Human mortality improvement in evolutionary context

Oskar Burger[a,1], Annette Baudisch[a], and James W. Vaupel[a,b,c,1]

[a]Max Planck Institute for Demographic Research, D-18057 Rostock, Germany; [b]Institute of Public Health, University of Southern Denmark, DK-5000 Odense, Denmark; and [c]Duke Population Research Institute, Duke University, Durham, NC 27708

Contributed by James W. Vaupel, September 10, 2012 (sent for review July 17, 2012)

Life expectancy is increasing in most countries and has exceeded 80 in several, as low-mortality nations continue to make progress in averting deaths. The health and economic implications of mortality reduction have been given substantial attention, but the observed malleability of human mortality has not been placed in a broad evolutionary context. We quantify the rate and amount of mortality reduction by comparing a variety of human populations to the evolved human mortality profile, here estimated as the average mortality pattern for ethnographically observed hunter-gatherers. We show that human mortality has decreased so substantially that the difference between hunter-gatherers and today's lowest mortality populations is greater than the difference between hunter-gatherers and wild chimpanzees. The bulk of this mortality reduction has occurred since 1900 and has been experienced by only about 4 of the roughly 8,000 human generations that have ever lived. Moreover, mortality improvement in humans is on par with or greater than the reductions in mortality in other species achieved by laboratory selection experiments and endocrine pathway mutations. This observed plasticity in age-specific risk of death is at odds with conventional theories of aging.

biodemography | cross-species comparison | life history evolution | phenotypic plasticity | human lifespan

The role of comparative biodemography is to elucidate fundamental demographic patterns and develop evolutionary explanations for their variation (1, 2). Identifying any fundamental pattern requires the appropriate metrics; understanding the evolutionary context for variation in human mortality patterns requires an evolutionarily relevant comparative baseline. Ideally, this baseline should approximate the average age-specific levels of mortality experienced through most of human existence and should not be limited to a specific calendar year or country (e.g., Sweden in 1751). Because of the hard work of field anthropologists, such a baseline exists, as the average mortality profile of ethnographically observed hunter-gatherers (3). Using this baseline as the standard for comparison provides the necessary means of gauging the rate and timing of mortality improvement in a broad evolutionary context.

Many studies have demonstrated the remarkable ability of humans to prolong the length of life. Oeppen and Vaupel (4) show that the best-case national life expectancy at birth has improved in a stunningly linear pattern between about 1840 and the present such that in the longest-lived national populations life expectancy has increased by about 3 mo per year. Similarly, Tuljapurkar et al. (5) show that the extensions in longevity are occurring because of rapid and steady progress in lowering mortality at all ages, and that the limits of these reductions are difficult to predict.

The social, economic, and health implications of the reductions in mortality have been discussed extensively (6–8). An element of the discussion that has been largely missing is the implication of mortality change for evolutionary theories of aging and, more generally, for biological understanding of plasticity of the mortality profile. This omission is odd given that age-specific mortality patterns are an essential driving force in the evolution of life histories (9). To better understand the evolutionary significance of the great changes in human mortality profiles and to more accurately describe the rate and magnitude of mortality reduction, we make a number of basic comparisons between the evolved human mortality profile and those of human populations from the present and recent historical contexts. We also make comparisons of the life expectancy extension achieved by humans with those achieved in the laboratory via experiments on model organisms. Such comparisons serve to frame human mortality improvement in a broad comparative light. We use this coarse-grained comparative approach to answer three basic questions about changes in human mortality profiles: (*i*) How can we place the observed human mortality improvement in a broader evolutionary framework? (*ii*) How much has mortality changed compared with the "typical" human mortality profile (where "typical" refers to the average mortality profile experienced during human evolution)? (*iii*) How does human mortality change compare with that observed in other species?

Results

Comparing low-mortality human populations to the hunter-gatherer profile reveals dramatic reductions in the probability of death at all ages (4, 5) (Fig. 1*A*). The percentage improvements have been greatest at younger ages, but the absolute improvements are larger at older ages when death rates are high. For instance, the expected annual probability of death for a 65-y-old hunter-gatherer is about 5.3%; in contrast, for 65-y-olds in Japan today, the chance of death is only about 0.8%. Although mortality drops at all ages, the age at lowest mortality changes very little and the shape of the mortality profile has not changed much despite the huge reductions in its actual values.

The variation between the highest and lowest mortality populations is remarkably large (Fig. 1*B*). The lowest age-specific death rates are enjoyed by the current populations of countries such as Japan and Sweden. The worst-case mortality for humans is approximated by 19th century slaves on Trinidad, who suffered death rates at all ages that were higher than those for hunter-gatherers. Acculturated hunter-gatherers experience lower mortality than hunter-gatherers due to modest access to Western medicines and food subsidies (3). The mortality trajectories of the populations of Sweden in 1800 and 1900, and Japan in 1947 are roughly similar to the trajectory estimated for acculturated hunter-gatherers, implying that this amount of mortality improvement is fairly widespread, especially in nonindustrial contexts, and can be achieved without the large improvements of infrastructure and medicine that came later.

The most striking finding is that the mortality profile of hunter-gatherers is closer to that of wild chimpanzees than it is to the recent profiles for Japan and Sweden. This result is implied by visual inspection of Fig. 1*B* and is consistent with life expectancies for each population, but can be more clearly seen by calculating ratios of age-specific chances of death (Fig. 1*C*). Up until age 15 or so, hunter-gatherers experience death rates >100-fold higher than in today's Japan and Sweden, and hunter-gatherer mortality remains >10-fold higher for the entire life span (Fig. 1*C*). In

Author contributions: O.B., A.B., and J.W.V. designed research; O.B. performed research; O.B. analyzed data; and O.B. and J.W.V. wrote the paper.

The authors declare no conflict of interest.

Freely available online through the PNAS open access option.

[1]To whom correspondence may be addressed. E-mail: burger@demogr.mpg.de or jwv@demogr.mpg.de.

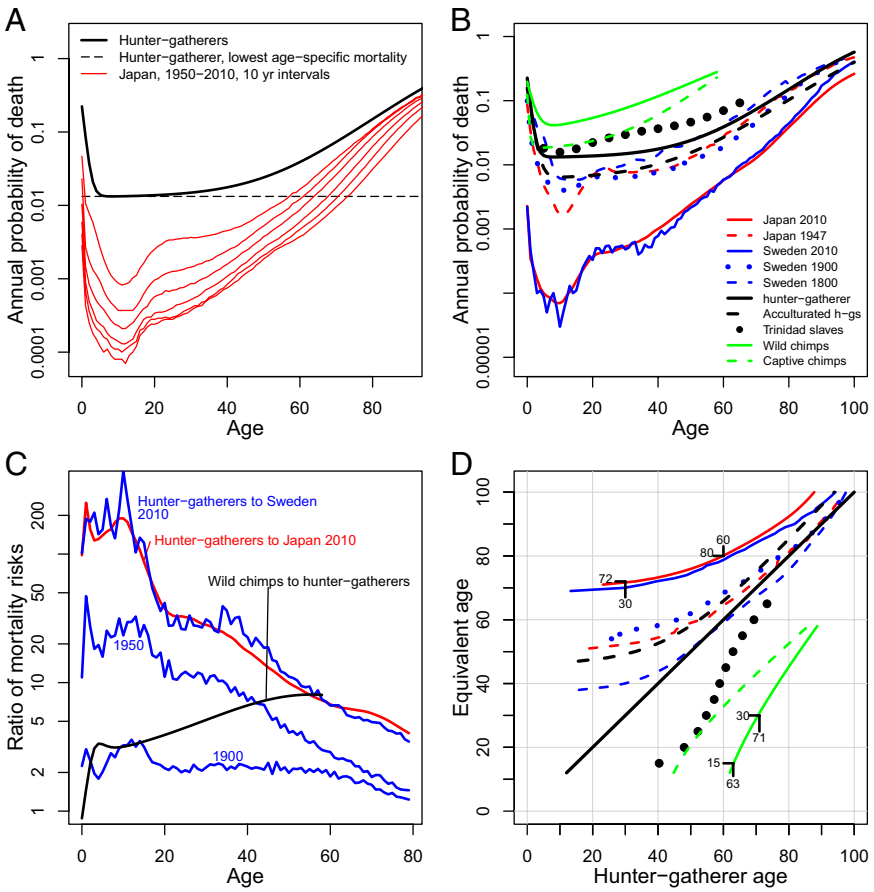

Fig. 1. (*A*) Annual probability of death for hunter-gatherers compared with today's longest living nation, Japan, at 10-y intervals through time. (*B*) Annual probability of death for a variety of human populations, along with wild and captive chimpanzees. The difference between present-day Japan and the Trinidadian slaves frames nearly all of the known variation in mortality across human populations. The hunter-gatherer curve approximates the typical human mortality profile over evolutionary time. (*C*) Ratio of mortality risks. Each curve gives the ratio of annual probabilities of death by age; hunter-gatherers to Sweden 1900, 1950, and 2010 in blue; hunter-gatherers to Japan 2010 in red; and wild chimps to hunter-gatherers in black. The magnitude of improvement of Japan 2010 and Sweden 2010 over the evolutionarily typical pattern for hunter-gatherers is much greater than the gap between hunter-gatherers and chimpanzees. (*D*) Ages of equivalent mortality. Each of the populations in *B* is compared with hunter-gatherers in terms of equivalent age. For each of the populations, the *x*-axis is hunter-gatherer age and the *y*-axis is the age at which each population has the probability of death equivalent to that of hunter-gatherers. Lines above the diagonal are for populations with lower mortality than hunter-gatherers. Lines below the diagonal are for populations with higher mortality than hunter-gatherers. Examples are indicated with ages and black dashes. For instance, reading upward from the middle of the *x*-axis to the top of the plot, we see that a hunter-gatherer at age 60 has the same probability of death as an individual in Japan at age 80. All curves are based on raw data except the Trinidad population and Sweden 1800, which were smoothed with a loess function for plotting purposes only. Data sources are as follows: Japan (17, 39); Sweden (17); Trinidad (37); hunter-gatherers, acculturated hunter-gatherers, wild and captive chimps are based on Siler functions from Gurven and Kaplan (3). In all cases, mortality is for the total population, both sexes combined. Note logarithmic increments on the *y*-axis in *A*–*C*.

contrast, the ratio of death rates for wild chimpanzees to hunter-gatherers is <10 at all ages and is greater at older ages than at younger ages. Remarkably, the mortality ratio of hunter-gatherers to Sweden in 1900 is smaller, <5 at all ages. That is, Swedes in 1900 had mortality profiles closer to hunter-gatherers than to the Swedes of today. This relative difference between Swedes recently and those 100 y ago has emerged in a rapid revolutionary leap, as this distance is far greater than that between hunter-gatherers and chimps (Fig. 1*C*). The recent jumps in mortality reduction are remarkable in the context of mammal diversity because age-specific death rates for hunter-gatherers are already exceptionally low, probably among the lowest of any nonhuman primate or terrestrial mammal (especially if body size is controlled for), and lower than even captive chimpanzees at all ages (Fig. 1*B*) (10). The human mortality profile, however, is so plastic that over the past century the populations doing best managed to achieve very large reductions in death rates that were already low compared with those of other species.

Differences in mortality between populations or species can be further illuminated with the concept of ages of equivalent mortality (11–13). With hunter-gatherer mortality as the evolutionary baseline, a population's equivalent age is the age at which it has the same probability of death as the average hunter-gatherer at some specified age (Fig. 1*D*). For example, hunter-gatherers at age 30 have the same probability of death as present-day Japanese at the age of 72: hence the age of a person in Japan that is equivalent to a 30-y-old hunter-gatherer is 72. In other words, compared with the evolutionary pattern, 72 is the new 30. Furthermore, the annual probability of death for a 15-y-old wild chimp (about 4.7%) is experienced by an average hunter-gatherer at the much later age of 63. In contrast, the annual probability of death for a 15-y-old hunter-gatherer is 1.3%; Swedes reach this probability of death at age 69.

Comparing the lowest annual probabilities of death reveals variation in the best age experienced by a population with respect to mortality, and tracking it through time reveals how recent the major reductions in death rate occurred (Fig. 2). Progress in mor-

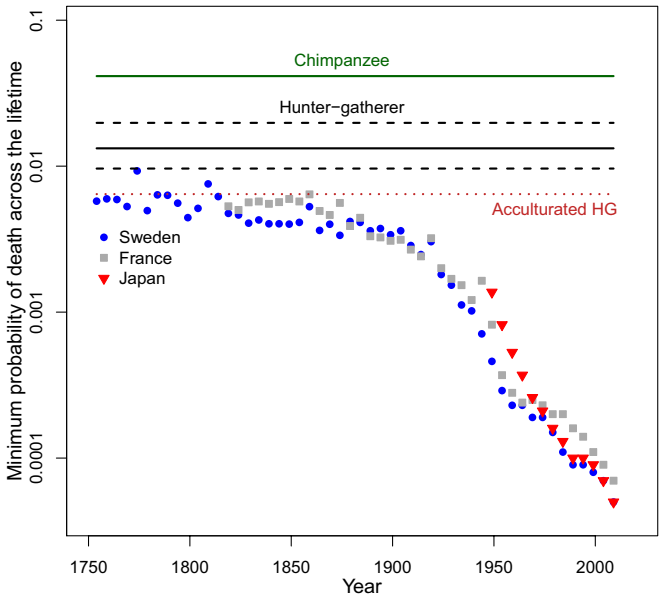

Fig. 2. Probability of death at the age when this probability is lowest. For humans, this generally occurs at ages from about 9 to 15, but note that mortality tends to change fairly little from about 10 to 30 or 35. For chimpanzees, the minimum mortality here is at age 9. France, Sweden, and Japan show similar trajectories. France and Sweden begin to achieve major reductions in the lowest level of mortality at roughly the same time, just before 1900. The solid black line is for the average hunter-gatherer and is bracketed by a pair of dotted lines representing uncertainty around the minimum probability of death, which we estimate as the highest individual value (the Hiwi) and the lowest (the Agta) from the populations represented in the average curve.

tality reduction below the evolutionary norm began to accelerate about 1900. Until the late 1800s, the world's lowest mortality populations were not far below the observed range of variation for hunter-gatherers around the prime of life (when mortality is lowest), yet a greater than species-level jump in mortality reduction has been made since. Overall, the bulk of this larger gap in mortality between the longest-living populations and hunter-gatherers occurred during the past century. The smaller gap between the evolved human mortality profile and chimpanzees developed over the vastly longer period of about 6.6 million years (14), since the split from a common ancestor with chimpanzees. In gross comparative terms, this means that during evolution from a chimp-like ancestor to anatomically modern humans, mortality levels once typical of prime-of-life individuals were pushed back to later ages at the rate of a decade every 1.3 millions years, but the mortality levels typical of a 15-y-old in 1900 became typical of individuals a decade older about every 30 y since 1900.

Reductions in age-specific human mortality over the past few generations can be compared, albeit cautiously, with experimental reductions in laboratory species. Although longevity can be defined by various metrics, we compare humans to laboratory organisms using percentage increase in mean life expectancy at birth (or from eclosion in the case of insects) simply because this is most readily attainable from the literature on laboratory organisms.

Fruit fly selection experiments achieve significant extensions in life span by rearing successive generations from eggs laid by old individuals. In one classic example, mean life span increased by about 30% in 15 generations (15), for a rate of change of almost 2% per generation, and in another by about 100% in 13 generations (16), or just over 5% per generation. For human hunter-gatherers, mean life span at birth is about 31 [ranging from 21 to 37 in several populations (3)]. For Swedes, it was about 32 in 1800, 52 in 1900, and is 82 today (17). So life expectancy increased by about 165% from hunter-gatherers to modern Swedes and at a rate of about 12% per generation since 1800.

Some of the most promising directions for understanding the physiological mechanisms of aging come from experiments with mutations that affect the endocrine pathway (18, 19). These impressive experiments have extended mean life span in nematode worms by >100% (19, 20), fruit flies by ∼85% (21), and laboratory mice by ∼50% (22). Dietary restriction, which involves suppressing caloric intake of an organism, has extended life span in nematodes by 100–200%, fruit flies by ∼100%, and mice by ∼50% (23). Hence recent human mortality improvement is often greater than that achieved by manipulated strains of model organisms relative to the wild type, especially when single mutations or physiological pathways are manipulated. However, experiments that simultaneously manipulate multiple pathways in organisms such as yeast and nematode worms can achieve much greater life span extensions (23, 24). The majority of laboratory studies where mammals are the model organism have been done on mice and yield percentage life span increases less than those gained by humans (23).

Discussion

Examining variation in mortality profiles from a large-scale comparative perspective provides insight into the general phenomena of aging and the shapes of age-specific mortality patterns. From these basic comparisons, we emphasize three findings. First, we find that mortality improvement has been very rapid and very recent, experienced by only the last four generations of humans that have ever lived (and primarily by those living in wealthy industrialized nations). Before the late 1800s, even humans in the lowest-mortality nations were not experiencing mortality much lower than was typical during most of human evolution. Second, today's lowest-mortality nations are further from the human pattern of hunter-gatherers than hunter-gatherers are from chimpanzees. That we observe more variation among human populations than we see between species is particularly surprising given how much better hunter-gatherers survive than even captive chimpanzees (10). Third, we find that human progress in lowering mortality is on par with or exceeds that made in the laboratory via various selection and dietary restriction experiments and endocrine pathway mutations.

Life span improvement in laboratory organisms and humans has been achieved via entirely different channels. Laboratory experiments on model organisms often manipulate mutations that affect specific physiological pathways that are relatively well understood (18, 25). Generally, these life-extending pathways derive from adaptations that divert energy from growth to maintenance at the cellular level (19). Mutations on these pathways can lead to dramatic increases in life span (23). In contrast, the dramatic rise in life span for humans is more complicated and less well understood, but was perhaps almost entirely due to environmental improvements (26); the increase in Sweden from 1900 to today was almost certainly not due to genetic change. Some experiments increase model organism life span by caloric restriction, which is a type of environmental change, but the human gains are generally greater than those achieved by such experiments. It might be hypothesized that some fraction of human mortality extension comes from selection for late breeders successively mating together because resource-rich environments correlate with later age at first birth and longer life span (27). However, given the speed of the recent mortality revolution and the fact that it is similar in widely separate nations (e.g., Fig. 2), it seems highly unlikely that genetic change has played more than a minor role.

In short, many of the manipulations that make round worms and fruit flies live longer serve to build a more shock-resistant, or "better," organism. Human mortality increase has been largely achieved by removing environmental shocks, by making injuries

and illnesses less fatal with medical technology, and by enhancing health at older ages by improving nutrition and reducing disease at younger ages, rather than by shifting the distribution of genotypes or changing fundamental physiological processes to make the organism more resistant to difficult circumstances. Reductions in human mortality come from many external sources, including improved standards of living, education, public health, sanitation, medicine, housing, and nutrition (28, 29). These can be thought of as building better environments.

The reductions in human mortality over the past century are biologically unique. In particular, the rate of mortality reduction, its occurrence at all ages, and its progressive continued reduction with time are features that probably only human populations in wealthy countries have experienced. Certainly the reductions in mortality are closely linked to the phenomenal rises in access to resources also typical of low-mortality nations (27). However, although no other organism has likely experienced the type or magnitude of environmental improvement that humans have, there is no a priori reason to suspect that other species are not capable of similar or greater levels of plasticity. Although the human life history has many unique features (30), our underlying biological potential for plasticity in mortality is likely to be shared with other taxa. That said, there are likely taxonomic differences in the shapes of mortality profiles (31), the age at lowest mortality, and the lowest mortality level (if any) that a species can achieve. Research is needed on identifying what those taxonomic signatures may be.

The environmentally driven plasticity documented here raises questions about evolutionary theories of aging. Theory suggests that fitness is most sensitive to change in mortality around the age of sexual maturity (32). At this age, selection pressure is highest and should have pushed mortality at maturity to its lowest possible level. When transfers of resources between kin are an important factor, as in humans (33), the force of selection against individuals at the onset of reproduction should be particularly strong (34, 35). However, the lowest mortality level achieved by evolution, as revealed by the experience of hunter-gatherers, was magnitudes above the >200-fold lower levels recently achieved in Japan, Sweden, and other postindustrial societies. Evolutionary theories of aging posit that the human genome carries a burden of mutations that are deleterious at older ages and neutral or perhaps beneficial at younger ages (32, 36). In fact, the lowest mortality achieved in the evolutionarily typical human experience is now experienced by people in their 70s and the annual chance of death of a 70-y-old Swede today is less than a 10th of the chance for hunter-gatherers.

The prediction that late-life mortality is determined by deleterious mutations that progressively accumulate as the force of selection declines with age is difficult to reconcile with the finding of exceptional environmentally driven malleability in mortality at all ages. What is the underlying explanation for this extraordinary plasticity? Why does the human genome give humans a license to drastically reduce mortality by nongenetic change? Are other species capable of comparable levels of plasticity? For how long will life expectancy continue to rise and by what means? Placing the record of human mortality reduction in broad evolutionary context reveals that we have much to learn about the evolution of mortality profiles and the processes of aging. Furthermore, fundamental research on the determinants of age-specific mortality patterns and on the malleability of these patterns by environmental as well as genetic change is needed, not only for humans but also for other primates and for species across the tree of life to put human mortality patterns into biological perspective.

Materials and Methods

The national data for Sweden, France, and Japan come from the Human Mortality Database (17). The estimates for hunter-gatherer, acculturated hunter-gatherer, wild chimpanzee, and captive chimpanzee populations are from Gurven and Kaplan (3): they fit Siler functions to ethnographically observed mortality data. We use these Siler fits to generate estimates of the force of mortality at various ages. We convert the age-specific force of mortality $\mu(x)$ to the age-specific probability of death $q(x)$ by $q(x) = 1 - \exp(-\mu(x + 0.5))$. The hunter-gatherer and acculturated hunter-gatherer curves are each aggregations of multiple groups. The hunter-gatherer groups included in the Siler fit are the Hadza, Ache Forest, Hiwi, !Kung, and Agta. The acculturated hunter-gatherer groups are the !Kung from 1963 to 1974, the Reservation Ache, Northern Territory Aborigines, Hiwi post-1960, Agta transitional, and Agta peasant (see ref. 3 for details). The demographic data on Trinidadian slaves are from John (37) and were also analyzed by Levitis and Lackey (38).

Equivalent age was calculated using the "approx" function in R with the syntax: approx([hunter-gatherer qx],[vector of ages for interpolation], [qx data for reference population]), where qx is the annual probability of death for the population.

ACKNOWLEDGMENTS. We thank Adam Lenart, Daniel Levitis, John DeLong, Zhang Zhen, Jim Oeppen, Susan Alberts, and Samuel Preston for helpful comments on the manuscript. Funding was provided by the Max Planck International Research Network on Aging (MaxNetAging), the Max Planck Institute for Demographic Research in Rostock, Germany, and the US National Institute on Aging (P30-AG034424).

1. Wachter KW, Finch CE, eds; Committee on Population, National Research Council (1997) *Between Zeus and the Salmon: The Biodemography of Longevity* (National Academy Press, Washington).
2. Bronikowski AM, et al. (2011) Aging in the natural world: Comparative data reveal similar mortality patterns across primates. *Science* 331:1325–1328.
3. Gurven M, Kaplan H (2007) Longevity among hunter-gatherers: A cross-cultural examination. *Popul Dev Rev* 33:321–365.
4. Oeppen J, Vaupel JW (2002) Demography. Broken limits to life expectancy. *Science* 296:1029–1031.
5. Tuljapurkar S, Li N, Boe C (2000) A universal pattern of mortality decline in the G7 countries. *Nature* 405:789–792.
6. Oeppen J (2006) Life expectancy convergence among nations since 1820: Separating the effects of technology and income. *Perspectives on Mortality Forecasting. III. The Linear Rise in Life Expectancy: History and Prospects. Social Insurance Studies, No. 3*, ed Bengtsson T (Swedish Social Insurance Agency, Stockholm), pp 55–82.
7. Brayne C (2007) The elephant in the room—healthy brains in later life, epidemiology and public health. *Nat Rev Neurosci* 8:233–239.
8. Lee RD (2007) *Global Population Aging and Its Economic Consequences* (AEI Press, Washington).
9. Edeline E, et al. (2007) Trait changes in a harvested population are driven by a dynamic tug-of-war between natural and harvest selection. *Proc Natl Acad Sci USA* 104:15799–15804.
10. Hill K, et al. (2001) Mortality rates among wild chimpanzees. *J Hum Evol* 40:437–450.
11. Baudisch A (2008) *Inevitable aging? Contributions to evolutionary-demographic theory* (Springer, Berlin).
12. Sanderson WC, Scherbov S (2005) Average remaining lifetimes can increase as human populations age. *Nature* 435:811–813.
13. Shultz GP, Shoven JB (2008) *Putting Our House in Order: A Guide to Social Security and Health Care Reform* (W. W. Norton & Company, New York).
14. Perelman P, et al. (2011) A molecular phylogeny of living primates. *PLoS Genet* 7: e1001342.
15. Rose MR (1984) Laboratory evolution of postponed senescence in *Drosophila melanogaster*. *Evolution* 38:1004–1010.
16. Luckinbill LS, Arking R, Clare MJ, Cirocco WC, Buck SA (1984) Selection for delayed senescence in *Drosophila melanogaster*. *Evolution* 38:996–1003.
17. Human Mortality Database. Available at www.mortality.org/. Accessed January 9, 2012.
18. Tatar M, Bartke A, Antebi A (2003) The endocrine regulation of aging by insulin-like signals. *Science* 299:1346–1351.
19. Kenyon C (2005) The plasticity of aging: Insights from long-lived mutants. *Cell* 120: 449–460.
20. Kenyon C, Chang J, Gensch E, Rudner A, Tabtiang R (1993) A C. elegans mutant that lives twice as long as wild type. *Nature* 366:461–464.
21. Tatar M, et al. (2001) A mutant *Drosophila* insulin receptor homolog that extends life-span and impairs neuroendocrine function. *Science* 292:107–110.
22. Coschigano KT, et al. (2003) Deletion, but not antagonism, of the mouse growth hormone receptor results in severely decreased body weights, insulin, and insulin-like growth factor I levels and increased life span. *Endocrinology* 144:3799–3810.
23. Fontana L, Partridge L, Longo VD (2010) Extending healthy life span—from yeast to humans. *Science* 328:321–326.
24. Hekimi S, Burgess J, Bussière F, Meng Y, Bénard C (2001) Genetics of lifespan in *C. elegans*: Molecular diversity, physiological complexity, mechanistic simplicity. *Trends Genet* 17:712–718.
25. Kenyon CJ (2010) The genetics of ageing. *Nature* 464:504–512.
26. Vaupel JW (2010) Biodemography of human ageing. *Nature* 464:536–542.
27. Burger O, DeLong JP, Hamilton MJ (2011) Industrial energy use and the human life history. *Sci Rep* 1:56.

28. Riley JC (2001) *Rising Life Expectancy: A Global History* (Cambridge Univ Press, Cambridge, UK).
29. Schofield R, Reher D, Bideau A (1991) *The decline of mortality in Europe* (Oxford Univ Press, Oxford).
30. Hawkes K (2006) *The Evolution of Human Life History*, eds Hawkes K, Paine R (School of American Research Press, Santa Fe, NM), pp 95–126.
31. Baudisch A (2011) The pace and shape of ageing. *Methods Ecol Evol* 2:375–382.
32. Hamilton WD (1966) The moulding of senescence by natural selection. *J Theor Biol* 12: 12–45.
33. Gurven M, Stieglitz J, Hooper PL, Gomes C, Kaplan H (2012) From the womb to the tomb: The role of transfers in shaping the evolved human life history. *Exp Gerontol* 47:807–813.
34. Lee RD (2003) Rethinking the evolutionary theory of aging: Transfers, not births, shape senescence in social species. *Proc Natl Acad Sci USA* 100:9637–9642.
35. Cyrus Chu CY, Lee RD (2006) The co-evolution of intergenerational transfers and longevity: An optimal life history approach. *Theor Popul Biol* 69:193–201.
36. Medawar PB (1952) *An Unsolved Problem of Biology* (H. K. Lewis & Company, London).
37. John AM (1988) *The Plantation Slaves of Trinidad, 1783–1816: A Mathematical and Demographic Enquiry* (Cambridge Univ Press, Cambridge, UK).
38. Levitis DA, Lackey LB (2011) A measure for describing and comparing post-reproductive lifespan as a population trait. *Methods Ecol Evol* 2:446–453.
39. Ministry of Health Labour and Welfare. Available at: www.mhlw.go.jp/english/database/db-hw/vs02.html. Accessed April 25, 2012.