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ScienceDirect
Journal of Human Evolution 52 (2007) 443–454

High adult mortality among Hiwi hunter-gatherers: Implications for human evolution

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Received 31 March 2006; accepted 29 November 2006

Abstract

Extant apes experience early sexual maturity and short life spans relative to modern humans. Both of these traits and others are linked by life-history theory to mortality rates experienced at different ages by our hominin ancestors. However, currently there is a great deal of debate concerning hominin mortality profiles at different periods of evolutionary history. Observed rates and causes of mortality in modern hunter-gatherers may provide information about Upper Paleolithic mortality that can be compared to indirect evidence from the fossil record, yet little is published about causes and rates of mortality in foraging societies around the world. To our knowledge, interview-based life tables for recent hunter-gatherers are published for only four societies (Ache, Agta, Hadza, and Ju/'hoansi). Here, we present mortality data for a fifth group, the Hiwi hunter-gatherers of Venezuela. The results show comparatively high death rates among the Hiwi and highlight differences in mortality rates among hunter-gatherer societies. The high levels of conspecific violence and adult mortality in the Hiwi may better represent Paleolithic human demographics than do the lower, disease-based death rates reported in the most frequently cited forager studies.

Keywords: Hunter-gatherer demography; Mortality rates; Human life history; Hiwi (Venezuela)

Introduction

Age-specific mortality rates are invoked in theoretical explanations of a multitude of human biological and behavioral characters. This includes comparisons between mortality in humans and other primates as an explanation of slow childhood development, late age at first reproduction, menopause, delayed senescence, and brain expansion in humans (e.g., Hawkes et al., 1998; Blurton Jones et al., 1999; Kaplan et al., 2000; Hill et al., 2001). Likewise, high mortality rates in some human groups have been implied to result in atypical childhood growth patterns (Walker et al., 2006), shortened life span due to early infections (Finch and Crimmins, 2004), changed marriage and mating patterns (Low, 1988, 1990; Hill and Hurtado, 1996), and accelerated sexual maturity and associated medical and behavioral problems (Draper and Harpending, 1982; Belsky et al., 1991; Ellis, 2004). In addition, high adult mortality has extensive implications for human social structure and settlement patterns (Howell, 1982; Early and Peters, 1990). The origin of humanlike mortality profiles, the biological vs. cultural causes of increased longevity, and the implications for human evolution have been hotly debated in recent years (e.g., Caspari and Lee, 2004, 2005a,b, 2006; Hawkes and O'Connell, 2005).

In order to evaluate models of human biology, and in order to understand the adaptive pressures that helped shape human traits, we need to document the causes and rates of mortality during different periods of human evolution. Although the mortality profiles of our distant ancestors can only be ascertained through indirect means, studies of modern hunter-gatherers provide direct information on the likely mortality profiles of more recent ancestors. While it has long been recognized that hunter-gatherers show different health profiles from people living in modern societies (e.g., references in Dunn,

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doi:10.1016/j.jhevol.2006.11.003

1968), systematic demographic studies began only with Howell's (1979) seminal research on the !Kung. In order to apply these studies to problems in human evolution, we must carefully consider whether their results reflect parameters typical of past forager populations or if these parameters have been significantly altered by subtle current conditions (see Blurton Jones et al., 2002).

Hunter-gatherers have very different lives than people in the modern societies. They are exposed to climatic challenges, risks from foraging activities, predation, nutrient and pathogen stress, violence from conspecifics, and other insults from a difficult lifestyle that is more typical of human ancestors and without the benefit of any modern medical treatments (Hill and Hurtado, 1996). Mobile hunter-gatherers often must recover or perish in a short time period before the residential band is forced to move on in search of food. Moreover, hunter-gatherers, because they live in small, mobile residential units, may be less exposed to infectious diseases that require large and dense groups and more exposed to zoonotic infectious agents and trauma than most sedentary human populations. This may result in a different immune profile than that found in sedentary and state-level societies (Hurtado et al., 2003). Hunter-gatherers eat a diet high in protein-lipid, low in carbohydrate (and very low glycemic load), low in salt, and high in fiber relative to modern people (Eaton and Konner, 1985; Cordain et al., 2000), and they also experience high rates of activity through much of the life span. They are minimally exposed to factors such as chemical toxins, hormone and antimicrobial animal-feed additives, and electromagnetic waves. Hunter-gatherer women experience a very different lifetime hormonal profile, with many pregnancies and long periods of lactation and anovulation, that appears to affect rates of reproductive cancers (Eaton et al., 1988, 1994). Finally, hunter-gatherers may experience very different psychological conditions (high social support but constant threat of violence) than people in modern societies. The suite of all these different factors suggests that hunter-gatherer mortality profiles might be very different from those of people in modern or even ancient horticultural, agricultural, and state-level societies. Because of this, it is critical that we document mortality rates in as many hunter-gatherer societies as possible before these groups cease to exist. This will allow us to seek out patterns common to foraging societies and assess the range of variation in mortality among these groups in order to determine the impact of factors specific to certain world regions, ecozones, or cultural practices.

The Hiwi

The Hiwi (often called Cuiva) are a subpopulation of the Guahibo-speaking peoples of Venezuela and Colombia. They live in the vast savannah plains referred to as the ''llanos.'' Although most Guahiban speakers were horticultural trekkers in the nineteenth and twentieth centuries (e.g., Metzger and Morey, 1983), a dialectal subpopulation of about 800 individuals living in the headwaters of the Capanaparo, Cinaruco, and Meta river drainages continued to live as hunter-gatherers until the late twentieth century (Kirchoff, 1948; Arcand, 1976; Fonval, 1976). The habitat of this area is predominantly grassland and swamps with patches of gallery forest, and the weather is characterized by extreme seasonality in rainfall. The Venezuelan Hiwi that we studied were dependent on hunting riverine mammals, turtles, and fish, and collecting wild roots and fruits during our field observation periods (for description of ecology and subsistence patterns, see Hurtado and Hill, 1987, 1990).

The Hiwi in our study population initiated first peaceful contact with Venezuelan nationals in 1960 and were subsisting from hunting and gathering along the upper Capanaparo and Sinaruco rivers at that time. Prior to direct peaceful contact, the Hiwi had obtained metal goods through trade with Pume (Yaruro) farmers and were thus likely exposed indirectly to pathogens of European origin for at least a century. Although there are large ceramic-bearing archeological sites along the rivers currently occupied by the Hiwi, it is not known whether these sites were created by a more sedentary Hiwi population of the past, by farmers such as the Pume who now live downstream, or by some vanished group no longer present in the area. At the time of peaceful contact in Venezuela, two politically independent and mutually hostile Hiwi groups (containing several residential bands each) were settled together on a government reservation. They soon split into three residential settlements, and small-scale violence between the groups continued without outside interference. The Hiwi were also victims of violence at the hands of ranchers invading their territory throughout the twentieth century (including the reservation period), and they occasionally conducted revenge raids on the ''criollo'' population. Members of the study population described here were victims of the ''Rubiera Massacre'' carried out on a Columbian ranch on Christmas Day, 1968, which resulted in the deaths of 16 men, women, and children (and left only two survivors who were interviewed as part of this demographic study).

Because the Hiwi's home range was drastically curtailed in the late twentieth century, malnutrition and associated diseases were commonly observed, and the health profile of the population was rather poor during the study and data-collection period. People frequently complained of hunger, children and adults were sometimes lethargic, parasite loads were high, and hematocrit values were very low (Hurtado and Hill, 1987; Hurtado et al., 1997, 2003). Most adult men took hallucinogenic drugs on a daily basis and accidents (possibly related?) were common. Most importantly, the threat of violence from both within and outside the ethnic group was a constant topic of conversation and concern, and a half dozen Hiwi from our study population were killed in the 1980s and 1990s. The threat of violence was so serious that Hiwi men almost invariably took weapons with them or stood guard while family members relieved themselves in the bush surrounding permanent camps. Despite this rather bleak situation, we discovered, through systematic interviews, that mortality rates had been even higher in the precontact period.

Methods

Data on individuals were ascertained by repeated census and reproductive-history interviews from September 1985 to January 1992. We completed six field sessions during this time for a total of 16 months with the Mahünemuthu group of Hiwi. Although KH and AMH became reasonably fluent in the Hiwi language during the course of fieldwork, all data were collected with the assistance of a bilingual informant (Hiwi-Spanish) to ensure accuracy. We used our linguistic proficiency to verify that questions and answers were interpreted correctly. Data on 779 individuals were entered into our demographic database during this period; 427 of those individuals had died by January 1992, and 17% of the reported deaths were from causes unknown to our informants. Because another small percentage could not be assigned an accurate year of death, we were left with 722 individuals, whose age, sex, and year of death were reported.

Causes of death were tabulated by reported symptoms rather than cultural interpretations of cause (e.g., we ignored witchcraft, etc., and focused on manifest symptoms). These were aggregated into categories and then assigned to one of four major classes: disease, degenerative/congenital problem, accident, and violence. Disease included infectious disease (respiratory infection, skin infection, microbial-caused blindness, tetanus, measles, systemic infection, diarrhea and vomiting, gastrointestinal infections, malaria, fever and headache, general lethargy, and miscellaneous ''illness''), organic and pathological conditions (heart problems, liver problems, body swellings, cancer, hemorrhoids, ''swallowed tongue''), nutritional deficiencies (skinny, ''ate dirt''), and mental illness. Degenerative/congenital problems included biologically based causes not due to pathogen exposure and deaths related to childbirth or old age. This category consisted of newborn death due to birth trauma, prematurity and early failure to thrive, death in childbirth, death due to mother's inability to produce milk, and death from old age. Deaths from old age are ultimately caused by some undetected pathology (e.g., cancer, stroke, heart attack), but we have no further information about these deaths. Accidents included outcomes associated with environmental hazards (drowning, falling, burns, animal-caused trauma, insect-caused trauma, choking, lost) and human-caused accidental deaths (self-stabbing, hunting accident, suffocated, poisoned, killed when playing or sleeping, accidents while intoxicated). Violent deaths consisted of intentional Hiwi-caused mortality (suicide, infanticide, child homicide, adult homicide, warfare) and ''criollo''-caused deaths (murders and massacres).

Ages were assigned through the use of a relative-age list of the entire population constructed from the opinions of a few dozen informants who ranked themselves and others as of similar age. Year of birth is known for all children born after 1983. Year of birth before 1983 was derived from a calendar of dated events (46 dated events between 1957 and 1992) that could be matched with each birth or by using the age-chain method. All individuals who were born before 1957 were assigned year of birth based on their place in the relative-age list, the age-chain year of birth, or the year of birth determined for individuals born at about the same time (see Hill and Hurtado, 1996). The age-chain was constructed using interviews with informants who reported having been the same developmental size as a 12-year-old when an individual of known year of birth was born. Such reports allowed us to assign probable year of birth to the older individual in the pair, and then we repeated the process back through time until all living individuals were assigned a year of birth. Small adjustments (a year or two in either direction for older individuals) were made in the age-chain when there were discrepancies between it and the relative-age list we had constructed. This method produced age estimates with likely error of less than 5 years for adults, as in our prior Ache study (for verification of birthdates obtained by this method, see Hill and Hurtado, 1996). Ages at death (and thus year of death) in the precontact period were assigned by matching the person's appearance at time of death to an individual in the living population of known age.

In this paper, mortality analyses are reported for four age periods of the life history: infancy (0 years), childhood (1–9 years), early adult (10–39 years), and late adult (40–69 years). Infant mortality rates reflect death due to lethal genetic abnormalities expressed soon after exposure to the extrauterine environment, and failure to thrive in the rigorous physical and pathogenic environment of Hiwi bush camps. Childhood mortality is mainly composed of decreasing size-specific vulnerability to environment and pathogens along with an accident rate that is high due to inexperience. Early adult mortality is mainly caused by the constant risk of accidents, violence, and exposure to environmental hazards, with disease somewhat less important. Mortality of older adults results from an increasing senescence in physiological function and disease resistance.

Rates of death were estimated by constructing synthetic life tables that include left censoring of individuals who entered the risk set after birth (due to interview quality) or who moved from the precontact to the postcontact period. Individuals were right censored if they were still alive at the end of the observation period or when they passed through the precontact period without dying. The number of individuals at risk of death in each age category was determined by the completeness of genealogical interviews with different categories of relatives (see below). The synthetic life tables obtained can be used to create age-specific mortality profiles based on different samples for each age category (for details, see Hill and Hurtado, 1996; Hill et al., 2001). The number of individuals who die at age x divided by those at risk in that age interval defines the yearly rate of mortality, q_x. The age-specific mortality rates estimated from the data can then be used to create a synthetic survival curve since $1 - q_x = s_x$ (age-specific survival rate) and the product of all s_x values from age zero to age $x - 1$ defines l_x, the probability of survival to age x. The synthetic life tables can be used for nonlinear parametric mortality analyses such as the Siler competing hazards model (Siler, 1979; Gage and Dyke, 1986).

Most of the individuals in our mortality-risk set were originally ascertained through a reproductive-history interview in

which the entire reproductive history from first to last child born was elicited from an adult or a close kin member. In order to estimate infant and early childhood death rates (age 0–4), we used only the highest-quality interviews in which it was unlikely that an informant would fail to remember an infant that had died at a young age. These included interviews of ego (man or woman) about her/his own reproduction, as well as interviews concerning a sister's reproduction (or ego's mother if ego had been born early in her/his mother's reproductive career). The mortality-risk set for children from age 5 to age 14 included the individuals mentioned above, as well as the children reported in reproductive histories of ego's brothers, ego's father, and ego's mother if ego was born late in her/his mother's reproductive career. This was based on the assumption that informants remembered and mentioned all children born to their parents or any sibling, except possibly a small number of infants who died soon after birth. For older ages, the risk set was expanded further. Individuals in the risk set after age 15 included all the above-mentioned relatives plus any additional children mentioned in reproductive histories of more distant kin (e.g., grandmothers, aunts, uncles, and cousins). We assumed that informants knew and mentioned all aunts, uncles, first cousins, nieces, and nephews who survived to adulthood, even though they may not have provided a perfect accounting of those who died in childhood. Another cohort entered into our expanding risk set at age 25. This included men and women who never were mentioned as part of a sibling set in the reproductive-history interviews, but who are identified as the first spouse of a person whose reproductive history was taken. These individuals were considered to be at risk of death from age 25 onward. Finally, a few additional individuals entered the risk set in 1985 when we first censused all of the Venezuelan Hiwi from three communities. Because we collected reproductive histories from only one of these communities, all individuals living in the other two settlements in 1985 were considered to be at risk of mortality only from 1985 until our last census in 1992. Our sampling procedure resulted in 342 infants at risk of death at age zero, with 215 individuals in the risk set by age 15, but only 17 individuals at risk of death by age 70. For this reason we truncated the life table at age 70 for statistical analyses (the oldest individuals that we observed were a male aged 74 and two females aged 81 and 82).

Mortality analyses were carried out separately for the precontact (prior to 1960) and postcontact periods because some medical treatment was available after peaceful contact, infectious disease exposure was higher, and violence rates were partially curbed by government interference. Because data from any single individual may span both of these time periods, we used all the person years at risk of death prior to 1960 for analyses of precontact rates, and the remaining years at risk were analyzed to obtain postcontact mortality rates. Because of this, and because the shapes of the age-specific mortality rates for different age-sex and period groupings of Hiwi, as well as those for the four comparison groups of hunter-gatherers, were not always well-captured by the Siler model, we chose to do statistical comparisons using logistic regression. Relative differences in the mortality rate are expressed as the impact of relevant variables (age, sex, contact period, hunter-gatherer group) on the odds ratio of deaths/nondeaths for each age category.

Results

Age-specific mortality rates

The Hiwi age-specific mortality rates and probabilities of survival are shown in Tables 1 and 2. The general pattern is one of high mortality, especially in the precontact period. Indeed, when we combine both sexes, the life expectancy at birth in the precontact period is only 27 years, and life expectancy at adulthood (age 15) is only an additional 31 years. When disaggregated, the data clearly show both sex differences in mortality and differences in the pre- and postcontact periods (Fig. 1a,b). We fit a separate model of mortality to each of the four developmental-age groups and examined differences by sex (female = 0, male = 1) and by time period (precontact = 0, postcontact = 1) using logistic multiple regression with an age term to control within-category age effects. The impact of each variable with the others controlled (e.g., multiple logistic regression) is shown in Table 3. Age-dependent decline in mortality is significant in childhood, and the mortality increase with age is substantial in the late-adult period. However, in the early-adult period there is no relationship whatsoever of mortality rate with age. Mortality rates after peaceful outside contact were higher for infants but lower for all adults. Males showed lower mortality than females in the infant age group, and male mortality was much higher than female mortality among older adults.

Summarizing then, we conclude that mortality rates after childhood decreased substantially in the postcontact period. The main sex difference was increasingly higher mortality in men relative to women as age increased (Fig. 1). Adult-male survival in the precontact period was particularly low, with an adult life expectancy of only 30 additional years at age 15. The infant age group showed two reversals of the general trends. First, infant mortality was higher in the postcontact period than in the precontact period, and second, female mortality was higher than male mortality in this age group. The latter trend is mainly due to female-biased infanticide (see below).

Causes of mortality

Percentages of all deaths from each cause and for each age, sex, and period category are shown in Table 4. However, differences in major causes of mortality across age, sex, and contact period are best compared by calculating the deaths per year at risk from each cause. This avoids invalid comparisons based on percentage of deaths, in which some causes may increase dramatically in relative importance simply because other causes of death cease to be observed. Therefore, we divided total number of deaths from each specific cause by the number at risk of death for each age group, sex, and time period and calculated the cause-specific death rates expressed

Table 1
Hiwi age-specific mortality data*

Age	Precontact (before 1960)				Postcontact			
	Male		Female		Male		Female	
	Enter	Die	Enter	Die	Enter	Die	Enter	Die
0	78	11	74	19	109	27	81	27
1	67	5	55	3	83	5	55	4
2	61	4	48	3	79	5	56	8
3	57	0	39	0	75	5	51	3
4	54	4	38	1	73	1	49	1
5	82	1	65	0	75	1	49	0
6	77	1	64	1	74	0	54	0
7	75	0	62	0	75	0	50	0
8	70	3	59	0	77	0	55	0
9	65	1	58	1	82	1	52	1
10	61	3	52	1	84	0	55	2
11	58	0	47	3	81	1	57	0
12	57	1	42	0	77	1	57	0
13	53	1	42	1	79	2	53	0
14	51	1	40	1	73	1	53	1
15	55	1	41	1	71	0	48	0
16	54	1	40	0	62	2	47	1
17	52	0	37	0	55	0	45	5
18	50	2	35	1	56	0	42	0
19	46	0	31	0	61	2	42	1
20	43	1	31	0	59	0	39	0
21	40	0	31	2	54	1	38	0
22	39	0	29	1	56	0	38	0
23	39	1	27	0	53	0	40	0
24	36	1	26	1	52	0	40	0
25	67	2	53	0	55	0	40	0
26	65	0	53	2	53	2	40	1
27	65	2	51	1	50	1	39	1
28	61	1	47	2	51	1	40	0
29	59	3	43	2	46	0	42	0
30	55	2	40	1	48	0	42	0
31	53	0	38	1	49	2	42	0
32	53	1	34	0	42	0	45	0
33	50	3	33	0	43	0	46	1
34	47	0	31	0	41	1	45	0
35	46	0	29	0	40	0	45	1
36	45	1	28	0	39	0	44	0
37	42	0	27	1	39	0	46	0
38	41	2	26	2	39	0	48	1
39	39	1	24	2	37	1	46	0
40	36	0	22	1	36	1	48	2
41	35	1	21	0	34	0	43	0
42	34	0	20	0	32	0	37	1
43	32	1	18	0	34	1	37	0
44	29	1	18	0	35	0	35	0
45	25	0	18	1	37	1	37	0
46	24	1	17	0	36	1	36	0
47	23	1	17	0	34	0	30	0
48	22	2	16	1	34	1	31	0
49	20	1	15	0	30	1	28	0
50	19	1	12	0	33	1	32	1
51	18	2	12	0	32	0	28	0
52	16	0	12	0	27	1	24	0
53	16	0	12	0	24	0	24	0
54	15	0	12	0	25	2	24	1
55	15	2	12	0	25	0	23	1
56	11	2	12	0	26	2	21	0
57	9	0	12	0	18	0	18	0
58	9	0	12	0	17	1	19	0
59	8	0	12	0	18	1	19	1
60	8	1	7	0	18	2	26	2
61	7	0	7	1	15	2	24	0
62	7	0	6	1	10	0	23	0
63	7	0	5	0	10	1	23	0
64	7	0	5	0	9	1	21	1
65	6	0	4	0	9	0	20	0
66	6	1	4	0	8	1	19	0
67	5	1	4	0	6	0	13	0
68	4	2	4	1	5	0	11	0
69	2	1	3	0	4	0	10	1
70	1	0	3	3	4	0	9	0
71	1	0			4	0	9	1
72	1	0			3	1	7	1
73	1	0			2	0	6	1
74	1	0			2	1	4	1
75	1	0			1	0	3	0
76	1	0			1	0	3	0
77	1	0			1	0	3	1
78	1	1			1	1	2	0
79							2	0

* Horizontal lines mark the age of entry into the risk set depending on the quality of reproductive-interview data.

as deaths per 1000 person years at risk for the relevant subgroups (Table 5).

Infancy. We recorded the cause for 92 deaths of infants (birth to age 1). The most frequent category of infant death was that comprising "congenital problems," which accounts for just over 30% of all infant deaths and took place at a rate of about 82 deaths per 1000 births (both sexes and time periods). "Congenital" deaths usually happened in the first few days of life and included being born small and weak, deaths due to birth trauma, and deaths because the infant's mother had no milk. The next most frequent causes of death to infants were infanticide/homicide and disease, each of which accounted for almost 30% of all deaths and took place at a rate of 79 deaths per 1000 infant years at risk. Sex differences were notable, with the infanticide rate four times higher for female infants than for male infants (123 vs. 27 per 1000, respectively, $p = 0.003$). Most of the disease deaths in infancy were due to infectious pathogens, with 55% of all the disease deaths caused by gastrointestinal problems. Respiratory ailments were the next most common among disease deaths in infants. Finally, accidents accounted for about 11% of all infant deaths. Thus, the total percentage of deaths from trauma (violence and accidents) was 40%.

Childhood. We recorded 66 deaths to children from the ages of 1 to 9 years. Disease accounted for about two-thirds of the deaths and took place at a rate of 19 deaths per 1000 child years at risk. Accidents and homicide each accounted for about 14% of child deaths and combined to give a death rate from trauma of about 8 deaths per 1000 child years at risk. There were no significant differences between time periods or by sex in the deaths of children.

Table 2
Hiwi age-specific mortality rates

Age	Precontact (before 1960)				Postcontact			
	Male		Female		Male		Female	
	q_x	l_x	q_x	l_x	q_x	l_x	q_x	l_x
0	0.14	1.00	0.26	1.00	0.25	1.00	0.33	1.00
1	0.07	0.86	0.05	0.74	0.06	0.75	0.07	0.67
2	0.07	0.79	0.06	0.70	0.06	0.71	0.14	0.62
3	0.00	0.74	0.00	0.66	0.07	0.66	0.06	0.53
4	0.07	0.74	0.03	0.66	0.01	0.62	0.02	0.50
5	0.01	0.69	0.00	0.64	0.01	0.61	0.00	0.49
6	0.01	0.68	0.02	0.64	0.00	0.60	0.00	0.49
7	0.00	0.67	0.00	0.63	0.00	0.60	0.00	0.49
8	0.04	0.67	0.00	0.63	0.00	0.60	0.00	0.49
9	0.02	0.64	0.02	0.63	0.01	0.60	0.02	0.49
10	0.05	0.63	0.02	0.62	0.00	0.59	0.04	0.48
11	0.00	0.60	0.06	0.61	0.01	0.59	0.00	0.46
12	0.02	0.60	0.00	0.57	0.01	0.59	0.00	0.46
13	0.02	0.59	0.02	0.57	0.03	0.58	0.00	0.46
14	0.02	0.58	0.03	0.56	0.01	0.56	0.02	0.46
15	0.02	0.57	0.02	0.54	0.00	0.56	0.00	0.45
16	0.02	0.56	0.00	0.53	0.03	0.56	0.02	0.45
17	0.00	0.55	0.00	0.53	0.00	0.54	0.11	0.44
18	0.04	0.55	0.03	0.53	0.00	0.54	0.00	0.39
19	0.00	0.53	0.00	0.51	0.03	0.54	0.02	0.39
20	0.02	0.53	0.00	0.51	0.00	0.52	0.00	0.38
21	0.00	0.51	0.06	0.51	0.02	0.52	0.00	0.38
22	0.00	0.51	0.03	0.48	0.00	0.51	0.00	0.38
23	0.03	0.51	0.00	0.46	0.00	0.51	0.00	0.38
24	0.03	0.50	0.04	0.46	0.00	0.51	0.00	0.38
25	0.03	0.49	0.00	0.45	0.00	0.51	0.00	0.38
26	0.00	0.47	0.04	0.45	0.04	0.51	0.03	0.38
27	0.03	0.47	0.02	0.43	0.02	0.49	0.03	0.38
28	0.02	0.46	0.04	0.42	0.02	0.48	0.00	0.37
29	0.05	0.45	0.05	0.40	0.00	0.47	0.00	0.37
30	0.04	0.43	0.03	0.38	0.00	0.47	0.00	0.37
31	0.00	0.41	0.03	0.37	0.04	0.47	0.00	0.37
32	0.02	0.41	0.00	0.36	0.00	0.45	0.00	0.37
33	0.06	0.40	0.00	0.36	0.00	0.45	0.02	0.37
34	0.00	0.38	0.00	0.36	0.02	0.45	0.00	0.36
35	0.00	0.38	0.00	0.36	0.00	0.44	0.02	0.36
36	0.02	0.38	0.00	0.36	0.00	0.44	0.00	0.35
37	0.00	0.37	0.04	0.36	0.00	0.44	0.00	0.35
38	0.05	0.37	0.08	0.35	0.00	0.44	0.02	0.35
39	0.03	0.35	0.08	0.32	0.03	0.44	0.00	0.34
40	0.00	0.34	0.05	0.30	0.03	0.43	0.04	0.34
41	0.03	0.34	0.00	0.28	0.00	0.42	0.00	0.33
42	0.00	0.33	0.00	0.28	0.00	0.42	0.03	0.33
43	0.03	0.33	0.00	0.28	0.03	0.42	0.00	0.32
44	0.03	0.32	0.00	0.28	0.00	0.41	0.00	0.32
45	0.00	0.31	0.06	0.28	0.03	0.41	0.00	0.32
46	0.04	0.31	0.00	0.27	0.03	0.40	0.00	0.32
47	0.04	0.30	0.00	0.27	0.00	0.38	0.00	0.32
48	0.09	0.29	0.06	0.27	0.03	0.38	0.00	0.32
49	0.05	0.26	0.00	0.25	0.03	0.37	0.00	0.32
50	0.05	0.25	0.00	0.25	0.03	0.36	0.03	0.32
51	0.11	0.23	0.00	0.25	0.00	0.35	0.00	0.31
52	0.00	0.21	0.00	0.25	0.04	0.35	0.00	0.31
53	0.00	0.21	0.00	0.25	0.00	0.34	0.00	0.31
54	0.00	0.21	0.00	0.25	0.08	0.34	0.04	0.31
55	0.13	0.21	0.00	0.25	0.00	0.31	0.04	0.30
56	0.18	0.18	0.00	0.25	0.08	0.31	0.00	0.28
57	0.00	0.15	0.00	0.25	0.00	0.29	0.00	0.28
58	0.00	0.15	0.00	0.25	0.06	0.29	0.00	0.28
59	0.00	0.15	0.00	0.25	0.06	0.27	0.05	0.28
60	0.13	0.15	0.00	0.25	0.11	0.25	0.08	0.27
61	0.00	0.13	0.14	0.25	0.13	0.23	0.00	0.25
62	0.00	0.13	0.17	0.22	0.00	0.20	0.00	0.25
63	0.00	0.13	0.00	0.18	0.10	0.20	0.00	0.25
64	0.00	0.13	0.00	0.18	0.11	0.18	0.05	0.25
65	0.00	0.13	0.00	0.18	0.00	0.16	0.00	0.24
66	0.17	0.13	0.00	0.18	0.13	0.16	0.00	0.24
67	0.20	0.11	0.00	0.18	0.00	0.14	0.00	0.24
68	0.50	0.09	0.25	0.18	0.00	0.14	0.00	0.24
69	0.50	0.04	0.00	0.13	0.00	0.14	0.10	0.24
70	0.00	0.02	1.00	0.13	0.00	0.14	0.00	0.21
71	0.00	0.02		0.00	0.00	0.14	0.11	0.21
72	0.00	0.02			0.33	0.14	0.14	0.19
73	0.00	0.02			0.00	0.09	0.17	0.16
74	0.00	0.02			0.50	0.09	0.25	0.13
75	0.00	0.02			0.00	0.05	0.00	0.10
76	0.00	0.02			0.00	0.05	0.00	0.10
77	0.00	0.02			0.00	0.05	0.33	0.10
78	1.00	0.02			1.00	0.05	0.00	0.07
79		0.00				0.00		0.07

Early adulthood. We recorded the causes for 88 deaths between the ages of 10 and 39 years. Warfare and homicide accounted for 44% of the deaths in this age category, with about 42% of these caused by intratribal disputes and 58% due to attacks by ''criollos.'' Surprisingly, women were just as likely to be killed as men, and deaths from violence were almost as frequent in the postcontact period as in the precontact period (about 8 per 1000 risk years in the precontact period and 6 per 1000 risk years in the postcontact period). Interestingly, we also recorded two precontact and one postcontact death due to suicide (all males). Thirty-five percent of all early-adult deaths were from disease, with infectious pathogens causing the vast majority of these deaths, and other organic and pathological conditions accounting for less than 5% of this category. Again, gastrointestinal pathogens and parasites were the most common cause of disease-related deaths, with respiratory ailments somewhat less common. However, the disease-related death rate of young adults was much lower in the postcontact period than during the precontact period (2 vs. 10 per 1000 years at risk, respectively, $p = 0.001$). Finally, accidents accounted for about 11% of all deaths to young adults, with no obvious differences due to sex or period. Adding accidents and violence, we found that trauma accounted for more than half of all early-adult deaths and took place at a rate of about 9 deaths per 1000 years of risk.

Late adulthood. We recorded causes of death for 67 individuals aged 40 through 79 years. Among late adults, disease was the most important cause of death (63% of all deaths), and among these infectious-disease deaths, gastrointestinal maladies predominated. In this age category, uncategorized deaths due to ''old age'' accounted for only 6% of reported mortality. Violence remained an important cause of death, with 25% of all deaths in this age group being due to warfare/homicide. Again, we discovered one male suicide in the precontact

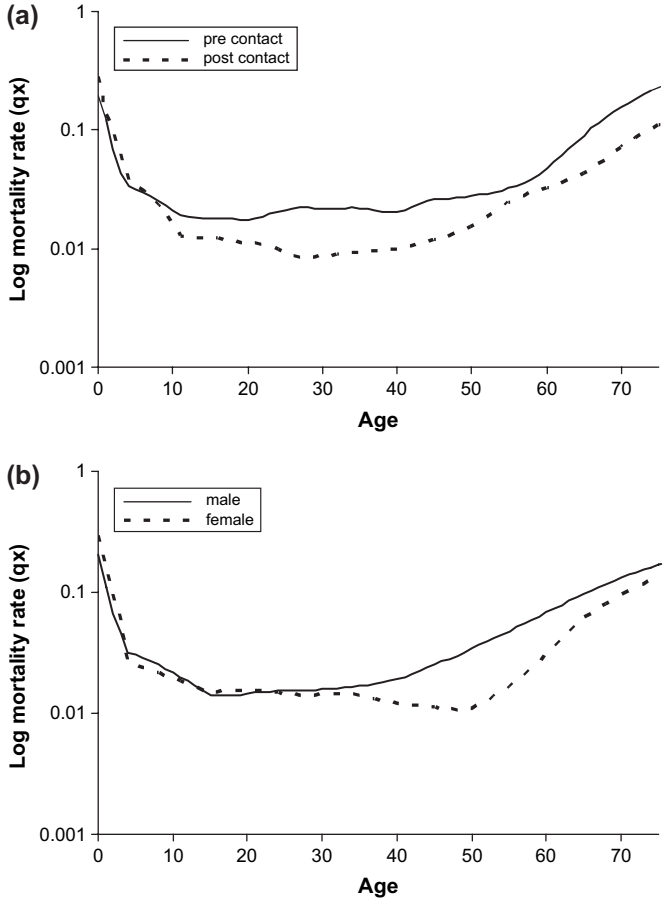

Fig. 1. Hiwi age-specific mortality-rate comparison by time period (a) and by sex (b). Rates smoothed by Lowess regression after age zero.

period. Finally, 6% of the deaths among late adults were due to accident, making the combined death rate due to trauma about 9 per 1000 years at risk of death. Men showed marginally higher death rates due to disease (23 vs. 13 per 1000 years at risk, $p = 0.08$) and substantially higher death rates due to violence (12 vs. 3 per 1000 years at risk, $p = 0.02$) than women.

Age pattern. The absolute rates of death from each of the four aggregated categories are U-shaped with age, mirroring the shape of overall mortality rates across the life span. However, disease, organic pathologies, and senescence were relatively more important in the very young and the old, whereas violence and accidents were relatively more important for older children and young adults (Fig. 2). The death rate due to trauma (accidents and violence) is remarkably stable at 8–9 deaths per 1000 risk years from childhood through late adulthood.

Violent and accidental death. The Hiwi mortality profile is characterized by notably high rates of violence and accidental trauma. The data show that 12% of all girls born were killed in the first year of life (and about 3% of all boys), and about 36% of all adult deaths were from warfare/homicide. Accident death rates were quite high for infants and young children (about 3% of children born die from accidents in the first year), declining to about 2 per 1000 per year by adulthood. Accidents accounted for about 10% of all deaths in all four of the age categories analyzed. We can also estimate that risk of death due to childbirth/pregnancy was about 4.4 per 1000 reproductive-aged women per year. Observed pregnancy was achieved only about once every three to four years (unpublished data), implying a death-in-pregnancy rate of one death per every 55–75 pregnancies.

Comparison to other hunter-gatherers

Detailed demographic studies based on interview data have only been published for three groups of hunter-gatherers, the !Kung (Ju/'hoansi) of Botswana-Namibia (Howell, 1979), the Ache of Paraguay (Hill and Hurtado, 1996), and the Agta of the Philippines (Early and Headland, 1998). The mortality and fertility profiles of the Hadza have also been estimated using more indirect census methods (Blurton Jones et al., 1992, 2002). Here, we compare mortality among the precontact Hiwi, precontact Ache, precontact Agta, the !Kung born before 1950 (minimal acculturation), and the Hadza from 1985–1995.

Cause of death among the groups differs considerably. Disease is an important cause of death in all groups, but represents only ~20% of deaths in the precontact Ache, ~45% among the precontact Hiwi, and about 75–85% of all Hadza, !Kung, and Agta deaths. Respiratory disease is the main killer of the Ache, whereas gastrointestinal pathogens are most important among the Hiwi and probably Hadza. Among the !Kung, respiratory and gut infections are about equally important. Violence is the major cause of death among the precontact Ache (~55% of all deaths) and very important among the Hiwi (~30% of all deaths), but notably less important in the two African societies and the Agta (3–7% of all deaths). Indeed, the crude homicide/warfare death rates per year lived are more than ten times higher among the Hiwi and Ache than among the Hadza or !Kung (~1/100 and 1/200 per year for precontact Hiwi and Ache, respectively, vs. 1/2500 and 1/3000 for the Hadza and !Kung, respectively). Blurton Jones et al. (2002) suggested that this may be due to the more pervasive effects of colonial governments in Africa and the reduction of intertribal warfare. Even so, within-group homicide and infanticide rates are also much lower among African foragers, suggesting real cultural differences in violence rates.

The most notable contrast among hunter-gatherer life tables is the overall similarity of child mortality followed by

Table 3
Logistic-regression mortality-rate model for Hiwi hunter-gatherers by age, sex, and time period

Variable	Odds ratio			
	Infant	Child	Early adult	Late adult
Age	—	0.70***	1.00	1.07***
Sex (male = 1)	0.58*	1.03	0.91	2.68***
Period (postcontact = 1)	1.68*	1.10	0.50**	0.66

*$p < 0.05$.
**$p < 0.01$.
***$p < 0.001$.

Table 4
Percentage of deaths for age-sex groups aggregated by causal category

Category	Aggregated cause	Infant (0 yrs)		Middle childhood (1–9 yrs)		Early adult (10–39 yrs)		Late adult (40–79 yrs)	
		Male	Female	Male	Female	Male	Female	Male	Female
Precontact (before 1960)									
Disease	Infectious disease	29	25	59	73	55	29	43	88
	Other organic/pathological							4	
	Mental illness								
	Nutritional								
Congenital	Senescence							4	
	Congenital infant death	29	25	9	9				
	Childbirth/abortion						21		
Accident	Environmental hazard		5	14			8	4	
	Human-caused accidental	7	5	5	9	3	4	4	
Violence	Suicide					3		9	
	Infanticide/child homicide	14	40	5	9				
	Hiwi killed	7				16	13	9	
	Venezuelan killed	14		9		19	25	22	13
Total deaths		14	20	22	11	31	24	23	8
Postcontact									
Disease	Infectious disease	35	15	35	69	16	14	52	54
	Other organic/pathological			6	6	5		13	8
	Mental illness							4	
	Nutritional	3	7	12	13				
Congenital	Senescence		4					4	15
	Congenital infant death	42	15	6					
	Childbirth/abortion		4				21		
Accident	Environmental hazard	10		12		16			8
	Human-caused accidental	3	11	6	6	11	7	4	
Violence	Suicide							4	
	Infanticide/child homicide	6	41	18	6				
	Hiwi killed					21	14		
	Venezuelan killed		4	6		32	43	17	15
Total deaths		31	27	17	16	19	14	23	13

subsequent high mortality of the Hiwi and Agta in adulthood compared to the Ache, !Kung, and Hadza (Fig. 3). The number of individuals at risk in each yearly category and the number of deaths observed have been only published for the Ache and Hadza. Thus, statistical analyses of differences in mortality rates between these groups and the Hiwi can be performed using logistic regression. The results suggest that all foragers are not characterized by a single ''typical'' mortality schedule. Analyses of the differences for infants, children, adults, and elderly using logistic regression (Table 6) shows significantly lower Ache infant mortality and early-adult mortality relative to the Hiwi, and lower Hadza adult mortality (both young and old) relative to the Hiwi. Particularly striking is the fact that Hiwi early-adult mortality rates are about double those of the Ache and Hadza.

Although raw-data counts are not available for two of the five hunter-gatherer groups, smoothed mortality curves can be constructed for all five groups based on published life tables for each (Fig. 3). The curves show that the Agta experience exceptionally high mortality in infancy and adulthood, with an age-specific mortality shape that is not typical of any hunter-gatherers or other human groups (possibly due to some errors in age assignment?). The Hiwi show typical mortality up to about 10 years; subsequently, mortality is consistently higher than it is in other hunter-gatherer groups. The Ache experience relatively low infant mortality, and the Hadza show markedly lower mortality in the late-adult years than do other hunter-gatherer groups.

A similar comparison of mortality in traditional societies (including our five hunter-gatherer groups), using Siler hazard modeling, became available while we were completing this manuscript. In that study, Gurven and Kaplan (in press) obtained results consistent with those reported here, and they suggested that infant mortality accounts for most of the variation in life expectancy at birth across remote traditional small-scale societies. They also showed a variety of species-typical patterns in the hunter-gatherer mortality profile and demonstrated that there is a huge discrepancy between paleodemographic life tables and those of all known hunter-gatherers, suggesting the presence of still unappreciated problems with reconstructions of prehistoric mortality.

Discussion

We present here, for the first time, a complete analysis of Hiwi mortality that supercedes all previous demographic estimates for this population (e.g., Kaplan et al., 2000). Hiwi adult death rates are quite high compared to the most commonly cited hunter-gatherer groups (Ache, Hadza, and !Kung). The contrast is most noticeable for the precontact period, where

Table 5
Cause-specific rates of death (per 1000 risk years)

Category	Infant (0 yrs)		Middle childhood (1–9 yrs)		Early adult (10–39 yrs)		Late adult (40–79 yrs)	
	Male	Female	Male	Female	Male	Female	Male	Female
Precontact (before 1960)								
Disease	51	68	21	16	12	6	23	20
Congenital	51	68	3	2	0	5	2	0
Accident	13	27	7	2	1	3	4	0
Violence	64	108	5	2	8	8	19	3
Total at risk	78	74	608	488	1522	1108	484	354
Postcontact								
Disease	110	74	13	30	2	1	23	10
Congenital	119	74	1	0	0	2	1	2
Accident	37	37	4	2	3	1	1	1
Violence	18	148	6	2	6	6	7	2
Total at risk	109	81	693	471	1645	1344	700	832

Hiwi adult mortality is about 2.3% per year, compared to only ~1.1–1.3% per year for the precontact Ache, Hadza, and !Kung. The cumulative effect of this difference during adulthood is quite striking. Only 51% of all Hiwi 10-year-olds in the precontact period were expected to survive to an age at which they might become grandparents (age 40, double the mean age of first birth). However, 72–76% of all precontact Ache, Hadza, and Kung 10-year-olds survived to grandparental age. Life-history theory predicts that high early-adult mortality should be associated with faster developmental rates of juveniles and earlier senescence. In fact, the Hiwi do show faster childhood growth rates and earlier age at sexual maturity than other foraging groups (Walker et al., 2006), and their mortality rates among older adults are higher at all ages (Fig. 3).

In examining the causes of Hiwi death, we note an important difference from the hunter-gatherer groups most commonly cited in the literature. Despite the postcontact massacres organized by local ranchers, the precontact period was characterized by even higher rates of violence, with 54% of the killings caused by the Hiwi themselves. In total, 36% of all precontact adult deaths were due to warfare and homicide. The Hiwi crude death rate due to warfare/homicide is 1,018 per 100,000 person years lived, six times higher than the hunter-gatherer median reported by Wrangham et al. (2006). The crude death rate from accidental trauma is 297 per 100,000. Indeed, among young adults, the combined death rate from violence and accident in the precontact Hiwi is about 1.1% per year, which is equal to the mean death rate for the Ache, Hadza, and !Kung *from all causes combined*. We suggest that reported modern African hunter-gatherer rates of violence may be low because of interference by powerful state-level societies prior to demographic study (cf. Blurton Jones et al., 2002), and there is considerable evidence that groups like the Bushmen engaged in much higher levels of violence prior to colonial repression (e.g., Schapera, 1930).

Paleoanthropologists have shown particular interest in the mortality patterns of Neandertals, premodern hominins, and Paleolithic humans (Bermúdez de Castro and Nicolás, 1997; Rosas et al., 1999; Bocquet-Appel, 2001; Bermúdez de Castro et al., 2003; Lozano et al., 2004). The suggestion that hominin life spans have been too short to expect grandparenting for some fossil hominins has been vigorously debated (Hawkes et al., 1998; Caspari and Lee, 2004, 2005a,b, 2006; Hawkes and O'Connell, 2005). Particularly contentious, but potentially important, is the suggestion that Paleolithic populations of humans and Neandertals may have experienced much higher mortality than that reported in most modern populations (e.g., Vallois, 1937; Acsádi and Nemeskeri, 1970; Trinkaus, 1995; but see Aykroyd et al., 1999). There is little doubt that a mortality rate that is very much higher than we report for the Hiwi in this paper would have drastic social consequences (Howell, 1982). Given the same age at sexual maturity, such high mortality would render impossible the three-generation cooperative-breeding pattern that is typical of modern humans (Kaplan, 1997; Kaplan et al., 2000).

If high mortality, warfare, homicide, and accidental trauma are typical of our Paleolithic ancestors, the Hiwi mortality patterns may be more representative of the past than those derived from other modern hunter-gatherers. If so, several observations about the Hiwi are important. First, conspecific violence was a prominent part of the demographic profile, accounting for many deaths in all age and sex categories. Most of the adult killings were due to either competition

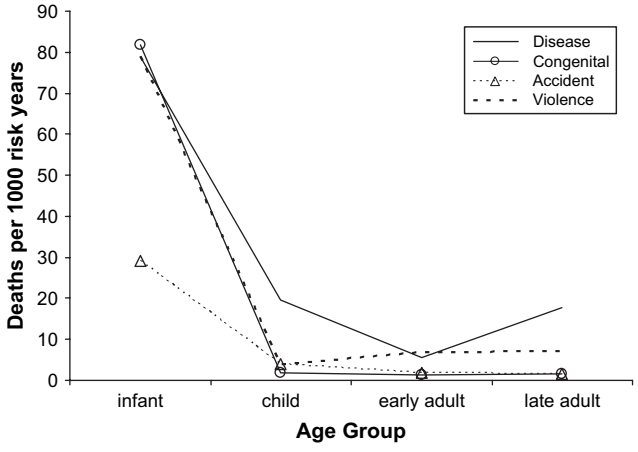

Fig. 2. Cause-specific death rates for Hiwi foragers (sex and period combined).

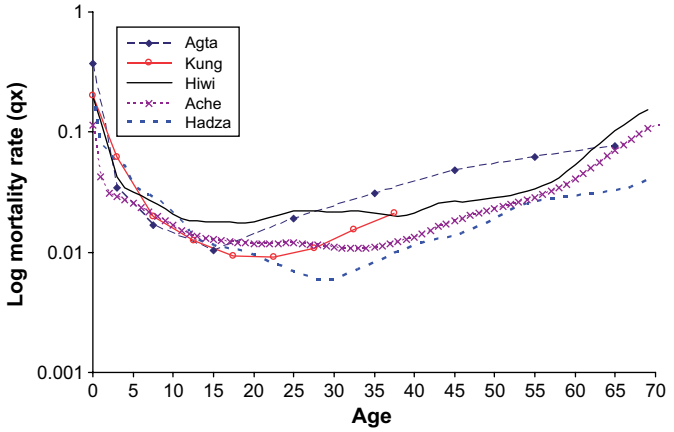

Fig. 3. Comparative age-specific mortality for five groups of hunter-gatherers. Rates smoothed by Lowess regression after age zero.

over women, reprisals by jealous husbands (on both their wives and their wives' lovers), or reprisals for past killings. The criollo-caused killings were motivated by territorial conquest. Moreover, infanticide (especially on females) constituted the highest mortality rate component of all Hiwi conspecific violence. Second, no predation deaths were reported despite attacks by anacondas, Orinoco caimans, and piranhas, and the presence of jaguars in the area. Accidents associated with the active-forager lifestyle were common, but disease was a more important killer, accounting for nearly half of all deaths. This suggests an adaptive landscape in which success in social relations, competitive violence, and disease resistance are paramount. This may partially explain why many of the genes that appear to have been under strong selection in the past 50,000 years affect either disease resistance or cognitive function (Wang et al., 2006), presumably related to success in an atmosphere of frequent violent social competition.

Hiwi mortality data present a very different picture of human adaptation than that derived from mortality statistics in modern society. Not only are mortality rates much higher in the Hiwi, they are disproportionately higher among infants, children, and young adults (cf. Gurven and Kaplan, in press). The relative importance of major causes of death in the Hiwi vs. modern America is also striking. In the U.S., the major killers are heart disease and cancers, both almost nonexistent among hunter-gatherers. In modern America, respiratory diseases are among the top 10 causes of death, but gastrointestinal infections are virtually absent as a cause of death. Suicide is more important than homicide in modern America, and homicide accounts for only 1% of all deaths. Finally, because of automobiles, accidents make up almost an equal portion of deaths in both modern societies and in hunter-gatherers (all U.S. data are from Zopf, 1992).

Table 6
Logistic-regression mortality-hazard model for Hiwi hunter-gatherers by age, sex, and time period

Variable	Odds ratio			
	Infant	Child	Early adult	Late adult
Hiwi	1.00	1.00	1.00	1.00
Ache	0.53**	0.97	0.52***	0.78
Hadza	—	1.43	0.43***	0.57*

*$p < 0.05$.
**$p < 0.01$.
***$p < 0.001$.

Despite their relatively higher mortality, the Hiwi generally resemble other modern hunter-gatherers in many respects. Infant mortality is high, and death rates reach their lowest levels around the age of sexual maturity. Early-adult mortality, while relatively high among the Hiwi, is still only half of that observed among young adult wild chimpanzees (Hill et al., 2001), and mortality rates among all hunter-gatherers are only a fraction of that of chimpanzees from age 5 onward (Gurven and Kaplan, in press: Fig. 8). According to mammalian life-history theory, reduced early-adult mortality should produce a longer growth period, later sexual maturity, and later onset of senescence in humans relative to chimpanzees (Charnov, 1993; Hill, 1993; Hawkes et al., 1998). Current data confirm all of these predicted differences in life history between chimpanzees and modern hunter-gatherers (Kaplan et al., 2000; Hill et al., 2001; Gurven and Kaplan, in press). Indeed, while mortality-rate doubling times are indistinguishable between hunter-gatherers and chimpanzees, early-adult mortality is much higher and onset of senescence much earlier in chimpanzees (Gurven and Kaplan, in press).

Two recent hypotheses for the extended human life span have been widely discussed. The "grandmother hypothesis" (Hawkes et al., 1998) proposes that humans live to old age in order for women to invest in their grandoffspring. The "embodied capital model" (Kaplan et al., 2000; Kaplan and Robson, 2002) suggests that the complex hunter-gatherer feeding niche requires a long learning period to master but provides high benefits later in life, thus favoring an extended life span. We suggest that neither of these is a fully adequate explanation for the initial change in human longevity. Instead, these hypotheses mainly identify adaptations that provide additional fitness benefits *once a significant portion of the population begins to survive to older ages*. Neither accounts for the 2–5-fold lower early-adult mortality in hunter-gatherers relative to chimpanzees, which is likely to "explain" why the mechanisms that delay senescence and extend the human life span were favored by natural selection in the first place.

Most exceptionally long-lived organisms do not engage in grandmaternal investment, nor do they occupy complicated feeding niches requiring long periods of human capital development. Instead, most animals with exceptionally long life spans (e.g., sessile worms around warm ocean vents, queens of social-insect colonies, tortoises, bowhead whales, deep-sea rockfish, echidnas, etc.) all show common features of extremely low vulnerability to predation, accidents, and disease in their adult years because of either a unique habitat or special protective adaptations. Moreover, the longest-living sexually reproducing organisms on the planet are several genera of bushes and trees, living in dry climates or high elevation. Those species of trees clearly did not evolve their long life spans due to grandmothering or required learning of a complex feeding niche. A consideration of contrasting

traits among hunter-gatherers and chimpanzees suggests that food-sharing and care of sick and injured individuals was most likely responsible for the initial improvement in early adult survival. Humans are unique in bringing back food resources to a base camp where sick and injured individuals can recover. No other food-sharing animal can carry resources (very few share food). Humans also carry sick and injured when they move, or remain in an area until they have recovered. Thus, even without any other direct forms of care (e.g., grooming, protecting, providing medicinal interventions), we suggest that sick and injured humans have a large survival advantage through childhood and adulthood. This lowered ''extrinsic'' mortality would then favor later onset of senescence and the evolution of mechanisms that slow rates of senescence through adulthood.

The Hiwi show an exceptional adult life span (relative to apes), as do other hunter-gatherers. Despite the relatively high adult mortality rates among the precontact Hiwi, old age is not rare. Just over 50% of all women who reached reproductive age (15) also survived to menopause (age 45). Moreover, the expected postreproductive life span is considerable, even under the high mortality conditions of the precontact Hiwi (47% of all Hiwi women who reached age 45 survived to age 70). Extensive consideration of factors that could change the hunter-gatherer mortality profile suggests that this pattern is robust and probably representative of all recent hunter-gatherers (Blurton Jones et al., 2002; Gurven and Kaplan, in press). The higher Hiwi early adult mortality rates may be more typical of Upper Paleolithic *Homo sapiens*, but even so, grandmothers and grandfathers were common enough under these conditions to provide significant help to kin and allow for the three-generation cooperative-breeding pattern that appears to be the typical human social arrangement. This life-history pattern is radically different from that of chimpanzees, in which growth is rapid, sexual maturity early, less than 20% of females born survive to twice the mean age at first birth (Hill et al., 2001), and there is no significant postreproductive period. The most interesting questions that now remain are what selective pressures led to this life history and when did it evolve?

Acknowledgements

We thank the Hiwi of Mahünemuthu, especially the families of Marcelo and David Machal, for research assistance and friendship. We also thank Ric Charnov, Mike Gurven, Osborne Pearson, and Robert Walker for useful discussions.

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