Evolution east of Eden

SIR—"No names, no pack drill"[1]. As in military service, in science criticism is safe only if nonspecific; the converse also holds: citation of alternative hypotheses must be accurate. The paper by Cann *et al.*[2] on mitochondrial DNA and human evolution misses in this regard, thereby blemishing the presentation of a methodology that otherwise holds every promise of providing an extremely valuable source of information on hominid evolution.

The authors' conclusion from restriction mapping is that all mitochondrial DNAs stem from an African woman who lived about 200,000 years ago, whereas other geographic areas were colonized more recently. They also outline an alternative view, that *Homo* has been present in Asia as well as Africa for at least one million years and that the transformation of archaic to anatomically modern humans occurred in parallel in different parts of the world.

Unfortunately, the reference[3] which they cite on *Homo* being in Asia for at least one million years says something rather different, beginning "A number of separate lines of evidence indicate that all Asian hominids are *less* than one million years old [emphasis mine]", and concluding "...the entire record of *Homo erectus* in Asia may only span a period of approximately 600 thousand years (that is from 0.9–0.3 Mya)."

What of the references[4,5] that Cann *et al.* cite supporting the idea that the transformation of archaic to anatomically modern humans occurred in parallel in different parts of the world? Coon[4] did hold such a view, but Wolpoff *et al.*[5] actually present a "multiregional evolution hypothesis" in which different hominid gene pools were established during initial occupation. Afterwards, "the pattern of regional variation was maintained throughout most of the Pleistocene by a balance between the local forces promoting homogeneity and regional distinction (selection, drift) and multidirectional gene flow". This evolutionary pattern is neither merely parallel nor traceable to a single centre save in the sense that all hominid lineages trace to a single source sometime in the past.

There are real uncertainties in estimates of rates of mitochondrial DNA evolution[6], and influences of stochastic processes from population size and structure should be considered[7]. Until then there is insufficient cause to discard the first half million years or so of Asia's hominid-fossil record, particularly as at least some of the skeletal characters[8–10] that are said by Cann *et al.* to make it "unlikely that Asian *erectus* was also ancestral to *Homo sapiens*" also occur in fossil hominid material from Europe (Arago, Petralona) and Africa (Bodo). Of greater weight on the side of hominid evolutionary continuity in Asia back beyond 200,000 years ago is that other skeletal and dental traits marking living Asian populations (frontal keel, torus mandibularis, os epactale, shovel-shaped central and lateral upper incisors) also occurred in fossil remains sampled from populations dated to several hundred thousand years before the supposed African replacement 200,000 years ago[11]. To have these features disappear via extinction and evolve anew in populations fresh from Africa would merely require parallel evolution of a different sort.

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Of how great significance?

SIR—Bazan *et al.*[1] deny the significance of the similarities between the nucleotide sequences that code for the scrapie-associated PrP27–30 protein and human immunodeficiency virus (HIV) that we recently reported[2]. They argue that other regions of HIV and cellular genes, such as the rat cytochrome P450c, show similarities that are as, or more, significant. But these similarities comprise unique regions of similarity, with an extensive number of gaps, whereas ours comprise four consecutive regions of similarity. Bazan *et al.* omit the fact that the probability of btaining the four reported similarities between PrP27-30 and HIV by chance is less than 1 in 1,000 while that of obtaining those with cytochrome P450c or the other HIV regions is about 1 in 100 (ref. 3). It is worth noting that Bazan *et al.* use one algorithm, ALIGN, to compare the PrP27–30 sequence with HIV and another, FASTN, to compare PrP27–30 with other regions of HIV and other products. The scores, as well as the mean and standard deviation of their distributions, appear not to be comparable.

Braun and Gonda[4] deny the significance of the similarities between the amino-acid sequences of PrP27–30 and the carboxyl terminus of HIV. Unfortunately, these authors misinterpreted or obviated the basis of our report. Their arguments are based solely on protein sequence comparisons and fail to consider the significance of the similarities that exist at the nucleotide sequence level. Notwithstanding this omission, the *P* value for the global alignment between the scrapie-associated PrP27–30 protein and the carboxyl-end of the HIV endonuclease is 0.1 which is in fact significant, although marginally.

Both Bazan *et al.* and Braun and Gonda argue against the significance of the alignment between PrP27–30 and HIV by grouping HIV together with visna virus and equine infectious anaemia virus (EIAV) and showing that the similarity between visna and PrP27–30 or EIAV and PrP27–30 are lower at both the nucleotide and amino-acid sequence levels. This, however, does not deny a possible common evolutionary origin for all these sequences. Even if HIV, visna and EIAV belong to the same group of viruses, it is not expected that they will undergo similar evolutionary rates.

We appreciate the pinpointing of the human errors we incurred in our report during the transcription of the sequences in the final formatting of the figures. Nevertheless, none of these errors affect the significance of the comparisons or the statistical values thereof.

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Alzheimer amyloid aspects

SIR—Kang *et al.* (*Nature* **325**, 733–736; 1987) suggest that the major protein subunit (A4) of the amyloid fibril of tangles and plaques of Alzheimer's disease is derived by inappropriate processing from a 695 amino-acid precursor polypeptide. As an alternative, we would like to suggest that there is inappropriate initiation of translation at methionine 596, which immediately precedes the A4 sequence.

Kozak (*Cell* **44**, 293–292; 1986) has described an optimal sequence for initiation of translation by eukaryotic ribosomes as ACCATGG, where at position