that the geography of β-globin variants is in itself compatible with an African, an Asian or a dual origin of mankind, but the fossil record does seem to indicate an African birthplace for modern humans. As geneticists fallen among palaeontologists, we are not qualified to comment on the controversy as to the dating of African fossils, but have accepted the consensus view and used this (and not the β-globin tree) to suggest an African origin for mankind. Our deduction of a bottleneck at the origin of the rest of the world's human population is the simplest model consistent with the fossil data and the geography of the β-globins. Simplicity has forced us to make a number of assumptions.

One of these is that β-globin variants are selectively neutral. Nothing would delight us more than to learn that the possession of alternative haplotypes altered their carriers' ability to escape from sabre-toothed tigers, but in the absence of evidence to the contrary we have accepted the view, implicit in most theories of molecular evolution, that natural selection does not act on noncoding sequences of DNA. As both your correspondents point out, this is not necessarily correct; and there certainly exist quite feasible modes of selection on noncoding variants by virtue of their linkage to strongly selected alleles such as that for sickle cell haemoglobin[7]. As there is simply no information on this possibility, we have, with Occam, chosen the more straightforward alternative. The model also includes the arbitrary, but simple, suggestion that the four commonest modern haplotypes were at equal frequencies in the ancestral African population. This assumption is not central to the existence of a bottleneck, but does influence its size. The frequencies in modern African populations pointed out by Giles and Ambrose[1] could either reflect a conservation of their ancient values — suggesting an even smaller bottleneck in the emergent population — or a change within Africa arising from genetic drift in small populations (which could be many times larger than our supposed bottleneck of emigrants).

Despite the widespread popularity among palaeontologists of models of a multiple origin of mankind (for example ref. 7 of Van Valen), theoretical population genetics suggests that any population's ability to undergo speciation is so limited that it is extremely unlikely that *H. sapiens* arose simultaneously in different parts of the world from different ancestors. For example, using a simple model of change by genetic drift, Sewall Wright showed in the 1940s[6] that the probability of a chromosome rearrangement with a selective disadvantage to hybrids of 5% becoming established in a local population of 200 individuals is about 10^{-5} per generation. The chance of this happening simultaneously in two separate populations is about 10^{-10} per generation, which is extremely small, but may still be greater than the chance of simultaneous speciation in two separate populations. Van Valen's alternative suggestion — that of simultaneous global appearance of *H. sapiens* — implies extensive gene flow among its evolving populations, so extensive that both the ancestral *H. erectus* and the derived *H. sapiens* must be thought of as effectively sympatric. Sympatric speciation is itself difficult to accommodate into population genetics theory, and in Van Valen's scheme one is forced to explain the observed patterns of β-globin divergence by postulating long periods when large African and Eurasian populations of mankind were isolated from each other.

Our model, like many others, exists simply to show the evolutionary implications of an observed genetic structure. Its main point is to show that some of the demographic consequences of human gene frequency change are quite startling; startling enough, perhaps, to lead us to try to formulate a better model. A successful model would also, as we point out, have to explain why different genes seem to imply different demographic patterns during the spread of mankind.

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Transfer of radioiodide to milk and its inhibition

SIR—Following the increased atmospheric levels of radioactivity resulting from the Chernobyl reactor accident[1], one of the main radiological concerns has been the entry of radioiodide into the human food chain through animal milk, and its subsequent uptake into the thyroid gland, as reported recently in your columns[2].

Current studies of transport mechanisms for various radionuclides in the mammary gland emphasize the rapidity with which blood-borne radioiodide can enter milk. The mammary gland of two goats was continuously infused for 15 min with ^{123}I (2.78 MBq; sodium iodide, AERE Harwell) through an indwelling polyvinyl chloride catheter placed in the external pudic (mammary) artery. Milk was completely removed by hand at 15 min intervals with oxytocin treatment (100 mUnits intravenously) and total radioactivity measured in a LKB Wallac 80000 gamma-sample counter. The total ^{123}I activity secreted into the milk rose rapidly to a peak concentration just 30 min after the start of infusion followed by a slower decrease. The fractional activity of ^{123}I secreted in milk during a 2 h period was 1.0 and 1.5% of the infused activities.

The iodide concentrating activity of the mammary gland has long been recognized[3,4], and earlier work showed that the secretion of radioiodide into milk could be inhibited by competing anions such as perchlorate and thiocynate[3,5]. We repeated the above experiment by infusing sodium perchlorate (100 mg) close-arterially for 30 min before and during ^{123}I infusion. The total fractional ^{123}I activity transferred into milk during 2 h was reduced by 60–66%.

Perchlorate has previously been found to be a more efficient inhibitor of radioiodide transfer into milk than thiocyanate, iodide or iodate[3]. Thiocyanate was not concentrated in rabbit milk[3], but we are unaware of any data that quantifies perchlorate transfer into milk. This information is urgently needed before perchlorate administration could be considered as a means of protecting against the entry of radioiodide into the human food chain[6] or into breast-fed infants after a reactor accident[7]. The use of perchlorate is not unprecedented since the compound has been used without deleterious effects in nuclear medicine to block, for example, the uptake of radionuclides into the maternal thyroid during diagnostic investigations[8]. There is also a clear need to identify inhibitors which block mammary transport of other radionuclides, including fission products.

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Scientific Correspondence

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