Socioeconomic status and cardiovascular disease risk

To the Editor: We congratulate Dr A Steptoe and associates on their extensive epidemiological research concerning cardiovascular risk and its association with pathogen burden depending on socioeconomic status.¹ Since the middle of the 20th century, cardiovascular risk in the populations of Europe and the United States has shown a reverse dependence on economic and professional status. Many researchers, including the authors of this study, have attempted to find the causes of this tendency.² The study referred to provides a lot of epidemiological information about the role played in vasculitis and the pathogenesis of atherosclerosis by universally appearing infectious factors. The idea of associating the infectious factors initiating inflammatory changes in vessels influencing the development of atherosclerosis with socioeconomic status is inventive and interesting.

Examinations were conducted on 1201 patients aged 40–60 selected from the population of Lublin (Poland).³ Obesity and overweight were the most frequent cardiovascular risk factors. The most frequent occurrence of this disease was noted among patients with primary education, the least frequent among those with higher education. Similarly, it was proved that the lower the patients' level of education, the greater and more severe the cigarette smoking habit. Hence the cardiovascular risk factors regarded as the indicators of social stress (obesity, smoking) occurred more frequently among the least educated patients. Moreover, social stress is believed to be one of the causes of obesity and smoking in the final phase. Having assessed the total cardiovascular risk, we observed the existence of higher-level coronary artery disease risk in patients with vocational education than in people with higher education.

Our findings are in accord with those recorded and currently observed in many European countries, where researchers have noticed a reverse dependence of coronary artery disease on high standard of living. Consequently, there are more cases of the disease among lower social classes. Identifying the causes of this tendency will allow better therapy and prevention strategies to be designed. My presentation and discussion of Dr A Steptoe's research aims at finding the underlying cause of this tendency.

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The authors' reply: We thank Drs Mieczkowska and Mosiewicz for their interest in our research on socioeconomic status (SES), pathogen burden and cardiovascular disease risk. They rightly highlight the strong social gradient in cardiovascular disease that is present in many European countries and is increasingly apparent worldwide.¹ Our study used employment grade as the indicator of SES, and it is interesting that similar patterns emerge in Poland with level of education as the marker of social position. The social gradient is not fixed, since there was a positive association between SES and cardiovascular disease in the UK before the 2nd World War, shifting to an inverse gradient in the post-war era.² Drs Mieczkowska and Mosiewicz also point out the important role of adverse lifestyle factors such as smoking and obesity in maintaining the SES gradient.

Our data on pathogens relate to cardiovascular risk factors such as adiposity, blood pressure and diabetes. However, we have recently had the opportunity to study a more direct measure of vascular pathophysiology in this population, namely arterial stiffness. Carotid arterial stiffness was measured using ultrasonography in conjunction with the Vascular Physiology Unit at the Institute of Child Health, University College London. The distensibility coefficient was calculated from the distension of the common carotid artery 1 cm proximal to the carotid bifurcation and simultaneous blood pressure data using standard formulae,³ with larger distensibility coefficients indicating lower arterial stiffness. Half the participants

(50.8%) were seropositive for cytomegalovirus (CMV), and they had significantly lower distensibility coefficients than the remainder of the sample, with average levels of 14.77 (SD 4.2) 10^{-3} kPa⁻¹ compared with 15.71 (SD 5.2) 10^{-3} kPa⁻¹ after adjustment for age, gender, body mass index, waist/hip ratio, smoking, blood pressure and high density lipoprotein-cholesterol ($p = 0.041$). This indicates that carotid stiffness is greater among individuals with a history of CMV infection. CMV seropositivity was also more common among individuals of lower SES (indexed by grade of employment).⁴ But interestingly, when SES was included as a covariate in the analysis, the association between CMV seropositivity and arterial stiffness remained significant ($p = 0.050$), endorsing the conclusion of our article that the risks associated with infection history and lower SES are somewhat independent of one another.

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CORRECTION

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T Stokes, *et al*. Prophylaxis against infective endocarditis: summary of NICE guidance. *Heart* 2008;**94**:930–1. The third author of this paper was spelt incorrectly and should be D Wray. The correct authorship is: T Stokes,¹ R Richey,¹ D Wray,² on behalf of the Guideline Development Group. In addition, the Guideline Development Group member J Gibb should be J Gibbs.

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