In this issue, Stephen Kunitz examines the scientific underpinning of social epidemiology, and in particular its ability to predict associations between health outcomes and exposures as diverse as sex, race and social position. General notions regarding ‘social determinants of health’ may not be particularly useful if insufficiently aware or curious about the importance of the contextual aspects of these relationships. If we cannot predict the effects of economic growth, of political change, or of social policies on health outcomes with any accuracy, is social epidemiology of much value? Kunitz argues that social epidemiology is valuable (and a science) but that generalizing theories are not well-founded.

So what should we do about the continued social inequalities in health outcomes widely observed in many diverse countries? The theme of this issue is socio-economic position and health and as is so typical in this field of research, we have volumes of descriptive analysis showing more or less strong associations between different measures of socio-economic position and disease. Showing that health outcomes are socially patterned was interesting two decades ago as the evidence was used to highlight the existence of other unknown but potentially preventable causes of disease. On this basis, the evidence of a social gradient in risk of cardiovascular disease, not explained by classical risk factors, led to a search for other causes of cardiovascular disease. The search for novel risk factors has become an industry with a life of its own, ranging from massive research efforts on genetic variants, biomarkers, to the shifting sands of psychosocial determinants. An equally large volume of work has been conducted describing social inequalities in a bewildering array of health outcomes ranging from self-rated health to mortality. Has any of this work led to improvements in adverse social gradients in disease? Please send me examples if you have them.

Having shown that gastric and oesophageal cancer are patterned by educational attainment in several European countries in the European Prospective Investigation into Cancer and Nutrition (EPIC) study, and that the associations were robust to adjustment for drinking, fruit and vegetable intake and smoking, but were attenuated by adjustment for Helicobacter pylori seropositivity, Nagel and colleagues conclude that a search for further correlates of educational level that are linked to cancer development is warranted. They acknowledge that residual confounding is a likely problem here, and that measurement error in dietary and other exposures is also a difficulty, but do not provide any quantitative estimates of how much of the effect might be explained by these factors. What is strikingly absent is any notion that educational attainment—grouped as primary school or less, vocational secondary, other secondary and college and university—is context dependent. Among participants aged 35–70 at recruitment between 1992 and 2000, the oldest would have been educated in the 1920s and the youngest in the 1960s—a period of dramatic changes in access to education. The meaning of limited educational attainment at times of full access and times of very limited access will have different implications for causal reasoning.

What is interesting is to examine whether health and social policies are having an impact on inequalities in health outcomes. In this issue, Korda and colleagues have examined avoidable and unavoidable mortality between 1986 and 2002 by socio-economic status in Australia—a country that prides itself on its guiding principle of equity in health care. These investigators found that declines in avoidable mortality were greater in more advantaged than disadvantaged groups, leading to an increase in relative inequality. However, absolute differences in mortality between 1986 and 2002 were greater among the disadvantaged than the advantaged. The authors conclude that health care has had an impact reflected in the narrowing absolute differences in mortality, but that advantaged people had gained proportionately greater benefit from the health care system than disadvantaged groups, leading to an increase in relative inequality.

South Korean boys have a much greater risk of road traffic injury than girls, and this risk appears to be socially patterned, with boys in the lower third of parental income having a 2-fold higher risk than those in the highest income group in both early and late adolescence. The analysis was based on a prediction that health inequalities are not present in early adolescence due to a declining effect of parents and home environment and an increasing influence of school, peers and youth culture. Health inequalities are then predicted to re-emerge in late adolescence due to ‘health-related social mobility’—sicker kids don’t climb the social ladder. Adolescents in Korea are an interesting population in which to test this hypothesis as the country has undergone rapid social and economic development, and patterns of inter-generational interaction and influence are changing rapidly. However, the data do not fit the predicted pattern of association.

In related vein, Osler and colleagues try to unpick the question of whether social inequalities in health arise because of causal links between exposures across the lifecourse (genetic, prenatal, family, occupational and lifestyle) or are non-causal and due to selection of healthier children into higher social positions in adulthood. Using data on height, BMI, grip strength, depressive symptoms, self-rated health and health behaviours, they found no major differences in health or health behaviours in monozygotic twin pairs discordant for adult socio-economic position. In dizygotic twins discordant for socio-economic position, cognitive tests were better in the twin with higher social position. For most of the health outcomes examined, twin pairs differed depending on zyosity and not...
social position, suggesting that adult social gradients in health are due to selection and not due to the effects of socially determined exposures. These are important findings that now require replication in larger twin data sets and using a wider range of health outcomes.

In a nicely opportunistic piece of work, Adda et al.\(^6\) examine the economic effects of the smoking ban in Scottish pubs. Their findings are of a 10% fall in sales and a 14% decline in customers. Our commentator emphasizes that these are short-run findings, with experience in other countries showing a rebound back to usual economic levels within a year.\(^7\) So why did we choose to publish what some may see as a paper demonstrating a downside of a smoking ban in pubs? Methodologically, there is much to praise in the approach adopted as it makes use of a natural experiment in which the Scottish ban was implemented before any action in England. Before and after the ban measurements were made by telephone interviews with landlords in Scotland and northern England (which shares similar drinking behaviour with Scotland). Finally, they included price of the best selling beer as a variable in analyses to explore whether reactive price increases might explain falls in sales and customers; they did not. Evaluation of public health interventions is crucial, and understanding economic impacts—even short-term effects—is an important part of moving to more evidence based public policy.

In common with many epidemiologists of an earlier era, I saw patients for much of my career, and remain interested in the causes and prevention of disability in older people. David Graham’s photo essay ‘Acts of God’\(^8\) provides a valuable account of his son’s dramatic change from an active young man to quadriplegia following a serious injury while swimming. Epidemiologists tend to think of disease in binary terms—one of my colleagues used to label mortality as ‘stiff: 1¼yes, 2¼no’—but this hides the complexity of many non-binary disease outcomes, like quadriplegia, that evolve over time. And it makes epidemiologists much less interested than they might be in the determinants of different patterns of adaptation to disease.

A topic of personal interest to a person of south Asian extraction is the increased cardiovascular risk among south Asians. Sniderman and colleagues\(^9\) put forward a novel idea—the adipose tissue overflow hypothesis. The thrifty gene hypothesis—food scarcity selects out those with low metabolic requirements—is usually trotted out as an explanation. Sniderman considers it lacks face validity if one takes a cultural and historic perspective on the highly successful societies that have emerged there. The adipose tissue overflow hypothesis is concerned with differences between whites and south Asians in the size of their body fat compartments. They suggest that south Asians have a much smaller superficial fat compartment which means that as energy excess is stored as fat, south Asians fill their superficial compartments before whites and go on to fill up their deep subcutaneous fat and visceral compartments, both of which are implicated in dyslipidaemia and hyperglycaemia. Unlike the thrifty genotype hypothesis, so far at least, this one is eminently testable.

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## References