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Social inequalities and cardiovascular disease in South Asians

Justin Zaman,¹ Eric Brunner²

The epidemiological transition provides a temporal framework for thinking about the decline of infectious disease and the rise in cardiovascular and other chronic diseases.¹ In Europe, the transition began in the late 19th century with improved sanitation and housing, and controls on food adulteration. Continuing public health measures such as vaccination² contributed to the steep rise in life expectancy during the 20th century, paralleled by a sharp increase in the prevalence of cardiovascular disease (CVD). In South Asia, the epidemiological transition is taking place against a background of economic globalisation that has greatly increased the size of the urban poor and middle classes, at the same time leaving many millions to continue living on the land at subsistence level. Development is socially and regionally uneven, and so too are the common causes of morbidity and mortality. There is a double burden of disease in the countries of South Asia, characterised by a combination of pandemic infectious disease and high rates of cardiovascular disease. That India's burden of coronary disease was approaching a similar magnitude to that of the established market economies was demonstrated as long ago as 1990.³

THE LIVES OF SOUTH ASIANS OVERSEAS

Among South Asians in the UK, the latest figures show a continuing excess of CVD and in particular ischaemic heart disease (IHD) deaths.⁴⁻⁵ Based on population data from the 2001 census, standardised mortality ratios for IHD for men and women born in Bangladesh, India and Pakistan are between 131 and 175, using the whole adult population of England and Wales as the reference (SMR = 100). It is important, from research and policy perspectives, to understand the origins of this excess

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mortality. The question, simply stated, is whether it is biological or social in origin.

Socioeconomic circumstances are often very different from those of white people. South Asians are more likely to live in areas with relative social and economic deprivation.⁶ Compared with white employees, South Asians working in the Civil Service 20 years ago reported less social support at work and greater effort-reward imbalance after adjustment for age, sex and occupational status.⁷ Among patients undergoing coronary angiography for suspected cardiac chest pain, South Asian patients are less likely than white patients to undergo subsequent coronary revascularisation, taking into account clinical need and socioeconomic status, despite more consultations with a general practitioner in the year before angiography and more severe angina and coronary artery disease than white patients.⁸ It is thus of considerable interest that Tillin *et al* present evidence⁹ (**see article on page 476**) that socioeconomic disadvantage, both in childhood and in middle age, confers a marked increase in CVD death rates in later life in migrant men of South Asian extraction.

Biological explanations for susceptibility to IHD among South Asians focus on the metabolic complications of obesity. Central obesity is associated with high triglyceride and LDL cholesterol levels, insulin resistance and type 2 diabetes, and all of these are risk factors for IHD. The thrifty gene hypothesis proposes that conditions of food scarcity increased evolutionary selection pressure for efficient metabolism, which then disadvantages those who become exposed to the modern obesogenic environment.¹⁰ The weakness of this explanation is that the Indian subcontinent is relatively fertile and has long supported a large population. The thrifty phenotype hypothesis is a parallel concept, based on fetal undernutrition rather than genotype, which links low birth weight to later increased CVD risk.¹¹ Such relationships have, however, been demonstrated in many ethnic groups, and thus do not appear to explain the particular

susceptibility of South Asians to IHD. A third and novel explanation is that South Asians have a smaller superficial subcutaneous adipose tissue compartment than white people.¹² The "adipose tissue overflow hypothesis" reflects observations that, compared with white babies, babies born to mothers of Indian descent in London are smaller and have less peripheral fat (triceps skinfolds). During subsequent growth and development in circumstances of positive energy balance, this primary compartment reaches its capacity for fat storage rapidly and the deep subcutaneous and visceral compartments become more prominent, with adverse consequences for risks of diabetes and IHD.

Tillin *et al*'s study is based in Ealing, West London. Increased IHD risk was most apparent when socioeconomic disadvantage was measured as fewer years of education, rather than paternal occupation. This finding is consistent with the operation of an "aspiration effect" that is both economic and behavioural in its consequences. Middle-class South Asian migrants tend to be aware of the raised IHD risk associated with their ethnicity. However, the on-average poorer Bangladeshi and Pakistani communities in Britain may be less aware of the potential risk with respect to IHD, and less empowered to act on such awareness. The predominantly Punjabi population of Tillin's study is not representative of British South Asian communities, but the findings remind us of the diversity of migrant living circumstances and the intimate relationship between health and social class. Socioeconomic status may be as potent a risk factor as ethnicity itself. The Southall study suggests that childhood and adulthood socioeconomic disadvantage are additive in predicting cardiovascular disease mortality. However, there was little association between socioeconomic disadvantage and cardiovascular risk factors at study baseline, around 20 years after migration. Socioeconomic disadvantage is not simply a proxy for poor cardiovascular risk factor status, but also an indication of the likely trajectory that an individual may follow.

THE SOCIAL GRADIENT PHENOMENON

Are these findings that surprising, and do they have any policy implications beyond their undoubted academic credential? The social gradient phenomenon was observed in a 1978 analysis of mortality in the Whitehall study.¹³ This cohort of 17 500 men aged 40-64 years in secure Civil Service employment experienced widely

differing rates of IHD mortality according to their occupational status. The mortality pattern showed that coronary disease was a disease neither of affluence nor of poverty, but rather a disease of low social status. Is it so surprising we see the same pattern in South Asians? As for policy, The Black Report (1980) was the first of several investigations by the UK Department of Health that addressed the issue of social justice and health in the general UK population. The decline in CVD mortality began in the UK during the 1970s and at the start of the decade CVD death rates showed a modest inverse gradient according to Registrar General social class. By 1981 the gradient had become considerably steeper. The inverse gradient across the classes expanded further to 1991, in both relative and absolute terms, with a rate among unskilled manual workers (social class V) little different from that in 1971. In contrast, the CVD rate among professional workers was 60% lower than at the height of the epidemic. It seems that the South Asian population in Britain may be recapitulating the disease experience of their white counterparts.

A LESSON FOR EMERGING MARKET ECONOMIES?

Tillin *et al* have presented childhood socio-economic disadvantage as a risk factor. An interesting implication is whether such developed-world research can – and should – influence policy in the developing-world country of origin. If childhood conditions in a poorer country are a factor in later adulthood disease among migrants in a richer country, then South Asian economic development may recreate a similar social gradient in IHD among those who do not migrate as their own country grows richer. As far as we know, coronary risk factors remain concentrated in the urban

populations of modern-day India.¹⁴ Within these populations, however, higher levels of obesity and tobacco consumption are now associated with low levels of education and income.^{15 16} Increasing inequality within India can only be bad news for the health of its own population, if this research is a sign of the future. Though the poorest socioeconomic groups in India are more likely to die of infectious disease than CVD, with improved public health today's lower castes and classes in India may survive into adulthood only to be tomorrow's Indian IHD patients. Determining the relative contributions of social and biological risk factors for coronary disease in today's rapidly developing economies will necessitate well-designed, adequately powered studies in these countries.

IMPLICATIONS FOR PUBLIC HEALTH POLICY

Growing recognition of the socio-economic determinants in health has led to a change in perspective away from the biomedical model of health which partnered the development of modern health services. Public health professionals, encouraged by health campaigners, are increasingly turning to a population perspective that considers social and ethnic differences in health status to be important.¹⁷ Reducing these health inequalities must involve culture and institutions outside the health sector, based on an appreciation of the economic and social causes of disease. Tillin *et al*'s research provides a rationale for a broader approach in preventive cardiology.

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Not only after myocardial infarction

To the editor: We read with interest the study by Norhammar *et al*¹ describing the treatment and myocardial infarction survival rates according to the presence of diabetes. In their study, 1-year mortality rates decreased between 1995 and 2002 from 16.6% to 12.1% in patients without diabetes and from 29.7% to 19.7%, respectively, in those with diabetes. Despite improved pre-admission and in-hospital treatment, diabetic patients were less often offered acute reperfusion therapy, acute revascularisation or revascularisation within 14 days, aspirin and lipid-lowering treatment at discharge.

Although acute management of patients with myocardial infarction is important, treatment of this population to prevent secondary disease, is also crucial especially of those at higher risk such as diabetics. In a study recently performed in a Spanish clinical practice, in 2024 hypertensive patients with chronic ischaemic heart disease, the influence of diabetes on the diagnosis and therapeutic approach was analysed.^{2 3}

According to the paper of Norhammar *et al*, the presence of other risk factors and cardiovascular comorbidities was also more common in diabetics. Overall, diabetic patients were taking more medication (96.9% of diabetics vs 85.4% of non-diabetics were treated with ≥ 4 drugs, $p < 0.001$). Antihypertensive agents, calcium channel blockers (49.1 vs 42.2%), diuretics (45.1 vs 30.4%) and renin-angiotensin system inhibitors (83.6 vs 72.9%) were more commonly prescribed in diabetics (all $p < 0.01$), while β -blockers were used more frequently in non-diabetics (63.8 vs 68.7%, $p = 0.01$). Blood pressure control ($< 130/80$ mm Hg) was more common in non-diabetics (19.7% vs 26.4% $p = 0.001$). Lipid-lowering drugs were more frequently prescribed in diabetics (77.8 vs 73.9%, $p = 0.036$), but, nevertheless, no differences in low-density lipoprotein-cholesterol control rates were seen between the groups. Notably, patients with diabetes were surprisingly taking fewer antiplatelet agents than non-diabetics (85.2 vs 89.7%, $p = 0.003$). Finally, no differences in diagnostic procedures were found in the performance of stress test (84.5 vs 86.9%) or coronary angiography (60.9 vs 58.1%).

In agreement with Norhammar *et al*, in recent years the management of diabetics with coronary heart disease has been improving, but our data also confirm that application of evidence-based treatment is still lacking.

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Competing interests: None declared.

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The authors' reply: It was with great interest that we read your letter to the editor of *Heart* about a suboptimal use of evidence-based treatment in patients with diabetes and chronic ischaemic heart disease. We agree with your findings that there is insufficient control of risk factors in these patients. The importance of secondary prevention of disease in a diabetic patient after myocardial infarction is indeed highlighted in our report¹ and, for example, clearly visible in fig 2 as successively increasing mortality differences among non-diabetic and diabetic patients over time. This indicates a need for improved secondary preventive measures. Furthermore, a recent analysis from the Euro Heart Survey on diabetes and the heart showed poor adherence to secondary guideline goals, particularly among patients with diabetes. The target level for blood pressure control ($< 140/90$ mm Hg) was only reached by 30% of the patients and low-density lipoprotein-cholesterol levels were unsatisfactory (> 3 mmol/l) among 57%.² Considering the considerably lower targets for blood pressure and lipids advocated in the new European guidelines for patients with pre-diabetes and diabetes and coronary artery disease³ the present

situation must really be considered far from satisfactory.

In conclusion, we agree with Drs Barrios and Escobar that it is not only the acute management of myocardial infarction but also secondary preventive efforts that must be paid much more attention in order to improve the poor prognosis for patients with diabetes mellitus.

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CORRECTIONS

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F Antonini-Canterin, *et al*. Left atrial remodelling early after mitral valve repair for degenerative mitral regurgitation. *Heart* 2007;**94**:759–64. In the print journal, the order of the authors is incorrect. The correct order is: F Antonini-Canterin, CC Beladan, BA Popescu, C Gingham, AC Popescu, R Piazza, E Leballi, B Zingone, GL Nicolosi.

The corrected paper is available online at: <http://heart.bmj.com/cgi/content/full/94/6/759>.

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