

**SI for mortality chapter.**

- 8.1. Alternative methods for scoring qx [scoreqx](#)**
- 8.2. Comparing census and interview data. [censusnintv](#)**
- 8.3. Life table for sexes combined. [lifetaboth](#)**
- 8.4. Resampling the mortality data. [resamprog](#)**
- 8.5. Survival from 1977 to 1985 [surv1977to1985](#)**
- 8.6. Sex differences in mortality and survival of late middle-aged men and women. [oldagesurvival](#)**
- 8.7. “Plenty of old people” versus “No old people” again. Age at death distributions and paleodemography. [paleodemography](#)**
- 8.8. Data table for text figure 8.5 [datfig85](#)**

### SI 8.1. Alternative methods for scoring $q_x$ .

Three other methods can be used, I tried each one and decided that the differences were unimportant. I use the results of only the first in all subsequent calculations and simulations.

I computed  $q_x$  and a life table in the same way as Howell (1979: 81, Table 4.1).  $Q_x$  was computed for each time unit as the number who die in that period divided by the number who lived through (completed) the period. For the latter Howell used the number who started the interval minus the number “currently in the interval” – the final year of observation. In my tables, this “at risk group” is the number who entered the year minus the number who were censored in that year. They are censored because our observations do not allow us to tell whether they survived to the end of that final year in which they were briefly observed. {My files denote this method as risk category “b”.}

The range of  $e_0$  given by these four methods is 32.7 to 34.15 for the genders combined. The range for females is 35.55 to 36.75. This is less than the difference between Coale & Demeny models West 7 and 8. The difference between  $e_0$  by my method and by H&H table 6.1 is 1.8%. The average difference in  $q_x$  between these two methods is 1.43%.

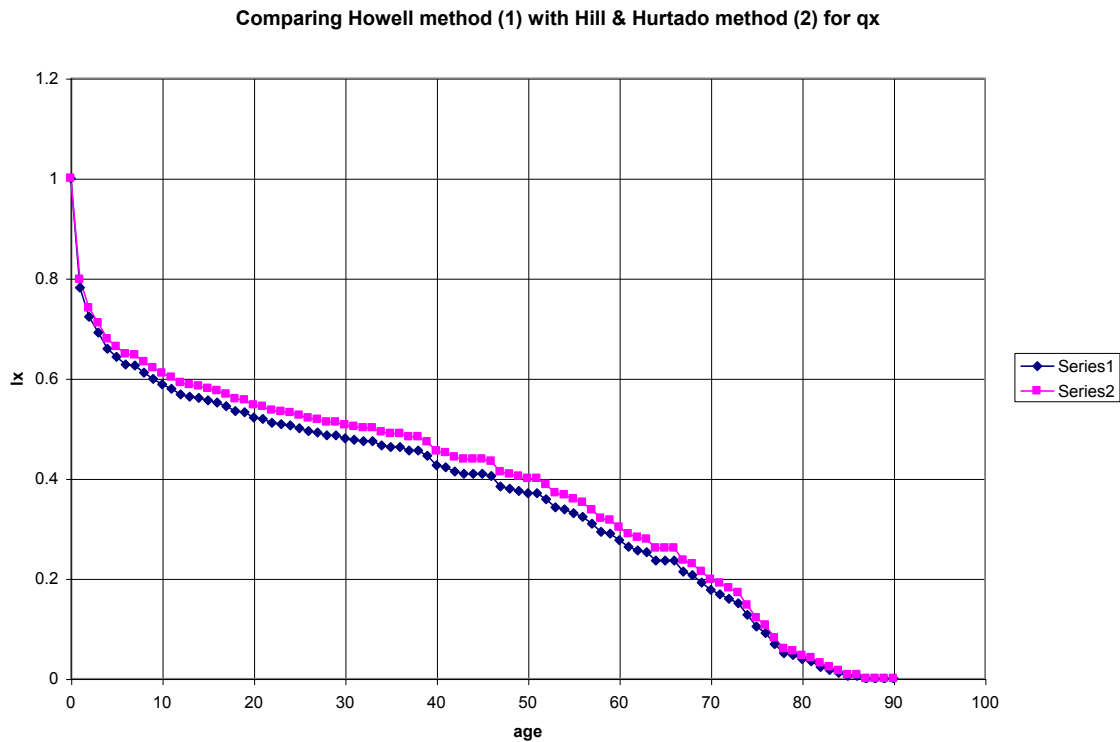
2. Hill & Hurtado (1996:195 Table 6.1) computed  $q_x$  as the number who died in each year of life divided by the number of years lived by those who entered the year. The number of years lived was calculated as the number who lived to the end of the year plus half a year for each of those who died during the year, a convention used by other demographers. They note that few of their people were lost to observation, partly because there was no emigration. In my table the “at risk group” by this method is the number who enter the year minus .5 x the number who died during the year. Hill & Hurtado’s Table 6.1 is the source for my comparisons between Hadza and Ache. {My files denote this method as “HH6.1”. This refers to their Table 6.1 and its caption on p 195. Hill & Hurtado equation 6.1 on page 181 is different, giving a lower  $q_x$  and would give a longer  $e_0$ . They support the use of this equation by reference to their use of logistic regression, in which censoring happens at the end of the final year of observation.}

3. The Kaplan-Meier statistics attribute half a year to the censored years. Thus in my data the number at risk in a year is the number who enter that year, minus half the number who were censored that year. {I noted this as risk category “half” in my files.}

4. But individuals who die in a particular year may have died at any time during the year, they entered the year but did not live right through it (just as argued in HH6.1). If we conceive of  $q_x$  as deaths per person year at risk, and equate this with person-years lived, then a good measure of the risk group would be the number who entered a year minus half the number that died and half the number who were censored. This measure would be equivalent to  $dx$  and to give  $q_x$  it should be adjusted by a formula offered by Barclay (1958). This is labeled half-half in my files.

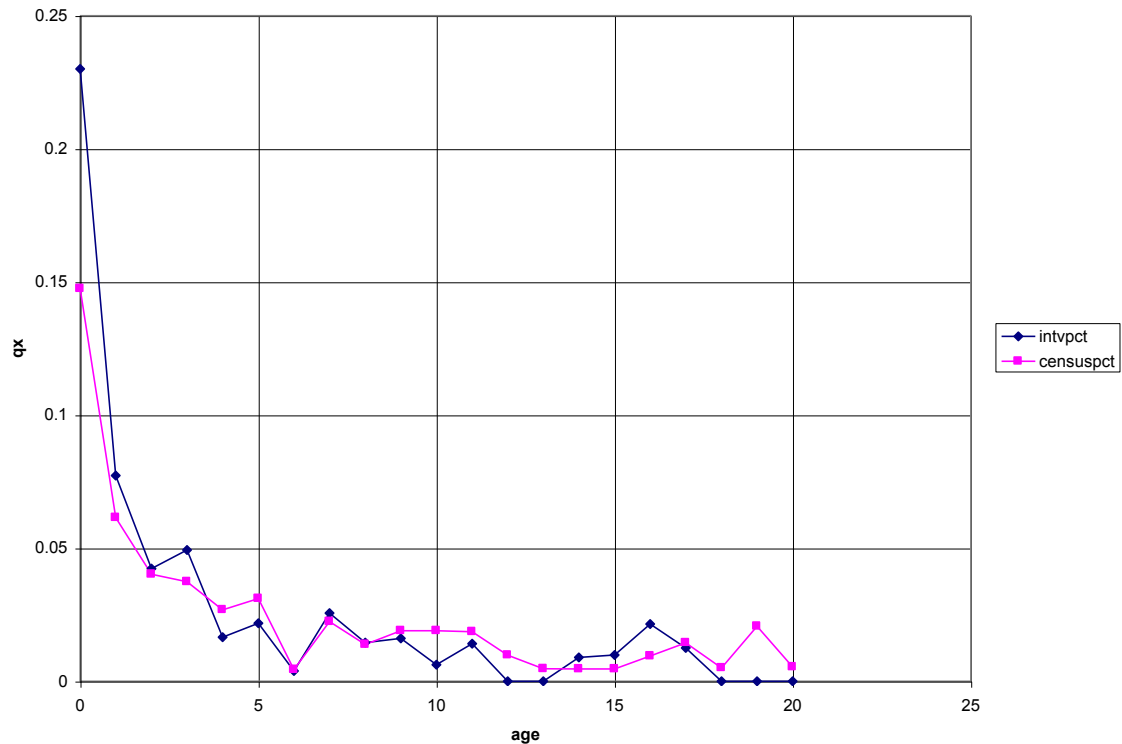
Comparing life expectancy at birth ( $e_0$ ) given by each of the four different ways of calculating  $q_x$ . Differences between rows 2 and row 1: female 2.7%, male 1%, both 1.8%.

	Risk	Name in files	Female $e_0$	Male $e_0$	Both $e_0$
1	Enter - censored (Howell)	b	35.55	30.81	32.7
2	Enter - .5*deads (H&H6.1)	HH6.1	36.5	31.14	33.30
3	Enter - (0.5 * censored)	Half	36.75	31.63	34.15
4	Enter - (0.5 * censored + 0.5 * dead)	Halfhalf	35.85	31.03	33.02



### SI 8.2. Comparing census and interview data.

More information on young children is acquired by interview.



### SI 8.3. Life table for sexes combined.

age	Enter both	Censored both	Dead both	risk	$q_x$	$l_x$	$L_x$	$T_x$	ex
0	697	51	141	646	0.2183	1.0000	0.8472	32.7001	32.7001
1	517	21	37	496	0.0746	0.7817	0.7467	31.8529	40.7464
2	470	34	19	436	0.0436	0.7234	0.7077	31.1061	42.9987
3	425	18	19	407	0.0467	0.6919	0.6757	30.3985	43.9351
4	396	32	9	364	0.0247	0.6596	0.6514	29.7227	45.0621
5	363	27	8	336	0.0238	0.6433	0.6356	29.0713	45.1919
6	338	16	1	322	0.0031	0.6280	0.6270	28.4356	45.2819
7	331	24	7	307	0.0228	0.6260	0.6189	27.8086	44.4214
8	311	22	6	289	0.0208	0.6117	0.6054	27.1898	44.4463
9	295	21	5	274	0.0182	0.5990	0.5936	26.5844	44.3780
10	289	25	4	264	0.0152	0.5881	0.5837	25.9908	44.1935
11	280	22	5	258	0.0194	0.5792	0.5736	25.4071	43.8658
12	266	17	2	249	0.0080	0.5680	0.5657	24.8335	43.7228
13	261	19	1	242	0.0041	0.5634	0.5623	24.2678	43.0728

age	Enter both	Censored both	Dead both	risk	qx	lx	Lx	Tx	ex
14	252	9	2	243	0.0082	0.5611	0.5588	23.7056	42.2494
15	257	16	2	241	0.0083	0.5565	0.5542	23.1468	41.5959
16	246	15	3	231	0.0130	0.5519	0.5483	22.5927	40.9398
17	241	20	4	221	0.0181	0.5447	0.5398	22.0444	40.4719
18	231	20	1	211	0.0047	0.5348	0.5336	21.5046	40.2087
19	221	18	4	203	0.0197	0.5323	0.5270	20.9711	39.3978
20	217	18	1	199	0.0050	0.5218	0.5205	20.4440	39.1797
21	211	11	3	200	0.0150	0.5192	0.5153	19.9235	38.3750
22	211	17	1	194	0.0052	0.5114	0.5101	19.4083	37.9518
23	206	17	1	189	0.0053	0.5088	0.5074	18.8982	37.1458
24	201	18	2	183	0.0109	0.5061	0.5033	18.3908	36.3408
25	201	18	2	183	0.0109	0.5005	0.4978	17.8875	35.7368
26	199	23	1	176	0.0057	0.4951	0.4937	17.3897	35.1261
27	191	22	2	169	0.0118	0.4923	0.4893	16.8960	34.3240
28	179	12	0	167	0.0000	0.4864	0.4864	16.4067	33.7291
29	178	14	2	164	0.0122	0.4864	0.4835	15.9203	32.7291
30	178	11	1	167	0.0060	0.4805	0.4791	15.4368	32.1270
31	175	5	1	170	0.0059	0.4776	0.4762	14.9577	31.3175
32	180	10	0	170	0.0000	0.4748	0.4748	14.4815	30.4999
33	179	16	3	163	0.0184	0.4748	0.4704	14.0067	29.4999
34	167	17	1	150	0.0067	0.4661	0.4645	13.5363	29.0436
35	157	11	0	146	0.0000	0.4630	0.4630	13.0718	28.2352
36	152	16	2	136	0.0147	0.4630	0.4596	12.6088	27.2352
37	140	11	0	129	0.0000	0.4562	0.4562	12.1493	26.6342
38	141	13	3	128	0.0234	0.4562	0.4508	11.6931	25.6342
39	129	11	5	118	0.0424	0.4455	0.4360	11.2423	25.2374
40	125	19	1	106	0.0094	0.4266	0.4246	10.8063	25.3320
41	110	10	2	100	0.0200	0.4226	0.4183	10.3817	24.5685
42	104	14	1	90	0.0111	0.4141	0.4118	9.9634	24.0597
43	96	6	0	90	0.0000	0.4095	0.4095	9.5516	23.3244
44	94	4	0	90	0.0000	0.4095	0.4095	9.1420	22.3244
45	103	10	1	93	0.0108	0.4095	0.4073	8.7325	21.3244
46	102	6	5	96	0.0521	0.4051	0.3946	8.3252	20.5508
47	97	10	1	87	0.0115	0.3840	0.3818	7.9307	20.6524
48	93	7	1	86	0.0116	0.3796	0.3774	7.5489	19.8868
49	87	3	1	84	0.0119	0.3752	0.3729	7.1715	19.1149
50	97	2	0	95	0.0000	0.3707	0.3707	6.7985	18.3391
51	99	6	3	93	0.0323	0.3707	0.3647	6.4278	17.3391
52	95	7	4	88	0.0455	0.3588	0.3506	6.0631	16.9004
53	89	5	1	84	0.0119	0.3424	0.3404	5.7125	16.6814
54	90	3	2	87	0.0230	0.3384	0.3345	5.3721	15.8764
55	103	9	2	94	0.0213	0.3306	0.3271	5.0376	15.2382
56	96	3	4	93	0.0430	0.3236	0.3166	4.7105	14.5586

age	Enter both	Censored both	Dead both	risk	qx	lx	Lx	Tx	ex
57	98	3	5	95	0.0526	0.3096	0.3015	4.3939	14.1904
58	92	5	1	87	0.0115	0.2933	0.2917	4.0924	13.9510
59	88	0	4	88	0.0455	0.2900	0.2834	3.8008	13.1074
60	93	10	4	83	0.0482	0.2768	0.2701	3.5174	12.7077
61	82	8	2	74	0.0270	0.2635	0.2599	3.2473	12.3258
62	75	2	1	73	0.0137	0.2563	0.2546	2.9874	11.6543
63	78	3	5	75	0.0667	0.2528	0.2444	2.7328	10.8093
64	71	7	0	64	0.0000	0.2360	0.2360	2.4884	10.5456
65	74	9	0	65	0.0000	0.2360	0.2360	2.2524	9.5456
66	65	1	6	64	0.0938	0.2360	0.2249	2.0165	8.5456
67	65	3	2	62	0.0323	0.2138	0.2104	1.7916	8.3780
68	60	5	4	55	0.0727	0.2069	0.1994	1.5812	7.6405
69	54	1	4	53	0.0755	0.1919	0.1847	1.3818	7.2006
70	54	13	2	41	0.0488	0.1774	0.1731	1.1971	6.7476
71	40	2	2	38	0.0526	0.1688	0.1643	1.0240	6.0680
72	39	4	2	35	0.0571	0.1599	0.1553	0.8597	5.3773
73	34	1	5	33	0.1515	0.1507	0.1393	0.7044	4.6729
74	29	2	5	27	0.1852	0.1279	0.1161	0.5651	4.4181
75	25	1	3	24	0.1250	0.1042	0.0977	0.4490	4.3085
76	25	0	6	25	0.2400	0.0912	0.0802	0.3513	3.8526
77	19	0	5	19	0.2632	0.0693	0.0602	0.2711	3.9113
78	14	1	1	13	0.0769	0.0511	0.0491	0.2109	4.1296
79	12	0	2	12	0.1667	0.0471	0.0432	0.1618	3.4321
80	10	1	1	9	0.1111	0.0393	0.0371	0.1186	3.0185
81	8	2	2	6	0.3333	0.0349	0.0291	0.0815	2.3333
82	4	0	1	4	0.2500	0.0233	0.0204	0.0524	2.2500
83	3	0	1	3	0.3333	0.0175	0.0145	0.0320	1.8333
84	2	0	1	2	0.5000	0.0116	0.0087	0.0175	1.5000
85	1	0	0	1	0.0000	0.0058	0.0058	0.0087	1.5000
86	1	0	1	1	1.0000	0.0058	0.0029	0.0029	0.5000
87	0	0	0	0	0.0000	0.0000	0.0000	0.0000	
88	0	0	0	0	0.0000	0.0000	0.0000	0.0000	
89	0	0	0	0	0.0000	0.0000	0.0000	0.0000	
90	0	0	0	0	0.0000	0.0000	0.0000	0.0000	

#### **SI 8.4. Resampling the mortality data.**

Stages in the program.

1. Individuals whose interview was marked as “use = 1” are noted.
2. Then among those not yet in the sample (i.e. not interviewed or otherwise not yet eligible) search for those in one or more census and these are added to the sample.
3. Individuals in the sample are given a serial number from 1 to 1358. The NID corresponding to each of these serial numbers is recorded in an array “countarr”.
4. The resampling runs begin, during each run, a sample is drawn by a routine that is repeated 1358 times. This routine calls a random number between 1 and 1358, looks up the NID that corresponds to the random number (in countarr(random number,0), checks whether it is an interview person or a census person (from countarr(random number,1) and calls the relevant sub-routine (intvqx or censusqx) that builds the arrays to estimate qx.
5. When this routine has run 1358 times, another sub-routine “qxresults” calculates qx for ages 0 to 90 for females, males, and both. The results from this are added to a summary array (qxsamparr(runnr, gender, age)).
6. Another sub-routine “calcLxTxex” is called, having been passed the run number. It calculates lx etc and e0 for each run and stores e0 in an array. Then the next run commences.
7. After the last run, frequencies of each  $e_0$  are displayed for females, males and both, and can be saved as an access file [Data6].
8. Some checking routines were added which show that each serial number is used about equally often in the runs, and that the number of cases in each run matches up to the sample size.

### SI 8.5. Survival from 1977 to 1985.

Another test for secular change is to look at survival to 1985 of individuals who were recorded in Lars Smith's 1977 census. The period 1977 to 1985 may have been a good time for Hadza, they were left pretty much alone during this time. Because Smith made no age estimates I use our recent estimates of their year of birth. If mortality in that period was similar to that observed between 1985 and the 1990s, we should be able to predict by using my life table, the proportion expected to survive the 8 - year interval.

I counted individuals from the 1977 cohort as surviving if they had been seen in any of our censuses between 1985 and 2000. The proportion surviving is plotted in SI Figure 8.5 along with the proportion predicted. If mortality between 1977 and 1985 was similar to mortality between 1985 and 2000, then the predicted and observed lines should follow each other closely. The fit between observed and predicted appears to be only approximate. The table below shows the observed and predicted numbers surviving. These results are more encouraging. The match between observed and predicted is quite close. But the values predicted by adding, or subtracting, 15% of the observed  $qx$  at each age show that this test is not very sensitive, the observed numbers are not far from these test predictions. The excess of 60-64 year olds surviving may be a form of age heaping, they may belong spread out among the 50 - 75 year olds.

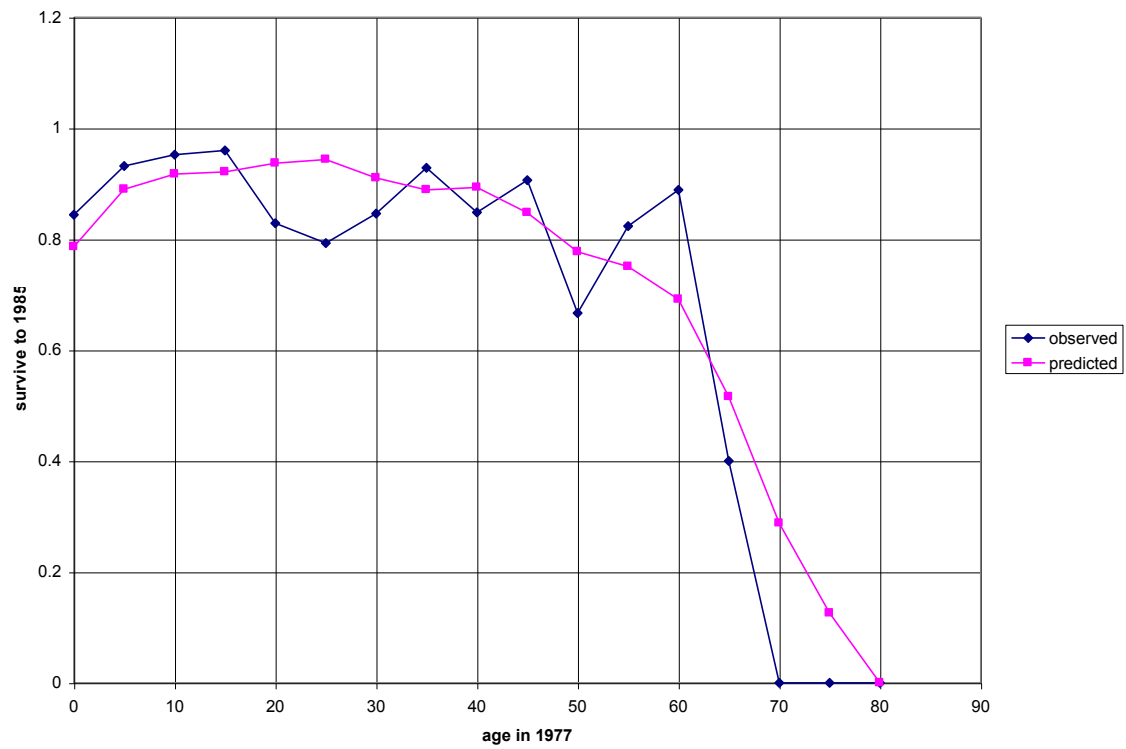
The 1977 cohort does not show the sex difference in mortality reported above. Males survived less well in childhood, aged 5 to 10, and slightly less well aged 45-55. But they appeared to do better than females at age 60-64, and just as poorly as females at age 0 to 4. Comparing the cohort follow-ups is made difficult by the different length of the follow up. The 1985 cohort were followed for 15 years, almost twice as long as the 8 years between 1977 and 1985. But this analyses does not give clear, or more than slightly suggestive, indications of a trend toward improvement or worsening in Hadza survival during the last four decades of the 20<sup>th</sup> century.

SI Table 8.5. Survivors of 1977 census cohort seen during 1985 - 2000. Number of cohort predicted to have survived to 1985 from mortality estimates. Numbers predicted from  $qx$  plus / minus 15%  $qx$ . The observed number are close to the predicted and between the 15% boundaries.

Survivors from 1977 to 1985	Females	Males
Predicted by $qx + 15\%$	199.9	176.3
Predicted	203.8	180.9
Observed	203	184
Predicted by $qx - 15\%$	207.8	185.7



SI Figure 8.5. Survival of 1977 cohort until 1985. Sexes combined.



## **8.6. Sex differences in mortality and survival of late middle-aged men and women.**

Our data on Hadza show a big sex difference in life expectancy at age 50. For females it is 20.37 years, for males it is only 15.7 years, a difference of 4.67 years. My doubts were of two kinds: 1) at these ages the sample is small; 2) there were slight differences in the methods we had used to estimate the ages of adult males and adult females.

My anxiety was much reduced when I looked yet again at table 6.1 and graph 6.3 in Hill & Hurtado (1996). Ache show the same phenomenon. Expectation of life at age 50 among the Ache in the forest was 19.2 for females, 14.8 for males, a difference of 4.4 years. Hadza appear to suffer much less homicide than Ache but Hadza men have accidents, such as falling out of Baobab trees (old men continue to climb trees for honey and fruit, sometimes fatally).

Sample size.

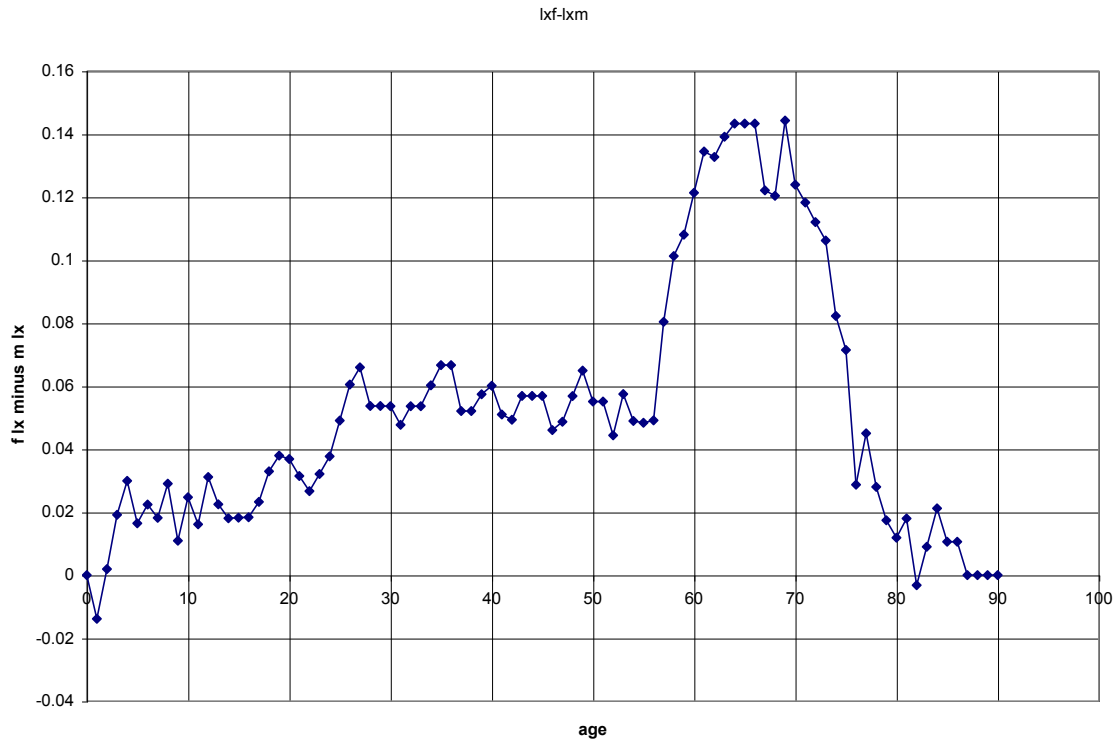
For Hadza females aged 50 – 65 we recorded only 12 deaths. The average number at risk in any of these years was 46. We recorded 26 deaths of males in the same age range. The average number at risk in any year was 37. This looks like a significant difference between numbers dying in this period compared to number at risk.

Methods of estimating ages.

In almost all studies of small, remote, subsistence populations, method of estimating individual ages is a very large issue. People cannot tell you their own ages, and there are few, if any, official records. You should be very cautious about reports that do not tell you how age was determined. Methods have varied from “eyeballing”, notoriously unreliable, especially for short field visits, to extremely strong evidence. One of the best is Pennington & Harpending’s (1993) study of Herero demography. Herero name years, and have done so for a very long time. Every Herero is told the name of their year of birth. Individuals remember sequences of year names. Early in the 20<sup>th</sup> century German missionaries in northern Namibia wrote down Herero year names and noted the western calendar year for them. Herero age estimates are thus extraordinarily good.

Hill & Hurtado describe their methods in detail and they seem excellent to me. Our methods were a different mix of some of the same methods: relative age ranking (who is older or younger than who), historical markers (previous censuses, early European visitors, including showing Hadza photographs from these visitors as aide memoires, a 6.4 earthquake, and so on). I have also used a number of methods to check our measures (chapter 4), re-estimating by our relative age information, the ages of those for whom we had well known birth dates, while blind to these birth dates. The estimates closely matched the known dates. Nonetheless I had concluded that while estimates were good to the nearest 2 years for most adults, the margin for error was greater for older adults, probably around 5 years.

SI Figure 8.6. Excess of female survival ( $l_x$ ) over male  $l_x$  (female minus male). The difference appears to accelerate during three periods: early childhood (1-4); adolescence-young adulthood (16-27); late middle age (56-64). Middle childhood (5-16) and adulthood (28-56) appear to be periods of relative equality in the survival of the two sexes.



Sex differences in mortality tested by resampling.

SI Table 8.6. Results of resampling mortality data - life expectancy at birth.

value of e0	females	males	both
26.50			
26.60			
26.70			
26.80			
26.90			
27.00		1	
27.10			
29.00			
29.10		1	
29.20			
29.30			
29.40			
29.50			
29.60			
29.70			
29.80		1	
29.90			
30.00			
30.10			1
30.20		1	
30.30			
30.40		1	
30.50		2	
30.60		1	
30.70		67	
30.80		344	
30.90		51	
31.00		11	
31.10		7	
31.20		4	
31.30		4	
31.40		3	
31.50		1	
31.60			
31.70			
31.80			
31.90			

value of e0	females	males	both
32.00			1
32.10			
32.20			
32.30			
32.40			
32.50			
32.60			
32.70			1
32.80			
32.90			2
33.00			1
33.10			242
33.20			212
33.30			11
33.40			10
33.50	1		10
33.60			8
33.70			1
33.80			
33.90			
34.00			
35.00			
35.10			
35.20	2		
35.30			
35.40	30		
35.50	303		
35.60	100		
35.70	28		
35.80	16		
35.90	11		
36.00	8		
36.10			
36.20			
36.30	1		
36.40			
36.50			
36.60			
36.70			
36.80			
36.90			
37.00			

I also used resampling to test the sex differences in another way. In each of 1000 runs, each individual in the actual study sample was allocated a gender at random, with no reference to his or her actual gender. In each run the program computes the  $qx$  and  $lx$  for the two false genders. The program reports the number of runs in which the difference between false “male” and “female”  $lx$  reaches the difference between real males and females observed in the actual real sample. The difference is significant at all ages.

### **SI 8.7. “Plenty of old people” versus “No old people” again. Age at death distributions and paleodemography.**

Because age at death structure is almost the only demographic measure that archaeologists can use, this is a good place to look again at the apparent conflict between data on longevity of living people, and conclusions about longevity in the past. In addition to estimating ages of skeletons, palaeo-demographers also examine pathology, and sometimes estimate fertility from pelvic bone changes. Because age at death distributions are so strongly influenced by fertility and population increase, independent fertility estimates might increase the value of death distribution data.

A contrast is often made (for example by Weiss 1981, and Austad 1997) between modern peoples, including ethnographically studied hunter-gatherers, whose populations include some numbers of vigorous 50-65 year olds, and prehistoric peoples, studied by palaeo - demographers using skeletal remains, among whom, reportedly, “almost everyone was dead by 45”. Studies using bone assemblages repeatedly report an age at death distribution quite unlike any modern population. It is widely believed that in the past people suffered much higher mortality, had much lower life expectancy at birth, shorter potential life spans, and fewer old people than contemporary hunter-gatherers. The issue is important to our understanding of many features of human life history: do we need to explain how longevity evolved? What is left of the research and theorizing on “cooperative breeding” and transfers of resources between generations if the over 45s (of either sex) are just a fleeting and novel oddity? This is so important for the study of evolution of human life history that I cannot avoid an extensive discussion. Even though there are signs that archaeologists have absorbed the lessons of human biologists it is still widely believed that the life histories of modern hunter-gatherers must be of very recent origin (eg Caspari & Lee 2004). I try to defend the view that the near universal modern human mortality schedule has some antiquity (as I have done elsewhere, Blurton Jones et al 2000, Hawkes & Blurton Jones 2005). Since the evidence that it does not, comes only from palaeodemography I have no alternative but to discuss some issues in that field. Its practitioners include some of the most able and accomplished, eminent and rigorously scientific people in anthropology. I am in awe of them. But I think they may be often wrong when they report on “old age” (which is seldom their central concern).

One issue of perspective needs to be cleared up first. What do we mean by “old people” or “older adults”? Contrasts between modern and “primitive” populations, discussions of “old age in primitive times”, are sometimes merely an issue of perspective on what we call “old”. A researcher or clinician involved with old age in North America or Europe today works in a context of 90 year olds. We have no confident record of a 90 year old hunter-gatherer anywhere. At most there might have been, during recent fieldwork, one individual in each of our study populations who could have been, just possibly, somewhere close to being 90 years old. None of us allocated quite so high an age to these individuals. The same could be said for almost any rural, third world population. Thus our data on contemporary hunters and gatherers in no way contradict those who find useful provocation in the view that natural selection can have exerted little pressure against a disease of extreme (usually) old age such as Alzheimer’s disease. Indeed the data support attempts to work out the implications of the much slower life cycle of the human primate (e.g. Finch & Sapolsky 1999, Sapolsky & Finch 2000). Hunter-gatherer researchers work in a different context, one of cooperative breeding (Hrady 2009), cross-generation helpers (Hawkes et al 1998), wealth flows (Kaplan et al (1994, Lee 2003), and species differences (Hawkes 2006, Hill et al. 2001, 2007, Muller & Wrangham 2014)). We know there are no 90 year olds, and there are at best tiny numbers of 80 year olds. Here the issue is 50 and 60 year olds. How important are men and women of these ages for economically supporting their descendents? Why are 50-60 year old humans so strong and vigorous when compared to 50-60 year old Chimpanzees? For how long in human history and pre-history have there been some of these people alive and strong enough to acquire resources they can supply to children and grandchildren? How far back in our evolution did we develop age structures like humans and different from Chimpanzees?

There are plenty of reasons to believe that the difference between the archaeological populations and the observed or recorded populations is methodological and not real. Many of the reasons have been given by palaeodemographers themselves. One of the classic papers in the field, Buikstra & Konigsberg (1985) contains (by my count) 17 cautions about inadequacies in data on older adults in the bone assemblages. Detailed suggestions about the methodological problems of “bone demography” can be found in the literature since its early days. Angel (1969) is one of the earlier authors to issue clear warnings about the difficulty of palaeodemography. But there are other observations that should make us wary. An issue that is often mentioned, only to be promptly forgotten, is cultural variation in customs concerning disposal of the dead. I will briefly discuss:

1. What do palaeo-demographers actually report?
2. Aging the bones. Is it just a giant “age heaping” phenomenon? [agingbones](#)
3. Preservation, loss of children and old people [babiesdisappear](#)
4. The time depths of Historical and Archaeological demography overlap. [timedepths](#)
5. What do Age at death distributions show? Influences of population increase and fertility on age at death distributions. [agdethdistr](#)
6. Methods for developing a life table from bone assemblages. [methods](#)
7. Attritional vs catastrophic assemblage – who gets found? [attritvscatstr](#)

## 1. What do paleodemographers actually report?

A great majority of paleodemography reports show the numbers of skeletons found and the age attributed to them. The resolution of the age estimates vary but most authors use very conservative age blocks. Most reports show very few individuals over 40, and a low percentage of the sample allocated to ages over 40, over 50, whatever they choose. If you compare these distributions to age at death distributions from Coale & Demeny or any contemporary population the difference is striking (e.g. text figure 8.1). But it also often takes a strange form. The archaeological populations have very large numbers of dead 25 to 40 year olds. In some samples children and infants are clearly under-represented and this complicates the comparison of proportions or percentages. But it is impossible to dispute that the reports show very few people estimated to have been older than say 40, or 50, people of the age of many vigorous Hadza grandmothers or expert hunters raising a second family of children by a new wife. We must also acknowledge that paleodemographers have put enormous and repeated effort into developing and checking their methods for estimating ages from bones.

## 2. Aging the bones. Is it just a giant “age heaping” phenomenon?

Demographers are familiar with ages being rounded up or rounded down by informants and researchers alike. They label this “age heaping”, and notice it when the tens and fives have more cases than the numbers in between. For example in chapter 4 I reported evident age heaping among our older Hadza, people were more likely to be scored as 65 or 70 than as 66, 67 or 68. Could the archaeological data simply be another example, on a large scale? Many of the age at death distributions have large numbers of deaths to 25-35 year olds. Where did all these young adults come from?

We could illuminate this by showing whether in an age at death structure from a contemporary population there are too few or too many old people to account for the excess of young adults in the bone samples. If we rounded their ages down all the way from 80 to 35 would there be far too many, or far too few to map onto an archaeological bone assemblage? Let's imagine an extreme age heaping of the Hadza population. Suppose we went to the Hadza and just made eyeball estimates of age. Like many Africans, slender and smooth skinned, Hadza adults look quite young to European eyes at first. We might classify all but a few wrinkled and stooped individuals as in their 30s or early 40s and keep those age estimates for the duration of the study (Howell (1976) quotes a very similar actual report from southern Africa). Then, suppose we used these estimates when tabulating the deaths that we recorded. What would the age at death distribution look like? We can simulate this extreme age heaping with model north 7 (or any population with a written record). If we take people away from their real ages at death and heap them all up among the 30, 35 and 40 year olds we get quite a close match to Libben. Libben often features in these comparisons because it is so well known, is a large well preserved sample, and was expertly studied and reported. In model North 7



there are just the right percentages of people dying after age 35 to mimic the Libben percentage distribution. This means that there are enough 30-40 year olds in the Libben sample to be stretched out enough to give a normal human age at death distribution. This exercise does not prove that the bone assemblages are severely “age heaped” but it should make us wonder.

Palaeodemographers have tested a number of standard samples from different countries and conclude that similar changes happen to our bones as we age whoever or wherever we are. Some reference samples are not so modern, and several were probably from very unhealthy people, who might show a high bone age at a low chronological age. The archaeological demographer has to suppose that the criteria of age described for a 50 year old 19<sup>th</sup> century factory worker represent all 50 year olds. It is possible that people who survive to 50 under the balanced, if limited, nutrition of the vigorous hunter-gatherer life have much younger looking bones than a 50 year old 19<sup>th</sup> century worker in a factory with no pollution or safety regulations. A “frailty” argument is also possible: under hunter-gatherer conditions where only the fittest survive early childhood, the 50 year olds could really have “younger” bodies than the average in a less fiercely selected (lower mortality) population. But Bengtsson & Brostrom (2009) found that individuals who survived a period of high child mortality nonetheless suffered higher adult mortality, the opposite of the frailty argument. Bocquet-Appel & Masset (1982) point out that cranial sutures and aging seems to have changed in the last 100 years. Some of the standard samples cover only part of the age range, and B-A & M’s argument about the effect of this on conclusions about life history may not have been fully appreciated.

There are comments in the literature about the greater inaccuracy of the bone aging methods for older individuals. One quite well known example is the Spitalfields study of 18<sup>th</sup> century Londoners (Mollesen et al 1993). Bone age estimates were compared with recorded, written ages of the same individuals. It was found that ages of many older people were seriously underestimated by bone age. Different methods of estimating ages from bones were compared by Wittwer-Backhofen et al. 2008. New methods are being sought and tested all the time (e.g. Griffin 2009, DiGangi et al 2009, Cardoso & Henderson 2010).

### 3. Preservation, differential loss of children and elderly.

Philip Walker and colleagues in an exceptionally important study, compared the written records of an early mission in California with the bone assemblage excavated from its cemetery. Walker et al (1988) was able to demonstrate that the bones of small children and old people disappear faster than the bones of young adults. Archaeologists are alert to differences in quality of preservation but do we know how the loss of the extreme ages varies with the state of preservation of those we find? Does judging preservation by the state of the young adult bones correctly assess the loss of bones of young and old? In working only with the best preserved do we risk other biases in the samples? Samples from less organized societies may differ in many ways, disposal of corpses can differ by age and sex. Differential burial and preservation of the bones of the

very young seems widely acknowledged. I have not seen this acknowledged with respect to the bones of older adults who had “reached their time”.

#### 4. The time depths of Historical and Archaeological demography overlap.

Bocquet-Appel & Masset (1982) began their famous critique by pointing to the startling difference between the results of historical and archaeological demography “graveyard populations (some of them barely older)” (1982:321). The contrast has fallen out of sight behind the efforts of palaeodemographers to strengthen their methods. We should look again at the contrast between written records and archaeological interpretations, it is even more startling than Bocquet-Appel & Masset suggested.

As Bouquet-Appel & Masset recognized, much of the detailed archaeology and statistical analysis by paleo-demographers is done on quite recent populations. Especially in North America, palaeo-demographers have worked on populations from 1000 BC to say 1500 AD. There are written records from Old World and Asian populations from this time period, and these fit well within the range of mortality of contemporary hunter-gatherers and rural third world populations, they are just like people. The difference has nothing to do with the antiquity of the population studied. It is something to do with the methods. Written people look like contemporary people, bone collections look like something else. For example, Bagnall & Frier (1994 figure 4.2) show a female age structure from censuses in Roman Egypt (AD 12 to AD 259) that closely matches C&D West 1 and includes plenty of women over 40 ( $e_{40}$  for West 1 is 19.3 years). Their population lived some 4-500 years before several of the well-known North American archaeological populations that are reported as if they had a completely different age structure. Others among the “written populations” pre-date some of the archaeological populations. For example the records used by Zhao (1997) begin in about AD 0.

Since the new world was populated rather recently (c.14,000 ya), and by modern *Homo sapiens*, we should be surprised if there were radical differences in their life histories. Howell (1976), Roth (1992), and Paine and colleagues are surely right to suggest we use modern population models to test for similarity or differences between the bone assemblages and model modern human populations. But when we want to think about more ancient populations, such as the Neolithic sites used by Weiss (1973: 96), or even older sites or peoples, such as Neanderthals (Trinkaus 1995), we may begin to wonder whether radical differences should be expected. But the size of the differences that are sometimes claimed is extraordinary. Why would the early farmers of Catal Huyuk have life expectancy at birth of 13.8 years (Weiss 1973 table A.24 from Angel 1969) while Hadza, !Kung and Ache have life expectancies over 30. If farming and city life was really so horrible, why put up with it?

Historical demography from Europe and Japan shows that non-industrialized farming can generate age at death and mortality patterns very similar to that of the Hadza and other foragers, and have done so for periods of up to several centuries (examples in Laslett 1995, Kinoshita (1998), and Jannetta & Preston 1991, including their Table 3, and

their appendix life table for 1776-1795). Japan is an interesting case because there are both archaeological studies of bone populations (such as Nagaoka et al. 2006, and Nagaoka & Hirata 2007) and historical studies. The studies in both fields seem to me to be reported comprehensively and with great expertise. Historical demographers have technical problems just as do archaeological demographers and these seem to be tackled with equal vigor in each field. The Japanese temple historical records are especially valuable, for example including records of the deaths of emigrants (Jannetta & Preston 1991). In Japan we will get close to having historical and archaeological studies of contemporaneous populations that lived in similar economies. While Nagaoka & Hirata (2007) samples are roughly contemporaneous with Kinoshita (1998) and Jannetta & Preston (1991), the former concerns a city population and the latter two deeply rural. The early city conditions described could generate a shortage of older adults, as could migration back to their rural homes.

Ancient Rome and Ancient Greece offer more comparisons, though it is difficult to disentangle city – countryside, homeland versus empire, elites versus plebians. The findings are enough to make us continue to puzzle about the scarcity of older adults in the bone populations. Even if Roman records were strongly biased in favor of the wealthy, well-known, and male, there are enough written records of adults past the age of 45 to show us that the potential for a contemporary forager pattern of longevity existed at a time when the paleodemography record suggests otherwise (examples in Angel 1969, and references in Woods 2007). Probably we will never have historical (written) records much older than 2000 years ago, whereas much older bone assemblages have been studied. But the startling difference between the historical and bone demography of the last 2000 years in more than one continent should make us very wary of the idea of a sudden arrival, in different places at different recent times, of the now universal human pattern.

5. What do Age at death distributions show? Effects of fertility and population increase on age at death distribution.

In simple societies with high mortality, it has been noted that fertility and population increase are far stronger than mortality as influences on age at death structure. Milner et al (1989) compared age at death distributions generated by fertility and mortality of !Kung and Yanomamo. They noted that the influence of fertility on age at death distribution was greater than that of mortality in this comparison, as we also just saw in the Hadza data. One can see the same thing by comparing various C&D models, looking at the age at death distributions.

Archaeologists have often noted the absence of infant and early childhood remains from their samples. In demography of high mortality living populations these age groups have a very large influence on figures such as life expectancy at birth (so do old people), and form the largest age group in age at death distributions. Their absence from archaeological samples makes all the arithmetic more difficult.

## 6. Methods for developing a life table from bone assemblages.

Methods used to interpret the data range from the nonexistent – simply assuming the age at death distribution is the same as the age distribution, via the notoriously chancy - building the life table from the deaths alone, to the much improved and logically defensible. Since its origin, palaeodemographers have repeatedly written about the difficulties of their field. An important series of papers by Paine, Milner, Harpending and colleagues (e.g. Milner et al. 1989, Paine 1997, 2000) and by Konisberg & Herrmann(2006) shows the difficulty of working from skeletal remains to an age structure, and promotes a different perspective. These authors propose that bone assemblages be matched to age at death distributions predicted by a range of modern human population models. The use of models of known populations has been repeatedly advocated, for example by Howell (1976, 1982), and Roth (1992). The starting assumption is that the archaeological collection was from regular humans, not markedly different from, for instance, the 326 world wide populations, studied in the 19<sup>th</sup> and early to mid 20<sup>th</sup> centuries and used to build the Coale & Demeny models.

Several authors directly derived survivorship and  $l_x$  from a death distribution, some of them warn that this procedure depends on the population being stationary ( $r = 0$ ). The total number of dead individuals is taken as the starting population. The number of dead infants divided by that number gives  $q_0$ , the risk group for 1-4 year olds is the total minus the dead infants, and so on. Let's think about using this method on a contemporary, observed population, quite tempting in view of the difficulties of being sure of the sample at risk. Howell (1979:Table4.4 and pages 87-90) showed how this might be done, suggesting “imagine for a moment...that the !Kung had established a graveyard in 1963”. Analyzing just the !Kung who died between 1963 and 1974 (her Table 4.4) she developed an  $l_x$  curve and found a life expectancy of 34.57 years, rather close to her estimate for a previous time period. She then remarks: “Demographers are generally wary of the validity of computing mortality from a collection of deaths ... because the denominator of the  $q_x$  measures, conceptually, is the number of people at risk of dying in the living population” (just like the palaeodemographer Angel cited above). When she calculated survivorship for the 1963-74 period by the usual demographic method the life expectancy was over 50.1 years (Howell 1979 Table 4.6). She then proceeds to simulation runs to assess the reliability of this surprisingly high figure. If we compare these two methods for the Hadza we again get radically different results. Life expectancy at birth calculated on 418 deaths of males and females is 20.7, well below my observed 32.7.

Why does this happen? Probably the usual reason is that few populations are stationary. In a contemporary population where we collect data on deaths in the past few years, each dead person has contemporaries who are still alive. Even the oldest dead people may have one or two age mates who outlive them. Among those who died in mid

life or earlier, there will be many who outlived them and still have not arrived in our list of ages at death.

## 7. Attritional versus catastrophic assemblages. Who gets found?

An important development has been the recognition that some assemblages result from a sudden, large scale catastrophe, a raid or epidemic, while others represent the gradual accumulation of the results of “day to day” attrition. The pattern resulting from a war (young men killed, young women stolen or killed while resisting, might be very different from the pattern resulting from an epidemic (children and very old people most at risk). Given the ability of archaeologists to distinguish injuries from marks of disease, confusion seems unlikely. Given perfect preservation, a catastrophic assemblage has some chance of resembling the living age structure (perhaps everyone was struck down indiscriminately). An attritional assemblage, like any age at death distribution, is a result of an interaction between age structure (the number of people in each age) and the rate of deaths at each age. By simulating population crashes Keckler (1997) was able to show how catastrophic assemblages might be recognized. Catastrophic events may be responsible for some of the well studied bone assemblages with unusual age at death distributions. Assemblages arising from sedentary populations presumably differ from those arising from mobile populations.

The Hadza can illustrate the difficulty of finding a good “attritional” collection from a mobile population. In such a population the chance of finding a collection of skeletons that represent the normal age at death distribution is very slender. There are on average 28 deaths per year. In the average census there were 25 camps. We can expect just over 1 death per year per camp. Camps move quite frequently, 6.5-9 times per year according to Marlowe (2010: 263). Let’s call it 8 moves per year. The chance of a death at a particular camp site is 1/ 8 per year, one every 8 years if people return to the exact same spot. If an adult dies, people leave the location. People often camp again in the same general area but seldom at exactly the same site as one of their previous camps. But eventually people may lose track of exactly where they camped and in some favored locations there is some chance of camps being superimposed on previous camps. Then, after some years, there is some chance of another corpse being left behind at or near (within 100 meters) of a previous corpse. The amount of time needed to accumulate a significant sample of remains would be truly immense. Suppose people forget where someone was buried or left behind, or no longer care, after 10 years (too soon), 20, or 30 (perhaps too long?). Then if someone dies at this camp, a 1 in 8 chance, a second corpse may be left nearby in 80 years, or 160, or 240 years. A collection of ten corpses would take between 800 and 2400 years to develop in one place, and cover a period of many droughts. There can be very few sizable attritional assemblages of mobile hunter gatherers out there to find. The perceptive archaeologist would easily recognize the difference in time between the deaths.

In contrast, if there are violent raids, or serious epidemics, and a large proportion of the people in camp died, the archaeologist has only to chance upon a raided or infected camp to find a useful sample of remains. This seems to me to imply that any sizeable collection of remains of mobile hunter – gatherers should a priori be assumed to represent a catastrophe, not the death distribution of a stationary, stable population. Even so, the age structure of the assemblage would depend on how people reacted to the raid. Would the old run away with the children and hide, while the young adults stayed to fight?

## 7. Explanations for differences between ancient and modern.

Those who believe that the longevity of all observed contemporary and historically known populations is very new, its antiquity disproved by the archaeological samples, have offered a variety of explanations for the supposedly recent change. Modern life is easier than prehistoric life, and modern medicine greatly reduces morbidity and mortality. It undoubtedly does. But does it account for the mortality and age structure seen among modern third world populations before modern medicine was widely available? !Kung in the 1950s-1960s, and Ache in the “forest period” before contact, and the Hadza, with minimal contact with modern medicine, show a “normal” human mortality and age at death distribution. Some attribute the difference to “culture”, as if the people of Libben, or Catal Huyuk had no culture. If the difference between bone populations and written (historical, or recently observed) populations is real we have a lot of complexity to explain. Why did North Americans around 1500 AD die by 45 while Japanese farmers in the 1700s, isolated from the western world, live into their 70s? What made the 14<sup>th</sup> century Japanese become able to live not just to 45 but to 75 by the 1700s? Why were ancient Chinese and the population of Roman Egypt able to live as long as Hadza, while the bone population of Athenian Greeks could not? It makes more sense to ponder the methodological difference than to conjure up the many ad hoc explanations for the many changes.

Lovejoy et al. (1977) made an interesting “frailty” argument about the difference between a sample such as Libben and contemporary, observed populations. All of the latter have been exposed to modern diseases, the mass killers like measles and whooping cough. These kill many infants and children, selecting in favor of individuals with the most competent immune systems. These are then able to live long lives by virtue of their greater resistance to infection. The pre-contact North American samples were exposed to few if any of these diseases. Less immune-competent individuals could survive childhood, only to be struck down by some disease during early adulthood, few would survive into old age. The argument seems plausible, although it is difficult to believe the effect would be extreme enough to account for the reported difference in age at death distributions. It is likely true that even the most isolated modern populations had by the time of their observation been long exposed to modern disease. Even the “uncontacted” Ache, to judge by Black et al. (1974) demonstration of exposure to “modern” diseases among other little contacted S American populations, may not contradict Lovejoy’s argument. But Old World archaeological populations dated any time in the last several

thousand years have been exposed to the modern diseases, as far as we can judge from the molecular history of these diseases. These populations, if Lovejoy's argument is correct, should show the death distribution of modern, observed, contacted populations. They do not, they show the typical archaeological pattern of very few older people.

However, somewhere back in our evolution our ancestors probably lived no longer than Chimpanzees. Judging by size, Australopithecines had a life history quite similar to Chimpanzees. Indeed the Chimpanzee  $e_{15}$  in Hill et al 2001 (15.4 years for females and 14.2 for males) closely matches the  $e_{15}$  estimated by Weiss 1973 Table A20 from the Australopithecine life table bravely created by Mann (1968) from fossils. When did our ancestors begin to live significantly longer than chimpanzees? Several offerings are in the literature. Weiss (1981) pointed out the strong correlation across taxa between body size, brain size and maximum life span, and suggested that a modern human maximum life span of 95 years was in line with the regression and needed no special explanation. He also predicted life spans for fossil hominids based on their estimated body weights and brain sizes and predicted 93 years for Neanderthals, and 69-78 for *Homo erectus*. O'Connell, Hawkes and Blurton Jones (1999) argued that a major change in life history accompanied the origin of *Homo erectus*. We suggested that the relatively sharp increase in size with *H. erectus* should imply later maturity, which is expected to follow from lower mortality and longer lifespans. Others have argued for longer life and lower mortality arriving with *Homo sapiens*, or even with only the more modern and local forms of our species (e.g. Upper Palaeolithic Europe Caspari & Lee 2004). These leave us without an explanation for the similar size of *erectus* and *sapiens*. Since age at maturity or first reproduction is well correlated with size and life span across taxa, the investigation of speed of development and age at maturity has implications for dating the arrival of human longevity. So far, these investigations, such as (Dean & Smith 2009) seem to conclude that development of *Homo erectus* individuals was quite a bit faster than seen among modern humans, which might imply greater mortality and shorter life spans.

A simple taxonomic parsimony argument appeals to me as a way to set a maximally recent date for a character. Characters found in all representatives of the species are likely to date from the origin of the species but may or may not also have been present in its extinct ancestors. Howell (1976) made a similar argument. But Caspari & Lee (2004, 2006) argue against such a view, on the grounds that their data suggest that only upper-palaeolithic Europeans showed a proportion of old to young adults approaching modern figures. If their tooth wear interpretations are correct, Caspari & Lee's logic is secure, though we might ask to see samples of reasonable size from other parts of the range of *Homo sapiens*, particularly Africa. The rather recent (AD 970-1200) Mapungubwe (Heeneberg & Steyn (1994 table1) show 11 old (30+) and 17 young adults (15-29) which gives a quite low OY ratio of 0.65. The simple alternative is independent evolution of the character in different parts of the range, and a subsequent filling in by the supposedly advantageous character. Imagine the capacity for language evolving independently everywhere in the world. Much simpler to suppose it originated at the latest with *Homo sapiens*. Nearly all living humans have a similar life table, one that differs quite markedly from Chimpanzees, mainly in adult survival. The real exceptions

are people in industrial societies with a convex lx curve, with a great majority of deaths in the 80s. Demographers know a great deal about their history. It is truly recent. So based on the taxonomic parsimony argument it seems most likely to me that the basic human life table evolved before the expansion out of Africa. Since that expansion came from a relatively small part of our range within Africa, and well after the divergence of Khoisan speakers, the human life table likely arose at least as far back as the origin of modern Homo sapiens. The argument from size implies a similar life table for Neanderthals and any other large archaic Homo. The description of the occasional very old Neanderthal may support this view. My taxonomic parsimony argument then brings us back to our 1999 suggestion, that long lifespans began with Homo erectus.

A new approach to determining when human lifespans lengthened is beginning to make its presence felt. The field will no longer be a simple contrast between the findings of anthropologists and archaeologists. The genes responsible for variation in life span are becoming known. And eventually their times of origin will be estimated, albeit with their characteristically wide confidence intervals. Already, Finch & Stanford (2004) in their paper "Meat-adaptive genes and the evolution of slower aging in humans" outline contributions of the ApoE3 allele to longevity by reducing inflammatory responses, increasing bone strength, and adjusting cholesterol responses to dietary fat. Citing Fullerton et al. (2000) they report the ApoE3 allele as dating from about 311 kya (176-579 kya) with an expansion around 226 kya. They comment that this dates the spread of apoE3 before expansion out of Africa and perhaps allows its presence in Neanderthals and earlier Homo in Africa. They tentatively implicate a few other genes in the process, with similarly timed histories. Other genes affecting vigor and longevity are found in mitochondria and among the nuclear genes controlling them (e.g. Wallace 2010). When the histories of these and other genes that increase life span are developed, and compared to those of Chimpanzees, we may have clusters of origin times for changes in longevity that can be attached to the fossil record.



**SI 8.8. Data table for text figure 8.5.**

Proportion of each age cohort surviving from 1985 to 2000.

Age in 1985	observed	predicted
0	0.698925	0.70448
5	0.793651	0.826481
10	0.857143	0.859804
15	0.837838	0.873367
20	0.93617	0.891264
25	0.913793	0.848494
30	0.833333	0.825411
35	0.625	0.776562
40	0.8	0.754803
45	0.625	0.666016
50	0.653846	0.619378
55	0.545455	0.524749
60	0.285714	0.31247
65	0.266667	0.128798
70	0	0.020513
75	0	0
80	0	0