Evolution of osteoporosis

Professor Purdie's article on the influence of ethnicity on osteoporosis was fascinating. However, we cannot support the argument that evolution has determined racial differences in bone mass and thus a low prevalence of osteoporosis in certain racial groups.

As the article points out, there are differences in bone mass and fracture incidence between white and black women in North America. The data on male fracture incidence are more contentious and the references cited relate to studies of black residents of mental institutions and hip fractures in a small population in Charlottesville, USA, with an incidence of fewer than 25 fractures per annum. However, Farmer reports no difference in fracture incidence in North America between black men, black women, or white men. In Africa, many women have multiple pregnancies, prolonged lactation periods, and low dietary calcium intakes. Despite these factors, fracture incidence is comparatively low in both sexes from black African communities. Vitamin D receptor alleles may offer some insights into geographical variation in osteoporosis. Recent results from The Gambia have shown a higher prevalence of the potentially protective BB genotype than in white women or black women from Boston, USA.

A strong argument against an evolutionary aetiology for advantageous bone mineral density (BMD) in black populations is that the incidence of fractures is low due to some other factor or factors, as yet unknown.

TERENCE J ASPRAY
ROGER M FRANCIS
Musculoskeletal unit, Freeman Hospital, Newcastle upon Tyne NE7 7DN

ANN PRENTICE
MRC Dunn Nutrition Unit, Cambridge CB4 1XJ


Author's reply

Teresence Aspray and colleagues make helpful observations on the complex problem of racial differences in bone fracture rates. Indeed, it is likely that the observed differences between black and white populations are due to a raft of factors of which mineral density and gross bone geometry are but two. The most interesting data cited by Aspray et al from the Gambia do not necessarily confound the hypothesis of an evolutionary component to Caucasian osteoporosis. We would not accept that frequent pregnancy and lactation risk-matched the African, since the overall net effect of the reproductive cycle upon bone mass is broadly neutral, with Caucasian–Bantu differences negligible, except for pregnancy number. The Gambian subjects studied in this area of Western Africa may represent a group in whom a lower BMD has evolved, following the migration of H sapiens from Africa, and perhaps also subsequent to the enforced migration of the West African ancestors of present day Afro-Americans, who, as is correctly pointed out, show a higher BMD. There remains a shortage of statistical and biological data from the evolution of the skeleton. Finally, the report by Baron et al has indicated that, in N America, age adjusted female rates of femoral neck fracture per 105 population were 214 for blacks and 968 for whites. If this difference is not all environmental and, if it is accepted that Africa is the homeland, then a degree of evolutionary disadvantage for whites remains likely. The original hypothesis proposed an evolutionary link between bone resilience and human geohistory. Like all hypotheses, it sought to explain observed phenomena through a synthesis of available data. Like all hypotheses, it awaits rigorous trial.

D W PURDIE
Centre for Metabolic Bone Disease,
The University of Hull