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Predicting Human Longevity

IN "PROSPECTS FOR HUMAN LONGEVITY," S. J. Olshansky and co-authors note that if the rate of mortality decline observed in France between 1985 and 1995 were to continue, life expectancy would reach 85 years by 2033 (average of male and female) and similarly in Japan by 2035 (*Science*'s Compass, Policy Forum, 23 Feb., p. 1491). They suggest that this represents an upper limit to the possibilities, unless scientists discover how to modify the aging process. Their corresponding calculation for the United States indicates that 85 would not be reached until 2182, a century and a half later. These long-run projections should not be based on such a short (10-year) observation period (*1*). For example, had they used the same method, but analyzed data for the most recently available decade starting just 3 years later, 1988 to 1998 (instead of 1985 to 1995), the United States would reach 85 years in 2052 rather than 2182, earlier by 130 years; extrapolation from 20 years, 1978 to 1998, gives a date of 2060. Life expectancy increased by 0.7 to 0.9 year (depending on data source) in just 3 years between 1995 and 1998, compared with a gain of 1.1 years over the entire decade 1985 to 1995. A more systematic approach to forecasting based on longer historical trends and more age detail suggests that a life expectancy of 85 would be reached in 2065, with a 95% probability range between 2043 and 2114 (*1*), indicating the high degree of uncertainty.

Ready or not, a long life awaits.

Analysts have repeatedly thought that death rates were approaching biological limits and could not fall much farther, only to be proved wrong by subsequent experience. The authors of the Policy Forum worry that continued decline at the long-run historical rates would reduce the death rates at ages below 30 to biologically implausible levels, and so constrains the infant mortality rate not to fall below 5 per 1000. However, 12 countries already report infant mortality below this threshold, with Iceland reporting 2.6. In any event, mortality below age 30 will have little effect on future life expectancy because it is already so low. Thus, infant mortality levels illustrate the perils in arguing that death rates at any age are near natural limits.

Olshansky and colleagues note that the Technical Advisory Panel recommendation that the Social Security actuaries raise their life expectancy forecast for 2075 by 3.7 years would require that death rates at each age decline twice as fast as "the already favorable rate of mortality decline projected by the [U.S. Social Security Administration]." This "favorable rate" projected by the actuaries, however, is only half the historical rate of decline (*1*). In fact, mortality decline at the historical rate would lead to life expectancy in 2075 that is higher by 3.7 years, as recommended by the panel. None of this is due to "ignoring the phenomenon of entropy in the life table," as the authors suggest. However, they are right that even this apparently modest increase in the life expectancy projected for 2075 requires continuing dramatic biomedical advances, which are implicitly assumed.

At several points, the authors qualify their predictions with phrases like "unless scientists can discover how to modify the aging process." Over the past century, science has made regular progress against disease and death, and given the dramatic biomedical advances for humans and other organisms in recent years, it would be risky to bet the long-term finances of the Social Security system on the assumption that this will cease. It is most prudent to assume that mortality will continue to decline on trend.

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Response

WE AGREE THAT THERE IS CONSIDERABLE uncertainty associated with demographic projections that extend far into the future, especially when those projections are based on an anticipated continuation of past mor-

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tality trends. In fact, we contend that future gains in life expectancy cannot possibly match those of the past, because they were achieved primarily by saving the lives of infants and children—something that happens only once for a population.

Substantial gains in life expectancy will now require large reductions in death rates at middle and older ages. Since these sorts of declines are a relatively new phenomenon, the use of recent time frames for projections is not only warranted, but necessary. Because our latest projections were based on the most recent data available at the time (through 1995), it would have been impossible to implement Lee's suggestion to shift the time frame to include data through 1998. As for the observed gain in life expectancy between 1995 and 1998 in the United States, the suggestion by Lee that this is somehow important appears inconsistent with his view presented earlier that changes in life expectancy observed over short time periods are of little interest.

Nevertheless, we agree with his observation that these projections are highly sensitive to the time frame chosen. This is one reason why we urge caution when interpreting confidence intervals for projections of life expectancy. Such projections can be misleading because they are based on the premise that the future will resemble the past—an assumption that is untenable in a developed world where external threats like infectious diseases that predominantly kill the young have been largely replaced by aging-related causes of death that strike the older members of a population.

In our opinion, given the important economic implications associated with official government forecasts of death rates and life expectancy, two sets of projections are warranted. The first set should involve short-term (for example, 20-year) forecasts with a parsimonious and proven approach like that presented by Lee and Carter (*1*), and the second set should be based on a broader range of possible changes in life expectancy that encompasses both more optimistic and more pessimistic scenarios for the remaining projection time frame (usually 50 years) than those that are currently used. By updating them often, projections of life expectancy can be obtained that are not only more realistic but also more sensitive to the rapidly changing social, biological, and biomedical forces that influence the life-span of individuals and the life expectancy of populations.

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Human Origins and Ancient Human DNA

IN HER ARTICLE "OLDEST HUMAN DNA reveals Aussie oddity" (News of the Week, 12 Jan., p. 230), Constance Holden overlooks several problems with the challenge to the "Out of Africa" theory of modern human origins posed by putative ancient Australian human mitochondrial DNA (mtDNA) sequences (*1*) . Ancient DNA discoveries are easily contaminated (*2*) and carry a considerable burden of proof, especially when they involve human sequences or surprising examples of preservation. Both concerns apply in the case of the ancient Australian remains (up to 60,000 years old) analyzed by Adcock and colleagues (*1*), because DNA is not expected to survive for this length of time outside of cold environments (*3*) and similar remains elsewhere have not yielded genetic material (*4*).

Journals continue to report studies in which standard ancient DNA authentication criteria have not been used, such as independent replication by other laboratories, biochemical studies of bone preservation, and cloning of DNA sequences (to reveal damage-caused amplification artefacts). Without such data, it is impossible to rule out the possibility that the ancient Australian mtDNA sequences such as Lake Mungo 3 (LM3) and Kow Swamp 8 (KS8) result from modern human contamination of the bone during handling over the years, complicated by DNA damage. DNA sequences from dinosaur bones were found to result from this process (*5*), and the high proportion of cytosine-thymidine transitions between LM3 and the reference sequence correspond well with the cytosine deamination common in damaged DNA (*6*).

Furthermore, analysis of the data does not support the interpretation of Adcock *et al.* that LM3 represents the most basal sequence found among modern humans, and that it diverged from the human nuclear insert on chromosome 11 (*5*) before the most recent common ancestor of modern humans. For example, LM3 and the human nuclear insert sequences differ by 13 substitutions (7) (Kimura 3-P distance = 0.0503), whereas LM3 is only 6 substitutions from LM15 and some modern sequences (for example, GenBank accession numbers AF236971, AF212406, B84892, AF228751, K3-P = 0.0264 to 0.0366). It seems implausible