Insulin resistance, high prevalence of diabetes, and cardiovascular risk in immigrant Asians

Sin.—We read with interest the findings of Dhawan et al., who highlighted the importance of hyperinsulinaemia, central obesity, and physical inactivity as risk factors in Asians with angiographically significant coronary artery disease. Both British and Indian Asians were found to share a predisposition to insulin resistance and its associated metabolic abnormalities—and hence a high cardiovascular risk. They conclude that this finding may help to improve the genetic risk rather than environmental basis for the recognised high mortality in this ethnic group. However, migrants are not a random sample of the original population, and their “selection” is likely to be determined by several health and socioeconomic factors, which are likely to influence their morbidity and mortality. If this environmental effect was a significant determinant of cardiovascular risk, one would not expect to see the high mortality from ischaemic heart disease (IHD) that has been recorded in Asians in South Africa, Trinidad, and Singapore, who emigrated over a century ago. We recently reported a survey in which we studied the cardiovascular risk factor profile of all Asian men admitted with acute myocardial infarction during 8 weeks to one of the city centre district general hospital in Birmingham, and to the San Fernando General Hospital, in Trinidad: 74 patients were studied (55 patients (mean SEM age 58-1 (1-4) in Trinidad (Trinidad group) and 19 in Birmingham (62-1 (2-6) (UK) Group) (table). We also found that mean systolic and diastolic blood pressures were higher in those with hypertension in the Trinidad group (146-6 (16-9)/93-4 (11-4) mm Hg than in the UK group (120-8 (25-4)/75-0 (13-4) mm Hg; P < 0.05). Though Asians in Trinidad have in many ways adapted to the lifestyle of the host population, this does not appear to have reduced their cardiovascular risk profile, because those admitted with acute myocardial infarction had, in fact, a greater prevalence of central obesity, smoking, and higher blood pressures than a similar group in England.

Central obesity and physical inactivity were common to both communities in England and Trinidad and their relation to insulin resistance may be particularly important in Asians with IHD. Our data support the hypothesis of a genetic predisposition to central obesity and diabetes in Asians that seems to have been retained by third generation Asian immigrants in Trinidad. This may explain the persistently high mortality from IHD in Asians.

1. TAS FABROOI GARETH BEEVERS GREGORY Y H LIP Department of Medicine, University of Birmingham, City Hospital, Dudley Road, Birmingham B18 7QH


1 Marmot MG, Adelstein AM, Bulusu L. Lessons from the study of immigrant mortality. Lancet 1984;i:1455-57. Indeed, as one would have expected the group with more deaths to have a better average quality of life for the reasons given, the possibility arises that quality of life actually diminished on enoximone treatment.

Apart from the baseline comparison, we are not able to interpret the Nottingham Health Profile (NHP) data at all. For example, the median physical mobility score in the enoximone group was 22 at one year, but there is nothing to compare this with. We are not told what the median baseline score was for the enoximone treated survivors: and the placebo treated patients no longer form an adequate control group (as they are no longer matched with the enoximone group). It would have been interesting to see the results on the other NHP dimensions.

There are ways round this problem, but none is fully satisfactory. We can assign all patients who died to the bottom half of the distribution of quality of life scores (for example, by giving them all the worst possible score). The median is then not biased for comparative purposes. However, if more than half the subjects die the median becomes uninterpretable (although the Mann-Whitney U test remains valid). Alternatively, end points can be dichoto-

ized into those alive with improved quality of life on one hand and those who died plus those who are alive with worse quality of life on the other. These can be analysed as proportions, with or without con
trol for statistical power. Finally, changes from baseline can be quoted for survivors only. These can be interpreted as uncontrolled observations (a so-called “before and after” study). So long as the questionnaire has reasonable test-retest reliability, a beneficial treatment effect is the most likely explanation for any improvement seen, although a placebo effect cannot be excluded.

ROWAN HARWOOD
Department of Public Health, Royal Free Hospital, London NW3 2PP

Responses of plasma concentrations of A type natriuretic peptide and B type natriuretic peptide to a placebo, an angiotensin-converting enzyme inhibitor, in patients with congestive heart failure

Sin.—Yoshimura and colleagues showed that the response of plasma brain natriuretic peptide (BNP) after the administration of the angiotensin-converting enzyme inhibitor, alacepril, occurred later, and lasted longer than the response of plasma atrial natriuretic peptide (ANP), and that the changes in pulmonary capillary wedge pressure did not correlate with plasma BNP. This lack of correlation may be the result of this difference in responses. It may be relevant to recall that BNP has a longer half life than ANP, and that the fall in BNP is bi-exponential, with a slow phase of about 20-7 minutes, as estimated by Nakao’s group.1 My own estimate of the short half life was 37 minutes.1 In addition, there may also be a qualitative difference in the synthesis and release of BNP. In