Correspondence

Selective migration by birthweight

Sir—Martyn et al suggest that selective migration by birthweight may confound epidemiological studies of early life experience and adult cardiovascular disease among migrants.1 They found that the mean birthweight of men born in the north of England (Preston and Sheffield) who subsequently left their county of birth was higher than that of men who remained. Higher birthweight, they argue, is associated with lower blood pressure in adult life.

In the British Regional Heart Study (BRHS), men who migrated from the north of England to the south had lower mean blood pressure than those who remained in the north.2 This finding among migrants in the BRHS points towards the adult environment exerting a greater influence on blood pressure in middle age than factors acting early in life.

Martyn et al suggest, however, that north-to-south migrants in the BRHS may have been of higher average birthweight than non-migrants, which could have accounted for their lower adult blood pressure. According to Martyn et al, the confounding effect of selective migration by birthweight renders the BRHS findings inconclusive so that they “tell us little about the relative importance of influences in early and later life.”

The issue of selective migration is of considerable importance. While migration provides epidemiologists with an excellent opportunity for disentangling the relative contribution of factors acting around the time of birth from those present in the adult environment, many studies have shown that migrants differ in a number of ways from non-migrants, particularly with regard to their health and social position.4

Migrants in the BRHS cohort proved to be no exception to this rule. Overall, the mean systolic and diastolic blood pressures of migrants in the BRHS were lower than those for non-migrants,5 as was the risk of a major ischaemic heart disease event after 6-5 years of follow up (table). Migrants were also more likely to be employed in non-manual occupations than non-migrants. Non-migrants comprised men who were born in their town of examination, while migrants (referred to as “internal migrants” in references 2 and 5) were born both in Great Britain, but not in the town where they enrolled for the study.

Since BRHS migrants differed from non-migrants, the analysis of blood pressure by geographic zone of birth and examination was confined to migrants only. In this analysis men who had moved within the same geographic zone (for example, from Sheffield to Preston, within the north of England) were compared with those who had moved between geographic zones (for example, from the north of England to the south). The confounding effect of selective migration was overcome by comparing migrants within a geographic zone with migrants between zones. Non-migrants were excluded from the analysis.

It is extremely unlikely, therefore, that selective migration could account for the BRHS findings that men born in the north of England who moved south had lower mean blood pressure than men who remained in the north. Of equal importance, BRHS men born in the south of England who moved north had higher mean blood pressure than men who remained in the south.6 According to Martyn et al, selective migration of high birthweight/low blood pressure men would dictate that migrants from the south to the north should also have lower blood pressure than men who stayed in the south. This was not the case.

Epidemiological investigations into the association between early life experience and subsequent cardiovascular risk will inevitably be subject to potential confounding.5 None-the-less, data from the BRHS have clearly shown that after controlling for the possible confounding effect of selective migration, the geographic zone of examination is a more important determinant of adult blood pressure than the geographic zone of birth. Factors acting in adult life appeared to exert a greater influence on regional differences in blood pressure than those acting early in life.

JONATHAN ELFORD
PETER WHINCUP
A G SHAPER
Department of Public Health and Primary Care, Royal Free Hospital School of Medicine, Rowland Hill Street, London NW3

Table
Mean systolic and diastolic blood pressure, and risk of a major ischaemic heart disease (IHD) event, 6-5 years of follow up in the British Regional Heart Study, by migration status

<table>
<thead>
<tr>
<th>Migration Status</th>
<th>Mean Systolic Blood Pressure</th>
<th>Mean Diastolic Blood Pressure</th>
<th>Risk of IHD Event</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-migrants</td>
<td>147.0 (0.4)</td>
<td>81.0 (0.2)</td>
<td>9.7</td>
</tr>
<tr>
<td>Migrants (n=141)</td>
<td>144.0 (0.3)</td>
<td>81.0 (0.2)</td>
<td>9.7</td>
</tr>
<tr>
<td>All men (n=2739)</td>
<td>145.2 (0.2)</td>
<td>81.0 (0.2)</td>
<td>9.7</td>
</tr>
</tbody>
</table>
| * Includes 422 born outside Great Britain and 22 men for whom data on migration status was not available.

Mortality among drug injectors and notified addicts

Sir—In our recent paper in this journal on mortality among injecting drug users,1 we may have given the impression that there were 51 deaths among Glasgow injectors in 1989 compared with only 69 for the whole of the UK from 1956-1966 and 65 from 1976-1981. In fact there are no published data specifically on death among injectors in the UK and previous mortality among drug addicts reported to the Home Office Notification Index (HONI). Before September 1987 it was not ascertained whether those who were notified injected drugs and, on reflection, we believe we should have avoided making comparisons between the two groups since the UK figures we cited refer to deaths among notified non-therapeutic heroin addicts which are probably not a good surrogate for mortality among injectors. The earlier study reported only deaths among UK heroin addicts, of which there were 69 in 1956-66,2 while the later study analysed in more detail 1273 deaths among non-therapeutic notified addicts between 1967 and 1981.3 Drugs caused or were implicated in 939 of the 1273 deaths, although the specific drugs involved were identified in only 745 cases; these included 65 which were associated with heroin, 107 with methadone, 141 with other opiates, and 287 with barbiturates. The annual number of deaths (including 226 in therapeutic addicts over the 15 years) ranged from 51 in 1967 to 130 in 1975-78, and the average number of deaths per year over the 15 years was 107. By 1969 there were 300 deaths in previously notified addicts out of almost 1200 deaths associated with drug dependence, non-dependent abuse of drugs, and poisoning by controlled drugs in the UK that year.4 The methodology described in our paper was an attempt to circumvent the limitations of reporting mortality in selected groups of injecting users, such as those reported to the HONI, and enable straightforward comparison with the general population for a given year. When one considers that the average annual number of all-cause deaths in the Glasgow population aged 15-34 years is about 300, our finding of 45 deaths in drug injectors in the same age group (a further six were aged 35 or more) indicates that injecting drug use is strongly associated with an increased risk of mortality in young adults, and is probably responsible for more deaths among this group in Glasgow than any other single factor.


MARTIN FRISCHER
MICHAEL BLOOR
DAVID GOLDBERG
JOHN CLARK
STEPHEN GREEN
NEIL MCKEGANEY
Glasgow HIV Behavioural and Prevalence Study, Communicable Diseases (Scotland) Unit, Ruchill Hospital Glasgow G20 9NB