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

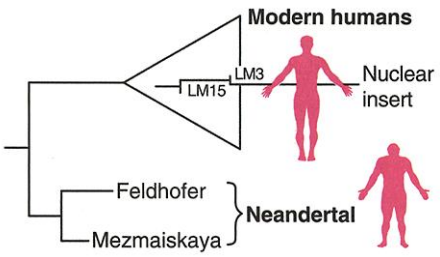
IN HER ARTICLE "OLDEST HUMAN DNA reveals Aussie oddity" (News of the Week, 12 Jan., p. 230), Constance Holden overlooks several problems with the challenge to the "Out of Africa" theory of modern human origins posed by putative ancient Australian human mitochondrial DNA (mtDNA) sequences (*1*) . Ancient 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

from these short branch lengths that LM3 diverged before the most recent common ancestor, because mtDNA has a higher substitution rate than nuclear DNA.

Such suggestions of homoplasy (random or systematic convergent evolution) are confirmed by phylogenetic analyses (*8*) using the same model as Adcock *et al.*, but with additional modern Aboriginal and African sequences (see the figure). These trees show that LM3 and KS8 are well within modern human variation; the nuclear insert is probably attracted to LM3 due to homoplasy. This phylogenetic position is also obtained when Adcock *et al.*'s original limited set of sequences is used if a model of heterogeneity of rate between sites is incorporated (*9*).



The roots of human origins. This simplified phylogenetic tree was obtained by using the same sequences and substitution model as Adcock *et al.* (*1*) with additional modern human sequences from Australia (*10*) and Africa (*11*).

Lastly, even if the problems with both the data and the analysis were ignored, the phylogenetic tree of Adcock *et al.* would not support the "multiregional model" for modern human origins, because all the modern human sequences are closely related to each other, whereas the Neandertal sequences form an outgroup. Consequently, to see the data of Adcock *et al.* as a significant problem for the Out of Africa model seems an exaggerated claim.

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7. Table 1 of Adcock *et al.* contains an error at site 199 suggesting that LM3 and the human nuclear insert differ by 14 substitutions.
8. Maximum likelihood trees were constructed using PAUP* 4.0b4 (Sinauer, Sunderland, MA). A heuristic search was performed by using the HKY85 model of substitution and a transition-transversion ratio of 18. The complete phylogeny is available at http://evolve.zoo.ox.ac.uk/data/Mungo/. A complete version of the phylogenetic tree represented in the figure is available to *Science* Online subscribers at http://www.sciencemag.org/cgi/content/full/292/5522/1655/DC1
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Response

COOPER AND HIS CO-AUTHORS SUGGEST THAT DNA is unlikely to have been preserved in the ancient Australians we studied. We do not know which environments will preserve DNA for 60 or 60,000 years, just as we do not know why there are fossil remains from some regions but not others. All our bone samples (*1*) were coated with thick carbonate crusts when they were excavated. The LM3 burial lay largely within a carbonate-rich horizon in the Mungo dune, "one of the best locations for the preservation of bone" (*2*). A relatively rapid encrustation of the bones might have produced conditions favoring preservation of the bones and any DNA they contained.

Our procedures were at least as stringent as the "standard" ancient DNA authentication tests cited by Cooper *et al.* None of the samples had been handled by either Aboriginal or non-Aboriginal people before extractions began. We took internal samples under sterile conditions. Our paper details the care taken to replicate and confirm results. Because cloning can cause polymerase chain reaction (PCR) artifacts, we sequenced amplification products directly. For each of our 10 ancient bone samples, a unique DNA sequence was consistently obtained from the independent isolations and PCR amplifications. In the initial Neandertal report (*3*), independent sequence results were not achieved. Only contaminant sequences were obtained in the second laboratory until primers, based on the Neandertal sequence from the first laboratory, were used to amplify a small portion (about 10%) of the mtDNA segment studied. This is not an independent replication. If our results were compromised by the occurrence of deamination, as Cooper *et al.* suggest, we would have expected sequence differences among the independent DNA isolations and PCR amplifications from each bone sample. We did not find any such heterogeneity.

We agree that the exact branching position of the lineage leading to LM3 and the nuclear insert sequence cannot be reliably estimated from any of the extensive phylogenetic analyses we conducted. Nonetheless, we are confident of the grouping of LM3 with the insert sequence. This is overwhelmingly indicated in all our analyses, particularly the likelihood mapping. The grouping of the LM3 and nuclear insert sequences is unlikely to be due to a long branch attraction effect because the branch leading to LM3 is very short and much shorter than branches leading to the many other sequences we analyzed. The relatively long branch leading to the insert sequence makes it highly unlikely that this sequence, and hence the LM3 sequence, diverged after the most recent common ancestor of the sequences in living humans (*4*).

We did not claim to have disproved the entire recent "Out of Africa" model. We suggested that mitochondrial sequence data from ancient human samples have to be considered in any model of human origins and that it is not sufficient to base a theory solely on data from extant populations. The significance of our study is that we have isolated ancient mtDNA sequences, including one that is 60,000 years old, from undisputed Australian modern humans. The fact that this LM3 sequence belongs to a lineage related to the nuclear insert and is now extinct suggests there may have been many mitochondrial lineages in Pleistocene populations of anatomically modern humans.

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CORRECTIONS AND CLARIFICATIONS

NEWS FOCUS: "New data in chemistry show 'zero' diversity" by Jeffrey Mervis (18 May, p. 1291). The chair of the division of chemistry at Harvard University was misidentified. His name is James Anderson.

REPORTS: "Presynaptic kainate receptor mediation of frequency facilitation at hippocampal mossy fiber synapses" by D. Schmitz, J. Mello, R. A. Nicoll (9 Mar., p. 1972). The electrophysiological traces in the report contained sharp transients and steps that were not present in the original data. The conclusions of the paper are not affected. The corrected figures can be viewed in full text version of the paper in *Science* Online.