, settlement duration, and in many other respects. They also vary their interactions with the larger world, and in the effects of those interactions. In the sixth section, we first examine the relationship between juvenile mortality and life expectancy at age 45 across populations to determine if the two are correlated. This would indicate either that better conditions affect all age groups, or that worse conditions during childhood (as indexed by juvenile mortality rates) increases the rate of senescence. Next,

we conduct internal comparisons within populations, when data exist for groups before and after increased contact with outsiders. This temporal component in several populations allows us to examine the variable effects of acculturation on mortality in different cultural and ecological settings. Here we find that adult death rates tend to decrease with increasing contact (following, in some cases, a period of high death rates and population decrease with epidemics at first contact), and the modal age at death increases.

We then examine causes of death, as they vary across populations to evaluate their impacts of mortality rates. These data are analyzed to determine if changes in causes of death with age can help explain mortality profiles, and to assess whether exigencies of being hunter-gatherers in the modern world cast doubt on inferences about ancestral populations based on our sample. We find that infectious and gastrointestinal disease are widespread killers early in the lifespan and degenerative disease uniformly impacts adults, While adult mortality rates are fairly uniform, causes of adult deaths are somewhat variable, owing to group-specific experiences with epidemics and homicide or warfare.

The sixth section compares our results with estimates derived from analysis of funerary samples from prehistoric populations and with our nearest primate relatives, chimpanzees. We attempt to reconcile our argument concerning the robusticity of a long human life-span with the common assertion of a “nasty, brutish and short” ancestral pattern. We argue that paleodemographic life tables derived from prehistoric populations are highly suspect, because infant and juvenile survival rates are unrealistically high and adult survival rates are unrealistically low. We also show evidence for positive population growth among most extant small-scale populations and discuss the implications for making inferences about past hunter-gatherer populations.

The last section summarizes and discusses our findings.

After publishing their life table for Yanomamo Amerindians of Venezuela thirty 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 any other previous study to date (Gage 1998, Kaplan et al. 2000, Kennedy 2003, Pennington 2001). This is our best opportunity to examine the mortality structure of humans living a lifestyle most similar to those of our foraging ancestors. These populations are acculturating at a rapid rate and so future data are unlikely to be forthcoming. For example, 33 of 105 isolated indigenous groups in Brazil went extinct from 1900 to 1957, and there are only 33 groups that remain isolated (Ribeiro 1967:92). There are currently very few extant groups of human hunter-gatherers, and probably no large group for which detailed demography on people of all ages will be available. The few groups that do exist are most likely to be 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 paper.

2. Methodology

2.1. Sampling Criteria

Our general approach is to assess the mortality profiles of all extant hunter-gatherers for which quality demographic data exist. We include in this data set other small-scale populations

that also engage in slash and burn horticulture because these populations 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 to no inclusion of horticulture nor market-derived foods in their diet. Other traits commonly associated with a foraging lifestyle are variable among hunter-gatherer groups, such as mobility, an egalitarian ethic, widespread sharing, minimal storage, and other social traits (Kelly 1995). Quality data means that age estimation is reasonably accurate, and there is no systematic bias in the under-reporting 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 table fits to scanty data or census data. They 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 over-estimated (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 a rigorous critical process for error-checking.

[INSERT TABLE 1 HERE]

2.2. The Sample of Societies

2.2.1. Hunter-gatherers

The ethnographic record of hunter-gatherers includes hundreds of cultures, but only fifty or so groups have been studied. The sample of foraging societies presented here does not adequately cover all geographical areas. Table 1 provides the study years, time period covered, sample sizes of individuals, total person-years and person-years for individuals age 50 and over, and source information for the data. 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 the Hiwi of Venezuela (Hill et al. 2006).

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 fifty years, and have been rapidly acculturating in close association with nearby Herero and Tswana herders. At the time of study, many of the adults had spent most of their lives foraging, despite ethnohistorical evidence showing interactions with mercantile interests in the 19th century and archaeological evidence suggesting trade with pastoral and agricultural populations (see Solway and Lee 1990). An early !Kung sample refers to the time period before the 1950's when the Bantu influence in the Dobe area was minimal. Later !Kung samples refer to the prospective time of study when the lifeways of the Kung were rapidly changing. At the time of study, there were about 454 people living in the study site. Two lifetables are used from this time period. One is based on the 94 deaths during the eleven 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). Since the early !Kung

sample clumps all adults aged 45 and over 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 through 40 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, a similar approach initiated by Rose (1960).¹

The **Ache** were full-time, mobile tropical forest hunter-gatherers until the 1970's. Hill and Hurtado (1996) separate Ache history into three time periods—a precontact "forest" period of pure foraging with no permanent peaceful interactions with neighboring groups (before 1970), a "contact" period (1971-77) where epidemics had a profound influence on the population, and a recent "reservation" period where they live as forager-horticulturalists in relatively permanent settlements (1978-1993). During this latter period, the Ache have had some exposure to health care. The pre-contact Ache period shows marked population increase, due in part to the open niche that was a direct result of high adult mortality among Paraguayan nationals during the Chaco War with Bolivia in the 1930's. No life table is published for the high mortality contact period which killed many older and young individuals. 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 built 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-1980's by 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 5-10% (Blurton Jones, Hawkes, and O'Connell 2002). The Hadza have been exposed to a series of settlement schemes over the past fifty years, but none of these has proven very successful. The 1990's saw a novel form 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 have not and continue to forage and rely on wild foods. The population was aged using relative age lists, a group of individuals of known ages, and polynomial regression. Two censuses done about fifteen years apart, with an accounting of all deaths during the interim, allowed Blurton Jones to construct a life table, and to further 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 1980's (Hurtado and Hill 1990, Hurtado and Hill 1987). They were contacted in 1959 when cattle ranchers began encroaching into 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 the entire diet was wild foods, with 68% of calories coming from meat, and 27% from roots, fruits, and an arboreal legume. The study population contains a total of 781 individuals. Nearby Guahibo-speaking peoples practice agriculture, while the Hiwi inhabited an area poorly suited for agriculture. As among the Hadza, repeated attempts at agriculture by

missionaries or government schemes had failed among this group. Mortality information comes from Hill et al. (2006).

The Casiguran **Agta** of the Philippines are Negrito foragers studied by Tom Headland from 1962-1986. 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 Idefonso group of about 200 people (Early and Headland 1998). Although the Luzon area is itself very 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 (1950-1965), a transitional period of population decline (1966-1980), and a “peasant” phase (1981-1993). These latter phases are marked by guerilla warfare, and subjugation by loggers, miners and colonists. Abridged life tables are available for each of these time periods. The age categories given are large (10 years for adult ages) and the last age category is 60+.

2.2.2. Forager-horticulturalists

The above five populations comprise the foraging sample because the typology “hunter-gatherer” defines their mode of subsistence, and therefore a 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-agriculturalist populations in Amazonian South America. Several different Yanomamo studies have been carried out over the

past thirty years. Although often construed as hunter-gatherers, **Yanomamo** have practiced slash and burn horticulture of plantains for many generations (Chagnon 1968). They mostly live in small villages of less than fifty people. The effects of the rubber boom and slave trade before the 18th century on Yanomamo were minimal (Ferguson 1995). The Yanomamo remained mostly isolated until missionary contact in the late 1950's. 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 precontact 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, Brazilian nationals and infectious disease. Ages for Xilixana (Mucajai) 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. Due to historical precedent, we include the Neel and Weiss (1975) life table for Yanomami 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 1960's, and ages were obtained by averaging different researchers' independent guesses.

The **Tsimane** inhabit tropical forest areas of the Bolivian lowlands, congregating in small villages near large rivers and small tributaries. There are roughly 8,000 Tsimane living in dispersed settlements in the Beni region. The Tsimane have had sporadic contact with Jesuit missionaries since before the 18th century, although were never successfully converted or settled. Evangelical and Catholic missionaries set up missions in the early 1950's, 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, hunt and gather for the majority of their subsistence. The demographic sample used here is based on reproductive histories collected by Gurven of 348 adults in 12 remote communities during 2002-2003. Changes in mortality are evident over the past ten years, and so mortality data used here are restricted to the years 1950-1989. Age estimation of older individuals was done by a combination of written records of missionaries, relative age rankings, and by 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, there were roughly 1,318 Gainj living in twenty 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 6.5% in 1969-70, and probably accounts for the dearth of older people in this population. Data were obtained from government censuses from 1970-77, include non-Gainj Kalam speakers, and it is likely that ages 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.

2.2.3. Acculturated Hunter-Gatherers

In addition to the Ache reservation sample, Agta transitional and peasant samples, 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 fairly isolated in the bogland of the Orinoco up until the last century. Apart from the fauna common to most of tropical South America, 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 had already been practicing intensive agriculture. Oil exploration, colonist intrusion and missionary influence all impacted Warao since the 1920's and pushed them to live near more accessible riverbanks. Increased sedentism and some minimal access to health care has probably improved infant and child mortality. Preliminary demographic data were collected in 1954 by Johannes Wilbert (Wilbert and Layrisse 1980) but most information was later collected by Heinen from 1966-1976 (Layrisse, Heinen, and Salas 1977). There were 1,360 individuals in the sample. Age categories for adult ages are ten years, and the last age category is 50+.

The **Northern Territory Australian Aborigine** mortality data come from analysis of vital registration from 1958-1960 by Lancaster Jones (1963, 1965). At this time, few Aborigines in the region were still full time foragers. There was a significant amount of age-clumping at five year intervals, and so a smoothing procedure was done on the age distribution of the population. It is likely that infant deaths and more remote-living individuals are under-enumerated, 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.

2.2.4. Summary of Data Limitations

Although this sample is the most complete set of preindustrial populations, there are some limitations which limit the sample of populations relied upon for specific analyses. The Agta, and Warao demographics lack sufficient age resolution at later ages for us to trust mortality profiles after the age of 45 or 50. Age estimates for older adults among the Gainj are unreliable, with no age categories beyond age 60. Data on older adults are sparse among Machiguenga. The Aborigine data may suffer from under-reporting of infant deaths. Highest quality data across the entire lifespan exist for Hadza, Ache, !Kung, Hiwi, Tsimane and Yanomamo Xilixana. These populations will be used in focused analyses of older age mortality.

2.3. Modeling Mortality

To illustrate patterns of mortality we present age-specific survivorship curves, $l(x)$, and log mortality hazard rates, $\ln(h(x))$. We model each group's mortality using a Siler competing hazard model due to its simplicity, robusticity and the interpretability of its parameters (Gage 1989, Gage 1991, Siler 1979, but see Wood et al. 2002). The Siler model includes three components of mortality: an immature component of declining mortality, a constant mortality hazard across the lifespan, and an increasing component in older ages. Infant and child mortality are thus modeled with a negative Gompertz function. The latter component is the famous 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 based on first biological principles although different explanations have been proposed to produce Gompertz-like mortality (Gavrilov and Gavrilova 2001, Olshansky and Carnes 1997). Other survival models which show monotonically increasing mortality rates over time, such as Weibull and Gamma, often explain data as well as Gompertz (Wood et al. 1994).

The Siler hazard has the following functional form:

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

This is a five parameter model. The parameter a_1 describes the initial infant mortality rate, and b_1 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 to be 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:

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

All parameter estimation is done on the original $l(x)$ profiles using non-linear regression (PROC NONLIN) with SAS version 9.1. Regression of log mortality hazards on age by population is done with the generalized linear model (GLM) procedure in SAS.

[INSERT FIGURES 1 and 2 HERE]

3. Results

3.1. 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, respectively, 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-gatherers, four forager-horticulturalists, and five acculturated hunter-gatherers. For comparison purposes, in the table and panel D of both figures, we compare the averages across groups with data from chimpanzees (Hill et al. 2001) and 18th 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.

[INSERT TABLE 2 HERE]

In Table 2, we see that on average 58%, 64%, 63% of children born survive to age 15 among hunter-gatherers, forager-horticulturalists and acculturated hunter-gatherers, respectively. Of those who reach age 15, 61% will reach age 45 among both traditional hunter-gatherers and forager-horticulturalists. The acculturated hunter-gatherers show lower young adult mortality rates, with close to 80% 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 (18.7, 18.6, and 21.5 for hunter-gatherers, forager-horticulturalists and acculturated hunter-gatherers, respectively). Traditional hunter-gatherers and forager-horticulturalists are almost identical in the adult life course, and on average, acculturation improves adult life expectancies.

There is some variability among groups (to be discussed in greater detail below). Among traditional hunter-gatherers, the average life expectancy at birth (e_0) varies from 24 to 36, the probability of living to age 45 varies between 26% and 41%, and expected lifespan at age 45 varies from 15 to 24 years (Figure 1; Table 2). 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. Parameter estimates for all Siler models discussed in this paper are presented in Table 2. Initial immature mortality (a_1) for the Ache is less than half than in other foragers, while for the Agta it is three to four times greater.²

Forager-horticulturalists also vary significantly in infant mortality with a three-fold difference between Neel and Weiss's Yanomamo sample and the Tsimane. Survival to age 45 varies between 19 and 54%, and those aged 45 will 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.

Acculturated foragers vary most in their likelihood to reach age 45 (ranging from 25% among the peasant Agta population to 66% among sedentary !Kung and Ache), but show a similar range of 14-27 additional years of life upon reaching age 45. Adult mortality is also highly variable. For example life expectancy is 49 for Aborigines, 50 for settled Ache, yet 22 and 29 for peasant and transitional Agta, respectively. The acculturated category shows a range of mixed experiences with acculturation (see section 3.5).

The rate at which mortality rates double is another measure of senescence rate. Finch et al. (1990) report mortality rate doubling times (MRDTs) of 7-8 for a variety of recent human populations with low and high mortality³. Despite the overall high mortality of hunter-gatherer populations, we find that the adult mortality rate also doubles in 8 years among Hadza and Ache, (Table 2). The Hiwi MRDT shows rapid senescence (4.6 years) although the 95% confidence interval is fairly large (ranges from 3-10). Among the Agta where high adult mortality is placed

in the a_3 rather than a_2 , senescence appears very slow for the Agta forager and peasant samples (MRDT=30, 12).

Several forager-horticulturalists and acculturated foragers show a similar MRDT, with Tsimane, Ache, and one Yanomamo sample at 7, 8, and 9 years, respectively. Groups with high adult mortality show increased, rather than decreased estimates of MRDT. The Gainj and Herero show slower rates at 11 and 14, while the Yanomamo estimate based on the life table of Neel and Weiss (1975) shows a rate similar to the Agta. The acculturated foragers show a range of MRDT from 7-10. Only the peasant Agta sample exhibits a slower rate of 12 years. Overall, the highest quality data show a consistent range of MRDT at 7-10 for our study groups (Table 2, bold groups).

Among groups, differences in early juvenile mortality explain much of the differences in overall mortality. Infant and child mortality varies widely among small-scale traditional populations. Indeed, a second-order polynomial regression of e_0 on infant mortality rate using the full sample of populations shows that 80% of the variation in e_0 is explained by differences in IMR. The largest departure from later age mortality hazards appears among the Agta and Gainj, whose mortality data we earlier described as being circumspect at later ages.

In spite of this variation, there does appear to be clear pre-modern human pattern. There is remarkable similarity in age profiles of mortality risk over the lifespan. By age 10, the mortality hazard has slowed to 0.01, 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 accelerations become more evident. The adult mortality rate doubles every 8-10 years (see below). The results obtained from these groups are quite similar to those from Sweden in 1751 (see panel b in Figures 1 and 2), where mean life expectancy was 34 years, and e_{45} was 21

additional years (Wilmoth, Berkeley Mortality Database). For populations living without health care, public sanitation, immunizations, or abundant and predictable food supply, it seems that at least one-fourth of the population is likely to live as grandparents for 15-20 years.

In contrast, 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 that of human foragers, the onset of mortality rate doubling occurs at least ten years earlier among chimpanzees. It is important to note how the protected environment of captivity affects chimpanzee mortality profiles. Captivity raises infant and juvenile survival greatly, from 35% surviving to age 15 to 63%, similar to the hunter-gatherer averages. However, while the probability of reaching 45 increases tenfold from 2% in the wild to 20% with captivity, it is still only just over half as high as humans, living in pre-modern conditions. The difference between chimpanzees and humans after age 45 is even greater, with an expected future life span of chimpanzees in captivity of only 7 years, less than half of the human expectation. It appears that chimpanzees simply age much faster than humans and die earlier, even in protected environments.

[INSERT FIGURE 3 HERE]

3.2. Convergent mortality and modal ages of adult death

An inspection of Figure 2 and additional analysis suggests that there is greater variance in mortality rates across populations at younger ages than at older ages. However, mortality rate variation in middle adult ages is not insignificant, and is likely due to population differences in the extent of violence and maternal mortality (see 3.4.3). Nonetheless, the convergence of mortality rates at late ages is especially evident in Panel D of Figure 2. Figure 3 shows expected

future years of life remaining (e_x), conditional on living to each age, for the groups with the most reliable data. While there is significant variation across groups in life expectancy at early ages, there is significant convergence after about age 30. The major exception is the Hiwi, who show up to 10 years less remaining during early ages and 3-5 years less remaining during adulthood. With the additional exception of the Hadza, whose life expectancy at each age is about 3 years longer than the rest at most adult ages, and the Hiwi, who, all other groups, including 18th century Sweden, are hardly distinguishable.

[INSERT FIGURE 4 HERE]

Following the lead of Kannisto (2001) and Lexis (1878), we examine the modal ages of “normal” adult death and the variance around these modes to examine the extent of stability in adult lifespans among and within our study populations (see Figures 4 and Table 3).. The extent of variation around the mode is usually defined as four standard deviation units around the mode (Cheung et al. 2005). Figure 4 shows the frequency distribution, $f(x)$, of deaths at age x , conditional upon surviving to age 15, for our composite categories of hunter-gatherer, forager-horticulturalists and acculturated hunter-gatherer samples using all populations with high data quality and age specificity. All curves (except Sweden and the U.S.) are based on the Siler models. Again data from prehistoric Sweden and modern U.S. are shown for comparative purposes. It is noteworthy that all pre-modern populations show modal life spans between 69-75 years (Table 3). Settled Ache show a slightly higher mode at 77 years, consistent with their improved conditions after settlement (see below). There is much more variability in the ages of adult death within each of these populations than typically found in modern industrial populations (Cheung et al. 2005), due in part to the higher age-independent mortality (a_2) (see section 3.4). Lexis (1878) considered such mortality as 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 73 to 77, and decreases the standard deviation around the mode from 8.1 to 6.1. The modes derived from the Siler model are therefore less peaked, accounting for no more than 3% of adult deaths. Of the three group sets, acculturated hunter-gatherers show the greatest density 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 important leading to a greater density of death around the mode.

[INSERT TABLE 3 HERE]

Another way of demonstrating the robustness of mortality convergence at older ages is through a comparison of the mortality hazards for our average hunter-gatherer with those of the U.S. in 2002. Figure 5 shows the ratio of hunter-gatherer to U.S. hazards. Infant mortality is over 200 times greater among hunter-gatherers and child mortality is about 100 times greater than encountered in the U.S. Not until the late teens does the relationship flatten, with a tenfold difference in mortality. This difference is five-fold by age 50, about four-fold by age 60 and below three-fold by age 70.

[INSERT FIGURE 5 HERE]

3.3 Nonlinearities in rates of senescence

Senescence is usually defined as an increase in the endogenous rate of mortality (Finch, Pike, and Whitten 1990, Rose 1991). It has been reported that in many populations mortality reaches its minimum at reproductive maturity and then increases thereafter at a constant proportional (Gompertz) rate (although noticeable decreases in vital functions do not occur until at least age 30 (Shock 1981, Weale 2004). The demographic literature often varies on the starting

point for expected mortality increases in the Gompertz relationship (Olshansky and Carnes 1997).

Here we examine whether traditional human populations reveal a constant proportional increase in mortality rates. 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+. Those results are shown in Table 4. For most populations, and 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 age 40. Among the Hiwi and Hadza mortality rates actually *decline* from 15 until about 35. The remaining groups show very little increase in mortality rates, ranging from about 1% a year among the Ache, Yanomamo and Tsimane to about 4% 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 (see Causes of mortality, below). Under normal circumstances, however, it appears that traditional humans experience about two decades of prime adulthood in which mortality rates hardly change, then experience a more sharply increasing mortality hazard in middle and old age.

[INSERT TABLE 4 HERE]

A second approach to examining departures from linear increases in mortality hazards is to model is to introduce a second-order age term in the Gompertz model:

$$\ln(h(t)) = a_3 + b_3t + b_4t^2 \quad (3)$$

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.

3.4. Variation in mortality rates and the effects of changing conditions

3.4.1 The relationship between juvenile and adult mortality rates across populations

There is some historical cohort evidence that high infant and child mortality have substantial impact on survivorship later in the lifespan (Costa 2000, Wilmoth, Vallin, and Caselli 1990). For example, Finch and Crimmins (2004) show that child mortality explains 85% of the variance in old-age mortality in cohorts of Sweden and U.S., and that early mortality has a three-fold greater predictive effect on old-age mortality for that birth cohort than period measures of early mortality. Figure 6 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 and northern Europeans. An additional 15-20 years of life are expected upon reaching age 45.

[INSERT FIGURE 6 HERE]

3.4.2 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 described in section 2, demography 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). Since similar data are also available for the Herero, a group of agro-pastoralists in Botswana and Namibia (Pennington and Harpending 1993), we include them in this comparison as well. Figure 7 shows the ratio of age-specific mortality hazards from more and less acculturated time periods. Figure 7a displays populations whose survivorship has improved with acculturation (!Kung, Ache, Herero, Hiwi) and Figure 7b those whose overall condition has worsened (Agta and Yanomamo).

[INSERT FIGURE 7 HERE]

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 population, and about 40% died in the course of less than a decade of contact. Here we compare the post-contact with the pre-contact period. With contact, there has been a small increase in infant mortality, but mortality at other ages has decrease by a third to a quarter. The effects of improved conditions are greater at younger ages and gradually decay with age. Reduced mortality among settled Ache is largely due to reductions in homicide and forest-related accidents with missionary influence and state intervention (Hill and Hurtado 1996). Medical attention also has helped decrease 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 shifted 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 due to gaps in the prospective life table created by Howell (1979: Table 4.6)⁴.

Among the Hiwi, contact has greatly increased infant mortality, but decreases it after infancy by about a third (there are not enough data on the very old to know what is happening 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 no longer maintain close trading relationships with nearby horticulturalists (Early and Headland 1998). Early and Headland suggest that cumulative effects of malnutrition and infectious disease such as measles have increased child mortality during the peasant phase, and 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 for both Agta and Yanomamo but reaching epidemic proportions in recent 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 infectious disease appears to negatively impact adults over age 40 among Yanomamo but reproductive-aged adults among Agta. One possibility is that susceptibility due to differential prior exposure may vary by age groups.

Other studies that have focused on the effects of sedentism on mortality show a general decrease in child mortality, consistent with our description 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). However, a slight increase in 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 before a noticeable decline in mortality.

Acculturation in the past several decades is likely to be very different than 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, 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, will occur when these benefits are lacking or unavailable.

3.4.3 Causes of mortality

It is important to investigate causes of death among groups and at different ages to identify if mortality patterns are due to unique circumstances and history, ecological conditions or genetic predispositions. Indeed, 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 medical physicians or autopsies, and especially when causes of deaths are elicited during retrospective interviews. Even in modern hospitals, cause of death is sometimes

elusive or ambiguous because of multiple levels of causation. For example, blood poisoning may be an immediate cause, pneumonia an intervening one, with an underlying cause of pancreatic cancer, and a contributing condition of diabetes (Preston, Keyfitz, and Schoen 1972).

Malnutrition, infections and disease are common in our study populations, thereby further making simple classification of primary cause of death problematic, if not inevitably incomplete. However, accidents and homicides are usually easier to identify, and estimates of these causes should be less subject to error. Table 5 presents the causes of death among the study sample 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).

[INSERT TABLE 5 HERE]

Table 5 shows the overall percentage of deaths attributed to illness, degenerative disease, and accidents or violence for !Kung, Ache, Yanomamo, Tsimane, Aka, Agta, Hiwi, Gainj, Tiwi, Baikari and Machiguenga. Our sample includes 3,328 deaths. Where age-specific data exist, cause of deaths are 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 sub-divide the illness category into respiratory, gastrointestinal, fever, and other illnesses (sensu Hill and Hurtado 1996, Howell 1979). Gastrointestinal illnesses account for 5-18% of deaths. Gastrointestinal deaths are higher among Tsimane than Ache, Yanomamo and Baikari, even though all three groups show high rates of parasitism in tropical environments.

This is likely explained by the younger age structure of the Tsimane. Diarrhea coupled with malnutrition is and remains one of the most significant causes of infant and early child deaths among foragers and peasant populations. People living in tropical forest environments are especially vulnerable to helminthic parasites (Dunn 1968), which although not usually lethal, can compromise growth and immune function.

A fifth or more of illness-related deaths among these groups is due to respiratory-related illnesses, such as bronchitis, tuberculosis, pneumonias, and other viral infections. Among many South American Amazonian groups, Black et al. (1975) report 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%. 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 perinatal problems early in infancy, late-age cerebrovascular problems, as well as attributions of “old age” in the absence of any obvious symptom or pathology. Heart attacks and strokes appear 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 heart disease and cardiovascular disease exist among active members of small-scale societies (Eaton et al. 1994), although we recognize that 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 ($VO_2\text{max}$) is high. Overall, degenerative disease accounts for 6-24% (average 9%) of deaths, with highest representation among Northern Territory Aborigines. Neoplasms and heart disease each accounted for 21% 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, Parkinsons or other forms of dementia.

Violence and warfare are variable across groups. Agta, Ache, Yanomamo and Hiwi each suffer from high levels of conspecific 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%) was accidental, with remaining groups showing 4-13% of deaths as unintentional or accidental. Together, accidental and violent deaths account for 4-69% (average 19%) 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% violent and accidental deaths, with 68% of deaths due to illness.

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

3.4.4 Traditional humans, chimpanzees and paleodemography

It is usually reported that Paleolithic humans have life expectancies of 15-20 years and that this brief lifespan persisted over thousands of generations (Cutler 1975, Weiss 1981) until early agriculture less than ten thousand years ago caused slight increases to about 25 years. Several prehistoric life tables have reinforced this trend, such as the Libben site in Ohio (Lovejoy et al. 1977), Indian Knoll (Herrmann and Konigsberg 2002) and Carlston Annis in Kentucky (Mensforth 1990). Gage (2002) compiles a set of over twelve reconstructed prehistoric life tables with similar life expectancies, and computes Siler estimates for a composite prehistoric mortality profile. This and most other prehistoric profiles show l_{50} of about 2-9% and e_{45} values of about 3-7 years. There is a large paleodemographic literature concerning problematic age estimation in skeletal samples, and bias in bone preservation among older individuals (see Buikstra 1997, see Buikstra and Konigsberg 1985, Hoppa and Vaupel 2002, Walker, Johnson, and Lambert 1988). (Howell 1976) has argued that there are many problems with the validity of prehistoric lifetables.

This literature is too large to discuss here and we direct readers to recent treatments by O'Connell et al. (1999) and Kennedy (2003).

[INSERT FIGURE 8 HERE]

Figure 8 examines five different mortality ratios as a function of age: traditional to acculturated foragers; 2) wild to captive chimpanzees; 3) traditional foragers to wild chimpanzee; 4) traditional foragers to captive chimpanzees and 5) traditional foragers to estimates from prehistoric populations. The first two comparisons reflect the effects of changed conditions. The second two comparisons allow for the assessment of the interaction of changed conditions and species differences. The last comparison 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 provides medical attention, abundant feeding and protection from predation, and shows a very large effect on infant and juvenile survival rates among chimpanzees. The effects of captivity diminish with age. Among traditional humans, the effects of improved conditions seem to be greatest during young and middle adulthood, tapering off with age.

The age-specific differences between humans and chimpanzees are quite revealing. The difference between foragers and wild chimpanzees increases dramatically with age. It is about 3 times as high during childhood, and then increases to about 9 times as high at age 45. Improved conditions for captive chimpanzees actually generate lower mortality during infancy and early childhood than among foragers. However, the species differences then overcome the effects of conditions, with captive chimpanzee mortality rates being about four times as high as foragers by age 40.

The comparison with the Siler estimates of prehistoric humans with traditional modern foragers reveals that the historical data must be incorrect (consistent with the criticisms discussed above). Mortality rates in prehistoric populations are estimated to be lower than traditional foragers until about age 4. Estimated mortality rates then increase dramatically for prehistoric populations, so that by age 45 they are over six times greater than those for traditional foragers, even worse than the ratio of captive chimpanzees to foragers! Since these prehistoric populations cannot be very different genetically from the populations surveyed here, there must be systematic biases in the samples and/or the estimation procedures at older ages where presumably endogenous senescence should dominate as primary cause of death. While it is true that excessive warfare could explain the shape of one or more of these prehistoric forager mortality profiles, it is improbable that these could represent the long-term prehistoric forager mortality profile without severe consequences for understanding life history evolution, particularly senescence rates in humans (see Discussion).

It may be possible to utilize the data from modern foragers to correct those estimates. 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:

$$\ln a_3 = -64.527b_3 - 3.0752 \quad (4)$$

Inserting (4) into the non-immature components of (1) yields

$$h(x) = a_2 + 0.046 \cdot e^{b_3(x-64.527)} \quad (5)$$

Equation (4) describes the set of mortality curves which intersect by about age 65 and explains 89% 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 twenty years earlier than in modern industrialized populations (e.g. Riggs 1990, Riggs, Hobbs, and Gerald 1998, Riggs and Millecchia 1992).

The Siler estimates of a_3 and b_3 for the prehistoric composite lifetable provided by Gage (2002) are extreme outliers from the regression equation (4). The average mortality profile for our forager populations is very similar to that of Sweden in 1751, and both 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 (4), we find that b_3 should be 0.038, or one half of what it is estimated to be by Gage. Calculation of the life table with this revised value of b_3 changes l_{50} from 4% to 24% and e_{45} from 6 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 (4) is 1/12th of Gage's estimate. A lifetable with this revised a_3 estimate shifts l_{50} to 36%, e_{45} to 18, and mean life expectancy to 33.

Critics may argue that the uniformitarian mortality assumption may not apply to prehistoric populations who tend to show relatively low infant and child mortality, but

excessively high adult mortality. If not due to erroneous assumptions about population growth and model life tables, such adult mortality would presumably be due to violence and warfare and not infectious disease nor intrinsic senescence. Infectious disease is unlikely to reach epidemic proportions in small populations. In our sample, widespread lethal infectious disease was most common in groups who had been interacting with large populations of outsiders. Intrinsic senescence is also unlikely to be much higher in the past because evidence also suggests that early and late-age mortality are closely linked among cohorts due to the cascading effects of early infections and under-nutrition (Costa 2000, Finch and Crimmins 2004, Kuh and Ben-Shlomo 1997).

3.5 Population growth and the use of modern foragers for making inferences about the past

One of the criticisms of the use of ethnographic foragers to make inferences about ancestral populations 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 varying 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 twenty three South American Amazonian groups. Additional pastoralists include the Datoga, Kipsigis, and Sebei. These were largely compiled from Hewlett (1991) and Salzano and Callegari-Jacques (1988). Although forager mortality is somewhat narrowly confined, fertility

ranges widely from below 4 up to 8 children. At the forager average where l_{15} is 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 41%. The average observed forager population shows a growth rate of about 1%. Although several groups do show 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 67% and TFR of 6.2. Their average growth rate is thus above 2%. Pastoralists have an average l_{15} of 0.72 and TFR of 5.6, with an average growth rate also above 2%.

[INSERT FIGURE 9 HERE]

Of the foraging populations, just under half show population growth rates of near zero (two are just below replacement and three are just above), and the remainder show higher population growth rates. There are at least two possible explanations of these data. 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. It is also possible that some groups during the sample period may have had greater access to food than in the past. For example, the northern Ache experienced high growth rates throughout the 20th century, and this may be due to depopulation of Guarani horticultural groups in the area, due to slave trading and warfare. The southern Ache, in contrast, were on the verge of extinction, due to increasing encroachment of 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 l_{15} 's from other foraging populations obtained from a

variety of sources mean survivorship drops only to 0.53 (s.d.=0.07)⁶. This probably means that fertility would have to have been closer to 4.5.

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

It is difficult to evaluate these two hypotheses given current knowledge about past conditions. While there is evidence suggesting that climate varied dramatically throughout the Pleistocene and including the Holocene (see Richerson, Bettinger, and Boyd 2005), the extent to which past foragers typically experienced increasing, declining or zero growth in past environments is still unclear. Perhaps some combination of the two hypotheses is the correct answer. There was likely to have been significant variance in mortality experiences of infants and children, as we see today, and variance in adult mortality due to variable violence and trauma.

4. Summary

We summarize our main findings:

- 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 twenty years.

- The modal age of later adult deaths for hunter-gatherers is in the range of 65-75 years. In this age range we find mortality rates converging across populations. This is the closest functional equivalent of an “adaptive” human lifespan.
- Life expectancies for modern foraging, and presumably ancestral populations are typically low due to high infant and child mortality, but adult mortality rates remain low through the fifth decade of life.
- After age 40, the rate of mortality increase in most populations seems to follow a Gompertz-like pattern with a MRDT of about 7-9 years. Mortality remains fairly low at around 1% from the age of maturity until around age forty, and thereafter increases exponentially. All populations show the standard bathtub J-shaped mortality profile. Departures from this general pattern in published estimates of life expectancy in historical and past populations are most likely due to a combination of high levels of contact-related infectious disease, excessive violence or homicide and methodological problems which lead to poor age estimates of older individuals and inappropriate use of model life tables.
- Illnesses account for 70%, violence and accidents 20%, and degenerative diseases 9% of all deaths. Illnesses largely include infectious and gastrointestinal disease, although less than half of all deaths in our sample are contact-related disease.
- Paleodemographic estimates of low child and high adult mortality for early humans do not match any modern pattern, and violate assumptions of uniformitarianism.
- 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, regardless of the protections afforded by captivity among chimpanzees.

5. Discussion and Conclusions: The evolved human lifespan

A fundamental conclusion to be drawn from this analysis is that extensive longevity appears to be a novel feature of *Homo sapiens*. Our results contradict Vallois' (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 lifespan. The data show that modal adult lifespan is about 65-75 years, and that it was not very uncommon for individuals to reach these ages, suggesting that inferences based on paleodemographic reconstruction are unreliable. One recent study that avoided several common problems of skeletal aging used dental wear-based seriation and relative macro-age categories (ratio of old to young), and showed 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).

We conclude the paper with a brief discussion of lifespan evolution with respect to the demographic portrait of hunter-gatherers we have described in this paper. Two issues are addressed. The first is why modal lifespans are about 7 decades, and not shorter or longer. The second issue is how to best conceive of lifespan evolution, especially in relation to the gene-environment interactions that determine age distributions of death at the population level.

5.1 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 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, mothers may benefit more from investing their energy and resources in existing descendants rather than from producing new ones. This idea became known thirty years later as the “grandmother hypothesis” (Hill and Hurtado 1991). A specific version of the grandmother hypothesis proposed by Hawkes, Blurton-Jones and O’Connell (1998a) 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 and increasing the value of the older women’s contributions. According to this view, extensions in the human lifespan 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 40’s 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 (Gurven and Kaplan 2006, Kaplan et al. 2000, Kaplan and Robson 2002) extends these ideas to both sexes in a formal life history model, relating intergenerational transfers to the ecology of the hunting and gathering lifeway. 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 thereby 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 the adult period. Since productivity increases with age, the time investment in skill acquisition and knowledge leads to selection for lowered mortality rates and greater longevity, because the returns on the investments in development occur at older ages. According to this model, the long human lifespan 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, which results in delayed productivity until older ages, selecting for lifespan extension in both sexes.

Each of these models relates the benefits of longevity to fitness effects achieved through transfers to dependent descendants. Indeed, an increasing number of studies focusing on the benefits of older individuals have found significant fitness effects via increased child and grandchild survivorship and/or fertility (e.g. Beise and Voland 2002, Hawkes et al. 1998b,

Jamison et al. 2002, Lahdenpera et al. 2004, Sear, Mace, and McGregor 2000, Sear et al. 2002). The demographic data derived from foragers and forager-horticulturalists allow us to assess how those benefits can change with age. Panels A and B of Figure 10, derived from data on Tsimane forager-horticulturalists, compare age-specific numbers of dependent descendants with mortality rates. Panel A shows the weighted sum of children and $\frac{1}{2}$ 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). Panel B shows age-specific mortality rates. Even though a woman still has descendants who could benefit from assistance, the number of offspring and grandoffspring, especially dependents less than 18 years old, drops considerably after about 65 years. This is the point when mortality begins to rise precipitously. The late age decline in dependents is similar to the modal age at death from Figure 4. In another paper (Kaplan et al. 2006), we tabulate actual flows of food and show that both 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 most support for the embodied capital hypothesis.

[INSERT FIGURE 10 HERE]

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

5.2 Gene-environment interactions and lifespan evolution

How can we reconcile the idea of an evolved lifespan with the evidence that mortality distributions are influenced by environmental conditions? The answer to this question may also shed light on the recent debate over modern changes in human lifespan. While average life expectancy has changed significantly over recent history, it is an open question whether gains will continue linearly, and whether maximum lifespan itself will still increase (Vaupel 1997; Wilmoth 1997).

One view is that there is a fixed upper limit to the human lifespan 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 lifespan and that improvements in medical care, treatments, and living conditions will continue to produce increases in longevity (Wilmoth 1997). Wood et al. (1994) characterize these two views as gerontological vs. epidemiological, respectively. The gerontological view treats the various outcomes of aging, as resulting from a single unitary process, leading to programmed death; whereas 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. (1994) argue against the notion of a unifying species-typical MRDT (cf. Finch, Pike, and Whitten 1990), citing MRDT estimates varying from 3-33 for different causes of senescent death, such as atherosclerosis, emphysema and cirrhosis.

We suggest that neither view is correct, and that a hybrid of the two approaches is more productive. It is important to consider not only the benefits of lifespan extension, but the costs as

well. Aging can be thought of as resulting from the combined effects of deleterious byproducts of metabolism itself and from environmental assaults. Natural selection will favor optimal effort at repairing the damage produced by both those 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 trumps productivity, and extremely high productivity of adults, especially in middle age. Together, the costs of slowing senescence and mortality prevention, and the benefits of extended investment in descendants produced selection for a characteristic human lifespan, with some variance around the central tendency. The comparison of the data from 18th century Sweden to the hunting and gathering populations suggest that relatively similar age distributions of adult deaths occur under a relatively broad range of environmental conditions.

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

We do not yet understand the mechanisms underlying the effects of modernization. Do members of industrialized countries senesce more slowly, in a physiological sense, than people exposed to higher assault environments? Alternatively, are most of the mortality improvements due to reductions in cause-specific mortality at specific ages through prevention of assaults or medical treatment of illnesses? Is a 50 year-old Hadza as robust and functional as a 50 year-old American? It has been argued that aging and the onset of chronic disease is accelerated in response to poor nutrition, infectious disease, and chronic inflammatory processes in general (Bengtsson and Lindstrom 2000, Blackwell, Hayward, and Crimmins 2001, Elo and Preston 1992). For example, there is increasing evidence that chronic diseases, such as diabetes, occurred at earlier ages in the 19th century in the U.S. than occur today (Fogel and Costa 1997). In contrast to the U.S., 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 associates with disease load and presence of parasites. There is also increasing evidence that malnutrition and health insults during fetal and peri-natal development produce a set of 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. Nevertheless definitive answers to these questions await further research.

The chimpanzee-human comparison does suggest, however, that species differences tend to overwhelm differences in environmental conditions in determining mortality hazards as individuals age. This might suggest that some differences in our respective genomes have resulted in basic differences in rates of repair and tissue maintenance that manifest themselves in physiological deterioration at older ages. It may be that when those differences are understood

and then subject to manipulation, there will be resulting changes in human mortality distributions at old ages that greatly exceed those due to disease treatment and assault prevention.

Figure Legends

FIGURE 1. Survivorship curves. Survivorship (l_x) for a) hunter-gatherers, b) forager-horticulturalists, 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 Sweden, 1751-59.

FIGURE 2. Mortality rates. Log mortality hazards ($\ln(h_x)$) for a) hunter-gatherers, b) forager-horticulturalists, c) acculturated hunter-gatherers, and d) same group averages from Figure 1. All estimates are based on raw data clumped into 5 year age categories.

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

FIGURE 4. Modal ages at death. Frequency distribution of ages at death $f(x)$ for individuals over age 15 show strong peaks for hunter-gatherers, forager-horticulturalists, acculturated hunter-gatherers, Sweden 1751-59, and the United States 2002. All curves except for the U.S. are smoothed by using on Siler estimates.

FIGURE 5. Mortality rate extremes. Ratio of Siler-estimated mortality hazards (h_x) for hunter-gatherer composite sample and the U.S. 2002

FIGURE 6. Juvenile survivorship and old age life expectancy. Regression line estimates of life expectancy at age 65 (e_{65}) and survivorship to age 15 (l_{15}). Relationship shown for our sample of small-scale populations, and for Sweden (using longitudinal cohort data from 1751-2003) and Denmark (from 1835-2002)

FIGURE 7. Diachronic changes in mortality within populations. The ratio of estimated mortality hazards from post-contact or acculturated time periods 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 lifespan, while (b) shows those with higher survivorship before contact and interaction with outsiders.

FIGURE 8. Ratio of mortality hazards for humans and chimpanzees. 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.

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}). Each data point refers to a single population.

FIGURE 10. Age-specific dependency and adult mortality. Number of children and $\frac{1}{2}$ number of grandchildren by age of a Tsimane woman (top panel) compared against age-specific mortality rate for Tsimane (bottom panel).

Table 1. Sample of study populations

Group	Type	years studied	years covered	# individuals	per-yrs	# deaths	per-yrs over 50	age intervals	data collection	Data source
<i>Hunter-gatherers</i>										
!Kung	HG	1963-74	<1968, 1963-74	500	1,767	96	<17	5,10	retro	Howell 1979 Table 4.1
Ache	HG	1980-94	<1971	439	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	106	3,565	107	341	1	retro	Hill personal communication
<i>Forager-horticulturalists</i>										
Yanomamo Xilixana	FH	1959-95	1930-56	120	2,843	64	180	5,10	retro	Early and Peters 2000
Yanomamo Xilixana "Brazilian"	FH	1959-95	1982- 1996	328	4,420	124	279	5,10	prosp / retro cens model	Early and Peters 2000
Yanomamo	FH	1960's	1960's	2,513	N/A	N/A	N/A	5	fit	Neel and Weiss 1975
Tsimane	FH	2002-03	1950-89	1,702	55,990	916	2,785	1	retro	Gurven, unpublished
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 1987; 1980
<i>Acculturated foragers and others</i>										
!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	1962-86	1980-94	212	3,185	147	223	5,10	prosp	Early and Headland 1998, Table 8.1
Warao	A-HG	1954,66, 72-73	1954,66 ,72-73	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 prosp vital	Lancaster Jones 1961; Crotty and Webb 1960
Tiwi	A-HG	1952-61	1952-61		7,243	150		5,10,15	regist	Lancaster Jones 1963
Hiwi	A-HG	1988-90	1961-89	106	4,370	131	614	1	retro	Hill et al. 2006
Ache (settled)	A-HG	1980-94	1978-93	962	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

Table 2. Parameter estimates of Siler mortality model

Population	a ₁	b ₁	proportion surviving immature	a ₂	a ₃	b ₃	MRDT	MRDT 95% C.I. lower	MRDT 95% C.I. upper	l ₁₅	l ₄₅	e ₁₅	e ₄₅
Hadza	0.195	0.387	0.60	0.0072	0.0002	0.083	8.4	7.4	9.7	0.54	0.41	44.4	23.8
Ache forest	0.093	0.347	0.77	0.0095	0.0002	0.084	8.3	7.2	9.8	0.66	0.45	40.0	21.1
Hiwi	0.866	1.813	0.62	0.0160	0.0000	0.149	4.6	3.0	10.1	0.56	0.30	32.3	18.2
!Kung	0.322	0.874	0.69	0.0108	0.0001	0.100	7.0	5.5	9.5	0.59	0.40	39.9	21.2
<i>Agta</i>	0.834	1.314	0.53	-0.0319	0.0276	0.023	30.1	14.4	-	0.52	0.26	30.9	14.5
Average Hunter-Gatherer	0.462	0.947	0.61	0.0109	0.0056	0.088	7.9			0.58	0.36	37.5	19.8
Yanomamo Mucaj	2.965	16.851	0.84	0.0110	0.0002	0.076	9.2	6.6	15.0	0.71	0.48	41.6	24.2
<i>Yanomamo</i>	0.912	2.020	0.64	0.0104	0.0086	0.027	25.5	11.6		0.46	0.19	27.8	17.4
Tsimane	0.211	1.156	0.83	0.0080	0.0001	0.096	7.2	6.9	7.6	0.74	0.54	41.4	20.7
<i>Machiguenga</i>	0.285	1.056	0.76	0.0133						0.63	0.42		
<i>Gainj</i>	0.262	0.872	0.74	0.0028	0.0026	0.063	11.1	9.6	13.1	0.67	0.34	30.2	12.2
Average Forager-Horticulturalist	0.927	4.391	0.81	0.0091	0.0029	0.065	10.6			0.64	0.39	35.2	18.6
!Kung 1963-73b	0.228	0.806	0.75	0.0022	0.0000	0.099	7.0	5.8	8.9	0.73	0.66	52.7	26.5
Ache reservation	0.221	0.835	0.77	0.0026	0.0001	0.092	7.5	5.2	13.8	0.74	0.65	52.3	26.9
Northern Territory Aborigines	0.231	1.043	0.80	0.0015	0.0004	0.072	9.6	9.0	10.3	0.78	0.65	48.0	23.4
Hiwi post-1960	0.451	0.760	0.55	0.0109	0.0000	0.117	5.9	3.7	15.8	0.47	0.33	42.1	23.4
<i>Tiwi</i>	0.129	0.557	0.79	0.0052	0.0000	0.173	4.0	1.0		0.73	0.62	44.3	19.7
<i>Agta transition</i>	0.397	0.848	0.63	0.0104	0.0000	0.163	4.3	3.1	7.0	0.54	0.38	37.2	16.2
<i>Agta peasant</i>	0.390	0.502	0.46		0.0030	0.056	12.4	11.3	13.8	0.43	0.25	33.7	14.5
Average Acculturated HG	0.292	0.764	0.68	0.0055	0.0005	0.110	6.3			0.63	0.51	44.3	21.5
Sweden 1751-59	0.369	0.999	0.69	0.0081	0.0002	0.084	8.3	7.8	8.8	0.61	0.43	41.3	21.4
OVERALL HUMAN AVERAGE	0.529	1.885	0.76	0.0056	0.0027	0.092	7.5			0.62	0.43	39.9	20.3
Wild chimpanzees	0.222	0.385	0.56	0.0177	0.0072	0.071	9.8	7.0	16.1	0.35	0.02	14.8	4.2
Captive chimpanzees	0.494	2.859	0.84	0.0185	0.0005	0.109	6.3	6.0	6.7	0.63	0.20	23.0	7.3

MRDT=mortality rate doubling time, defined as $\ln 2/b_3$

TABLE 3. Modal ages at death

Population	Modal age at death	standard deviation	% of adult deaths at mode	% adult deaths at and above mode
Hadza	73	8.1	2.2	29.1
Hiwi	70	7.4	2.5	10.8
Ache	69	8.9	2.1	28.1
Yanomamo Xilixana	75	7.6	1.7	22.3
Tsimane	69	7.8	2.5	28.9
!Kung 1963-74	70	7.6	2.6	29.5
Ache reservation	77	6.3	3.1	32.4
Aborigines	71	8.8	2.6	35.8
Wild chimpanzees	15	16.8	3.9	100.0
Captive chimpanzees	45	7.9	2.7	31.9
SWEDEN 1751-59	72	7.4	2.3	24.3
US 2002	85	1.7	3.5	35.3

TABLE 4. Model of log mortality hazard $\ln h(x)$ by age

<i>Group</i>	Age 15-40					Age 40+				
	$\acute{\alpha}$	P	$\exp(\beta)$	R^2	MRDT	$\acute{\alpha}$	P	$\exp(\beta)$	R^2	MRDT
Ache	0.0006		0.0113	0.00	1216.0	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
<i>Average Hunter-Gatherer</i>	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
<i>Average Forager-Horticulturalist</i>	0.0188		0.0156		36.9	0.0603		0.0224		11.5
!Kung 1963-74b	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	-				
<i>Average Acculturated HG</i>	0.0451		0.0050		15.4	0.0747		0.0126		9.3
<i>Overall average</i>	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 $\ln 2 / \acute{\alpha}$

In the Gompertz model, $\ln[h(x)] = \beta + \acute{\alpha} * x$

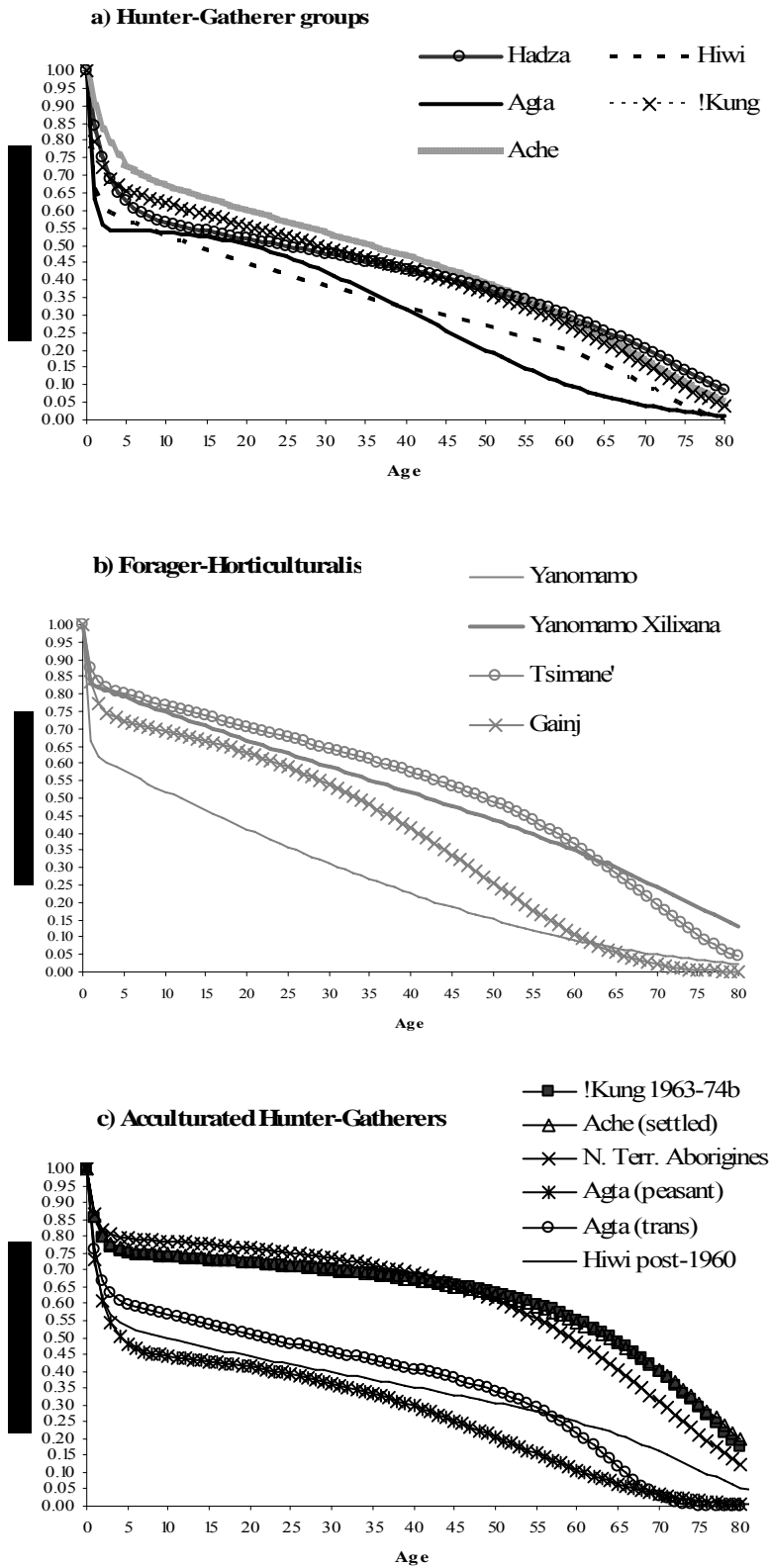
‡ Parameter estimates for 40+ regression are based on small samples or age estimation problems (see text)

TABLE 5. Causes of death among study populations

	(1)	(2)	(3a)	(3b)	(4)	(5)	(6)	(7)	(8)	(9)	(10)	(11)	(12)	TOTAL		
	Hadza	Yanomamo	Ache forest	Ache settled	!Kung	Tsimane	Aka	Agta	Hiwi	Machig.	North Territ.	Baikari	Gainj	#	%	
	%	%	%	%	%	%	%	%	%	%	%	%	%			
a) <15 yrs old			(n=230)	(n=84)	(n=164)	(n=423)		(n=112)	(n=94)	(n=82)	(n=74)					
all illness			22.2	65.5	87.8	79.9		95.5	44.8	63.8	67.6			825	65.3	
degenerative			8.3	20.2	3.7	10.4			10.4	9.6	24.3			120	9.5	
accidents			6.1	3.6		9.7		1.8	15.6	11.7	6.8			102	8.1	
Violence			63.5	10.7	8.5	7.4		2.7	27.1	2.1	1.4			216	17.1	
b) 15-59 yrs old			(n=125)	(n=22)	(n=127)	(n=192)		(n=77)	(n=31)	(n=19)	(n=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			(n=27)		(n=52)	(n=60)				(n=2)	(n=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	(n=125)	(n=111)	(n=382)	(n=104)	(n=343)	(n=690)	(n=669)	(n=364)	(n=139)	(n=117)	(n=175)	(n=65)	(n=44)			
respiratory		21.6	0.8	31.1		19.9				6.8	28.6	56.9		292	23.7 ^a	
gastrointestinal															13.8P ^a	
		5.4	5.5	13.2		18.2				34.2	17.1	3.1		239	^a	
Fever		6.3	8.1	21.7		5.7				1.7	0.0	7.7		107	7.3 ^a	
Other		40.5	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	2333	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	

^aCategory average includes only groups for which data are available; Unknown deaths for Hadza, Agta and Aka distributed among illness categories; Unknown deaths for Baikari and Tsimane left out of analyses

FIGURE 1.



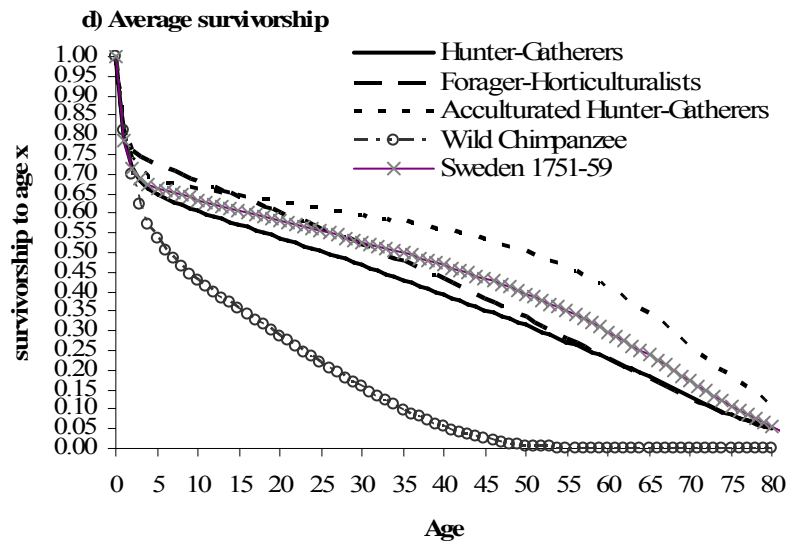


FIGURE 2.

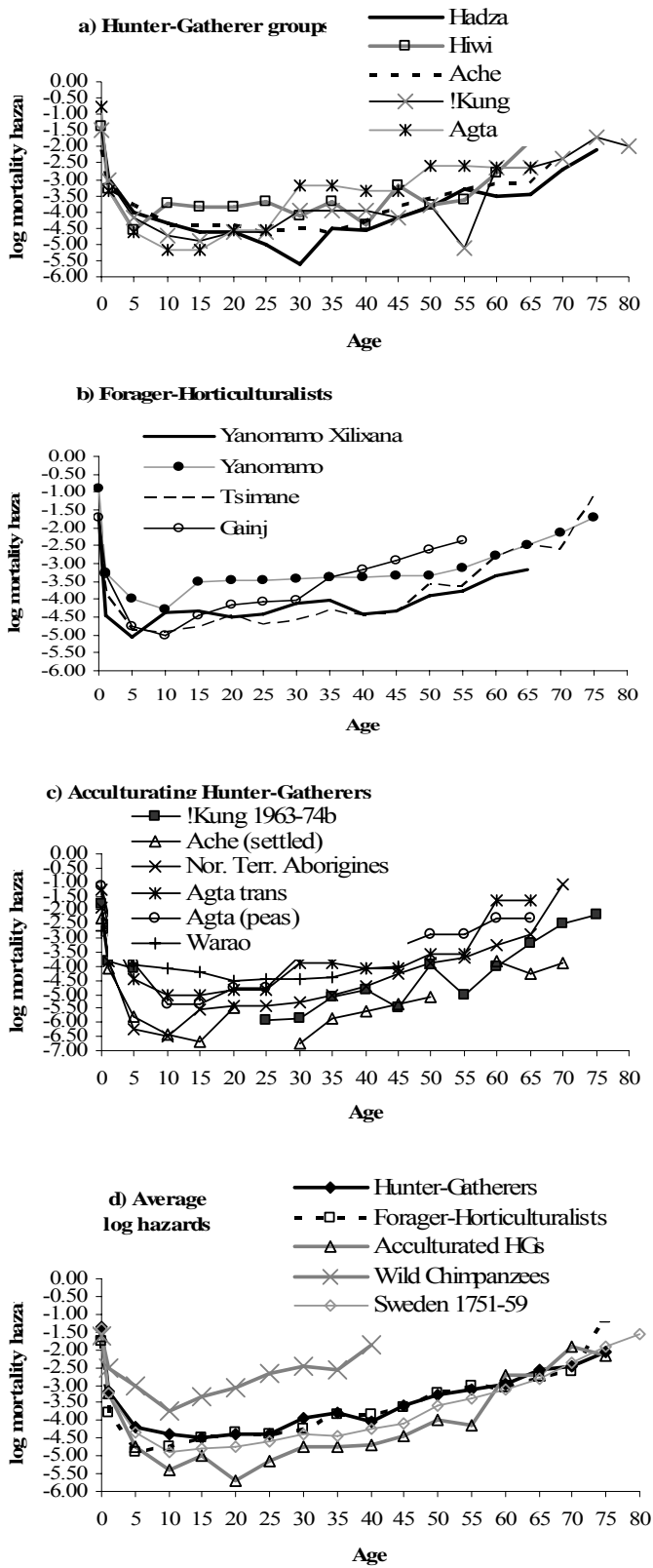


FIGURE 3.

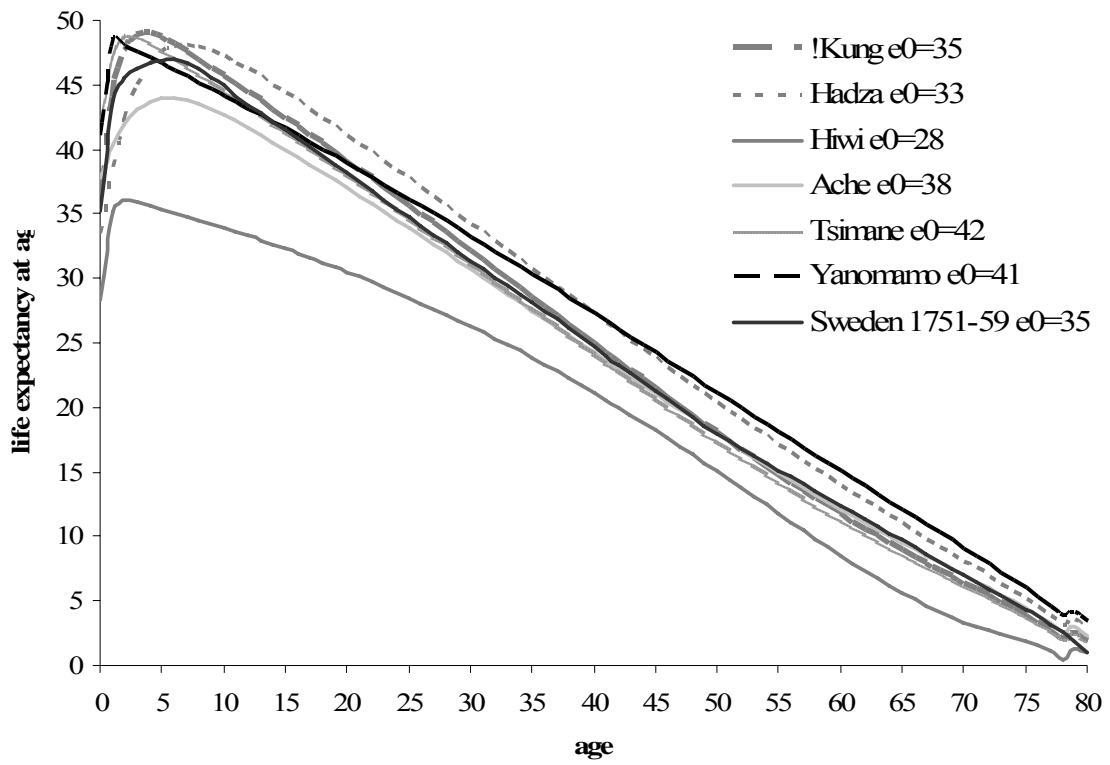


FIGURE 4.

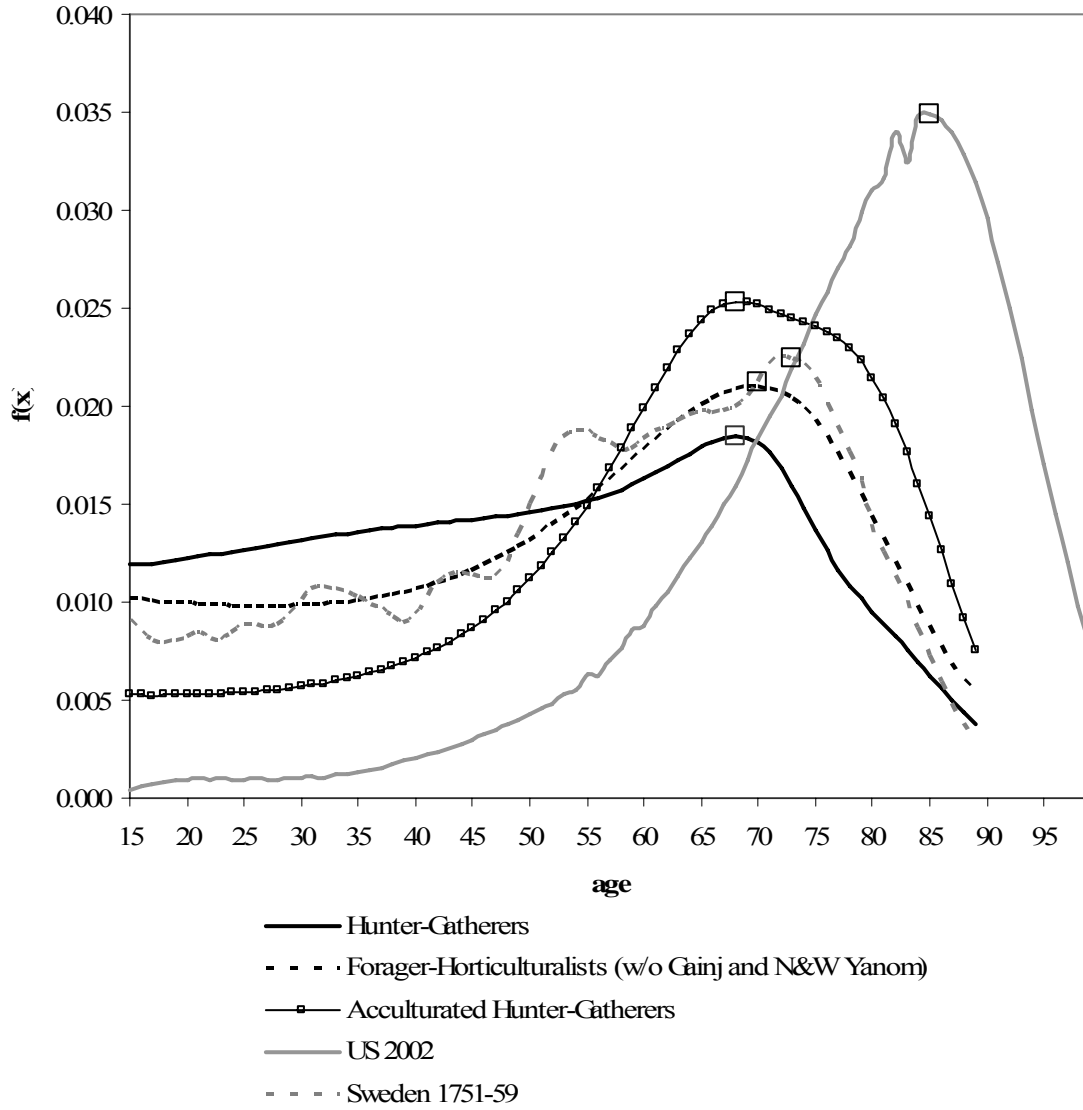


FIGURE 5.

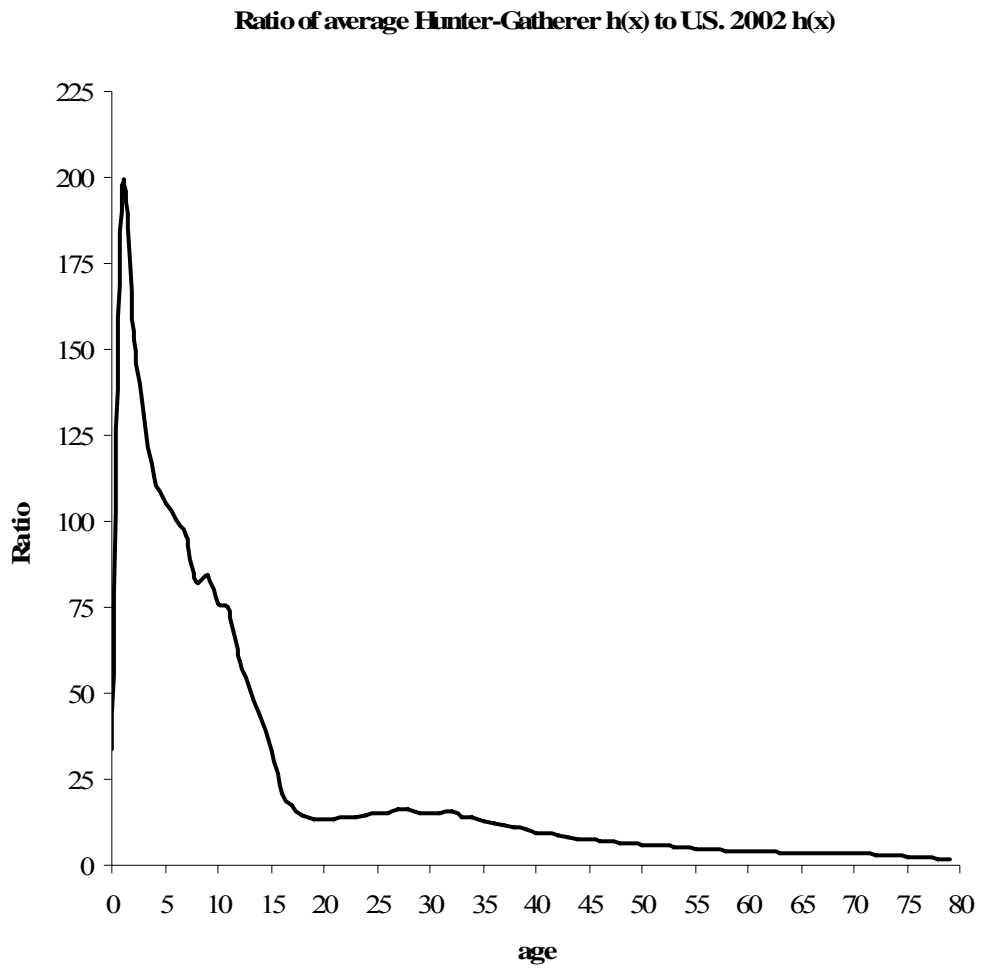


FIGURE 6.

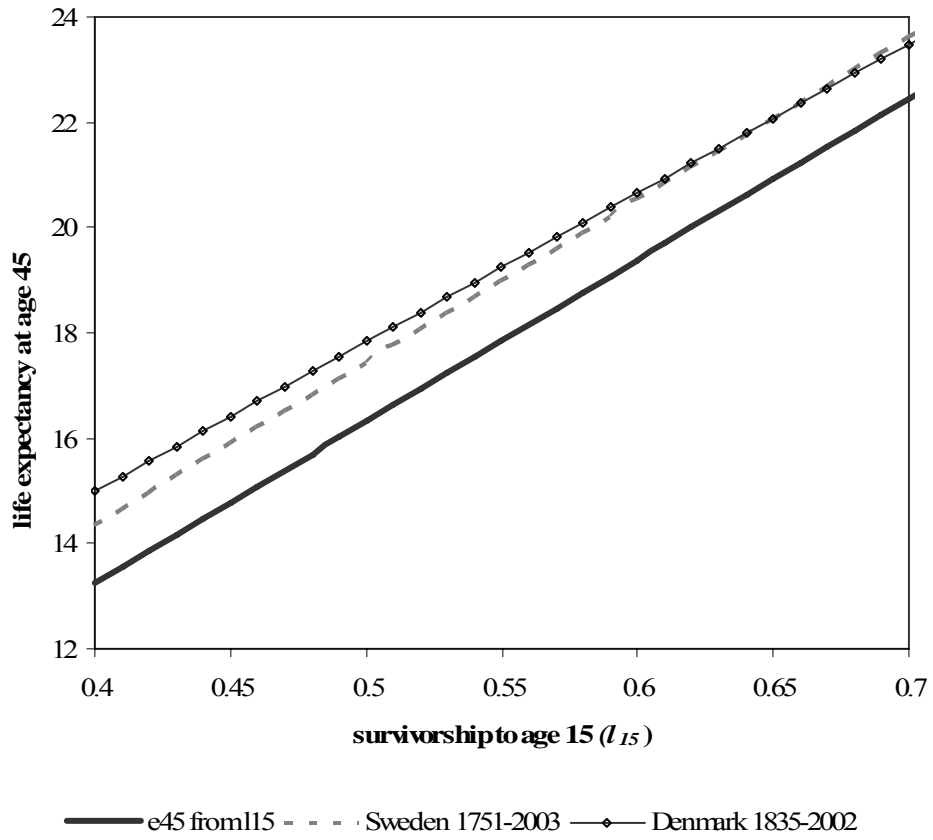


FIGURE 7.

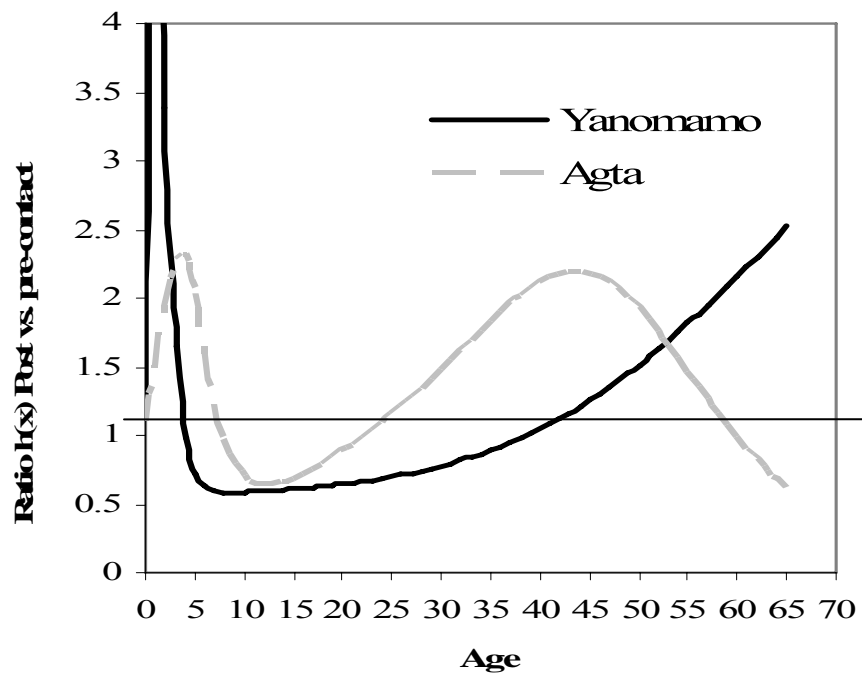
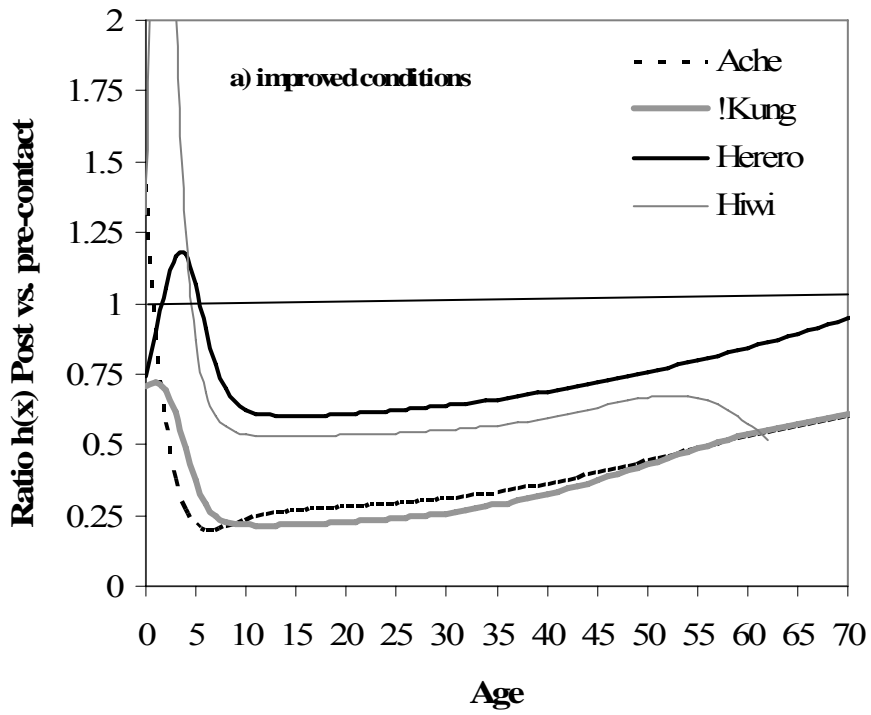


FIGURE 8.

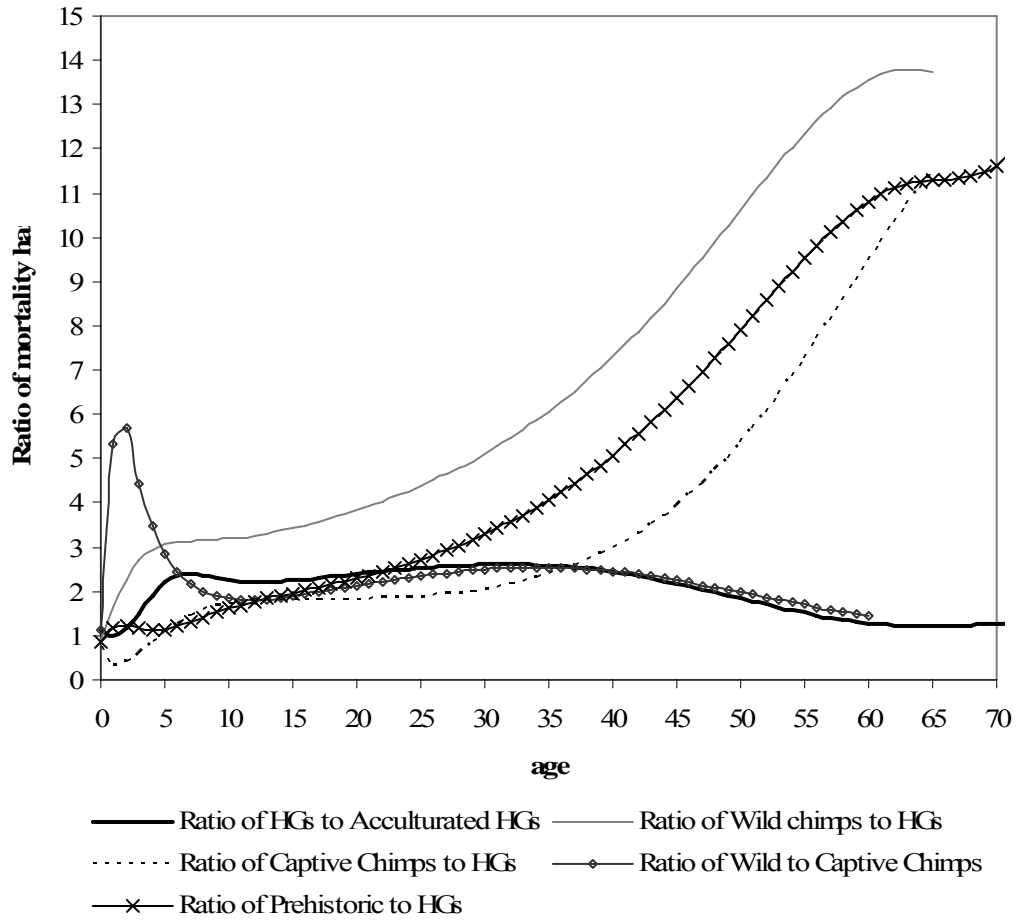


FIGURE 9.

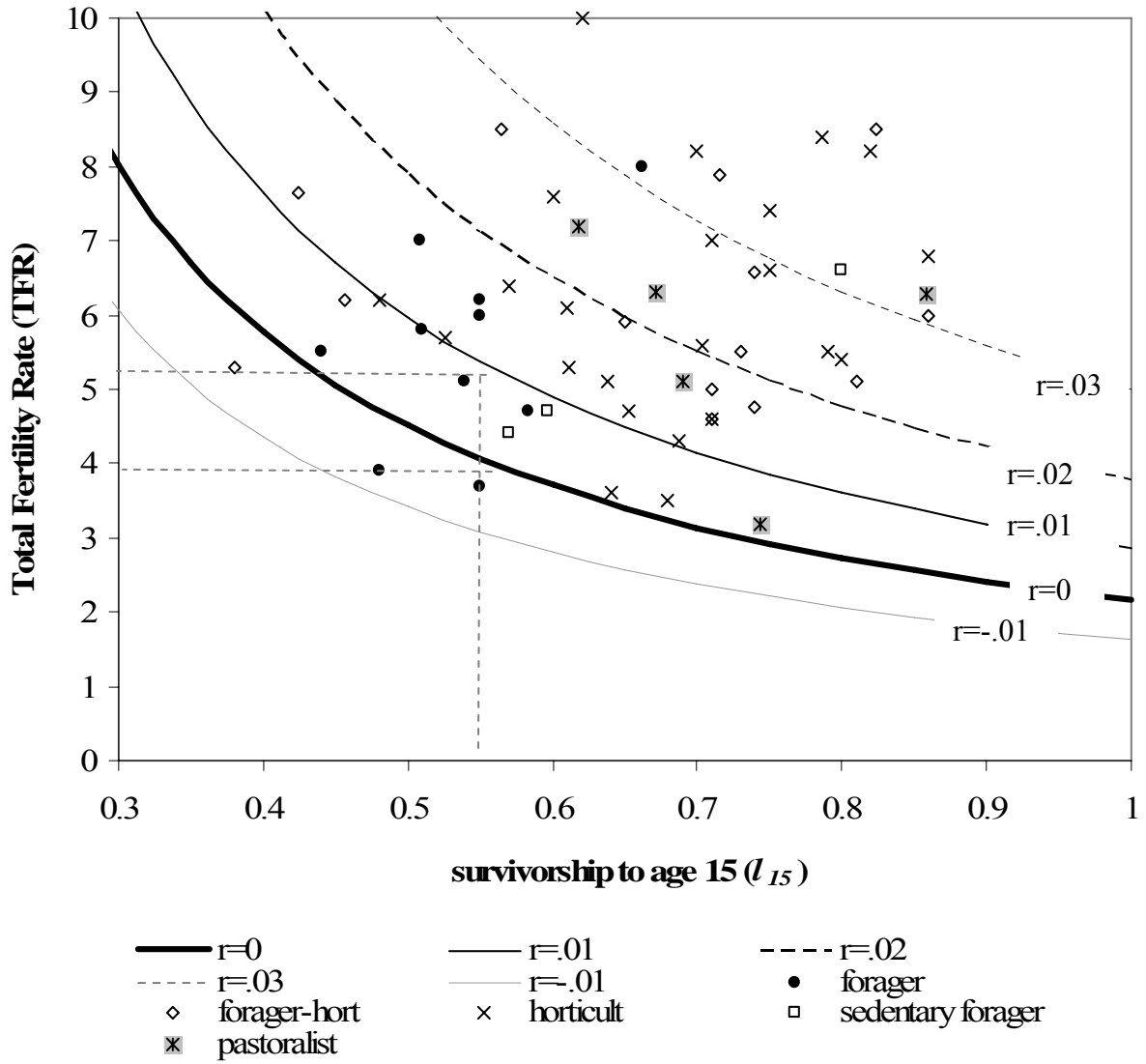
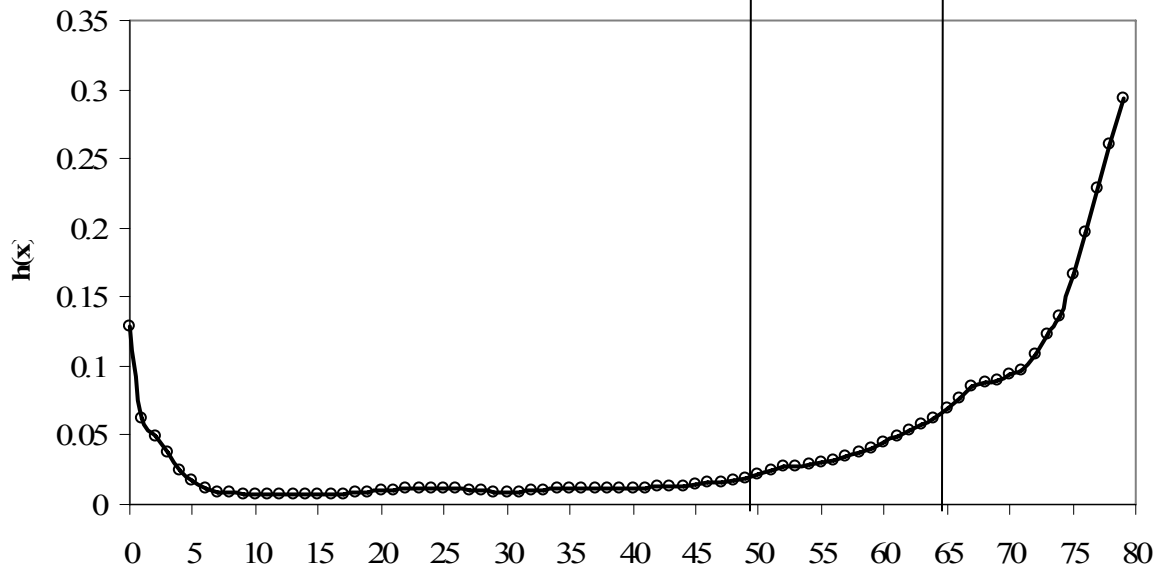
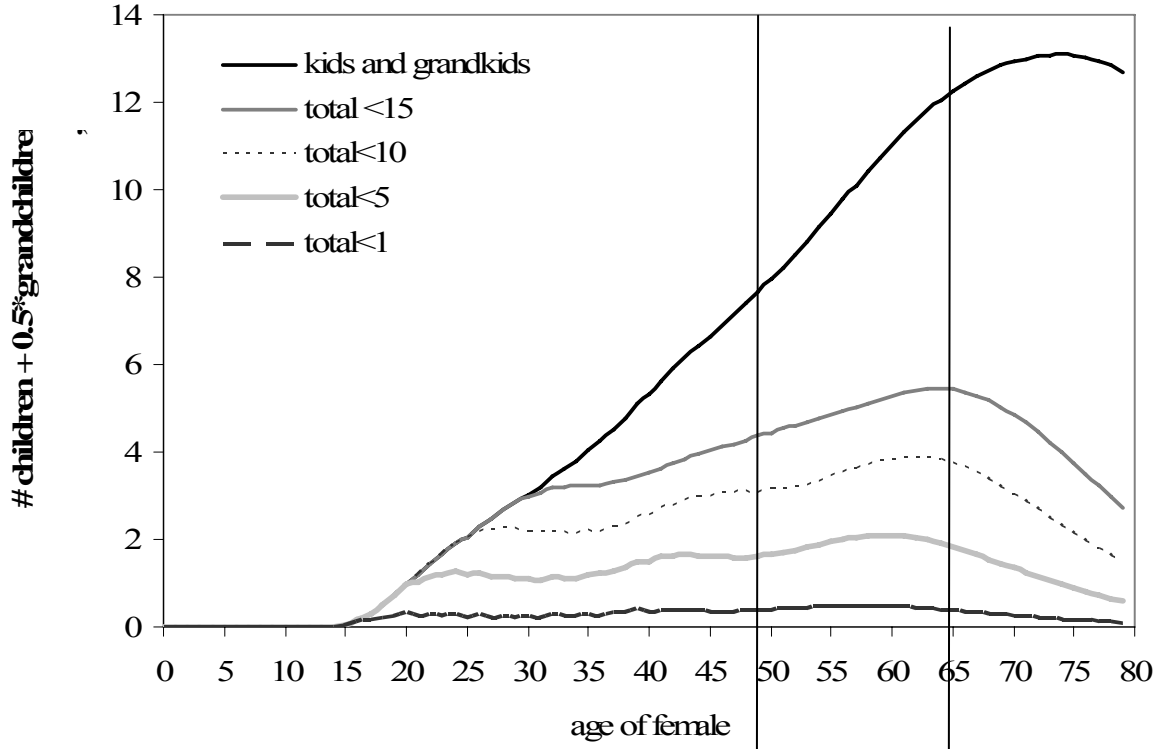


FIGURE 10.



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¹ 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).

² 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, T. B., and B. Dyke. 1986. Parameterizing abridged mortality tables: the Siler three-component hazard model. *Human Biology* 58:275-291. Correspondingly, the senescence rate (b_3) is one-fourth that of other foragers.

³ These analyses, however, did not control for age-independent mortality (a_2), a factor which could bias Gompertz estimates Gavrilov, L. A., and N. S. Gavrilova. 2001. The reliability theory of aging and longevity. *Journal of Theoretical Biology* 213:527-545.

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, defined 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, S. Editor. 1979. *Pygmées de Centrafrique: études ethnologiques, historiques et linguistiques sur les Pygmées "BaMbenga" du Nord-Ouest du bassin congolais*. Paris: SELAF., Bofi Pygmies (0.59) Fouts, H. N., B. S. Hewlett, and M. E. Lamb. 2005. Parent-offspring weaning conflicts among the Bofi farmers and foragers of Central Africa. *Current anthropology* 46:29-50. Mbuti Pygmies (0.44) Harako, R. 1981. "The Cultural Ecology of Hunting Behavior among the Mbuti Pygmies in the Ituri Forest, Zaïre," in *Omnivorous Primates: Gathering and Hunting in Human Evolution*. Edited by R. S. O. Harding and G. Teleki, pp. 499-555. New York: Columbia University Press., Batak (0.48) Eder, J. F. 1987. *On the Road to Tribal Extinction: Depopulation, Deculturation, and Adaptive Well-Being among the Batak of the Philippines*. Berkeley: University of California Press., Australian Aborigines (0.55), Seri (0.39) (cited in Weiss 1973).