Commentary: Risk factors or social causes?

Michael Marmot

In many industrialized countries, there has been a welcome decline in coronary heart disease (CHD) mortality. In England and Wales, this has progressed for two decades. Many would like to take the credit for this decline, but the reasons for it are not entirely clear. Contributors to the decline are likely to include reductions in smoking, dietary change—but not the increase in obesity—and improvements in treatment, both of risk factors and the disease after it hits.

The shadow over this bright picture is cast by the uneven nature of the improvement. In general, the higher the socioeconomic position the greater has been the improvement. As a result, social inequalities in CHD have increased. It is useful to keep this background in mind when considering the paper by Emberson et al. in this issue of the International Journal of Epidemiology. Based on analyses from the British Regional Heart Study, they argue that population wide control of risk factors—blood cholesterol, blood pressure, body mass index, cigarette smoking, alcohol, physical activity, and lung function—is a better strategy than one designed specifically to reduce social inequalities in CHD.

The main reason for this conclusion is that the contribution of social class to CHD is ‘modest’. In their calculation, social class accounts for ‘only’ 22% of the CHD that occurs in a population of middle-aged British men. I must confess to having used the word ‘modest’ frequently in scientific writing. If 22% of CHD deaths can be described as modest, this literary habit needs examination. None of us in public health is in any doubt about the damage to population health caused by smoking. Indeed, the recent WHO global burden of disease study confirms tobacco as the single population health caused by smoking. None of us in public health is in any doubt about the damage to population health caused by smoking.

The ‘latter day’ view to which they refer comes, of course, from Robert Koch. Koch wrote of his discovery of the tubercle bacillus:

One has been accustomed until now to regard tuberculosis as the outcome of social misery and to hope by relief of distress to diminish the disease. But in the final struggle against this dreadful plague of the human race one will no longer have to contend with an indefinite something but with an actual parasite.

Collis and Greenwood consider the view that now we have the cause, the tubercle bacillus, we can ignore the contribution of social inequalities. They conclude:

that the general belief of our fathers and grandfathers is sound, and the policy which ought to have been, and to some extent was inspired by that belief is a sound policy. What is the matter with the poor is largely poverty. Not through any special intensive measures of campaigning against the tubercle bacillus, not even by the segregation of the actively tuberculous, does there seem any real hope of salvation. We have to improve the homes of the working classes in the first place . . .; in the second place, we have to ensure better factory conditions.

I will only rehearse the McKeown arguments to the extent of reminding us that most of the decline in tuberculosis in the 20th century occurred before there were specific methods available to treat the disease. It is likely that improved social conditions led both to interrupted transmission of the infectious organism and increased resistance of those infected to the clinical effects of tuberculosis.

How is this relevant to the present discussion? The Emberson et al. argument could be characterized as: we have the ‘cause’—coronary risk factors—we do not therefore need to pay attention to the social conditions that give rise to social inequalities in CHD. I am more with Collis and Greenwood. What is the matter with people of lower social position is their social position. Ignoring that and attempting to control risk factors is not working to prevent CHD in those of lower social position. Witness the increased social inequalities in CHD.
Attempts to control risk factors have not led to reductions in inequalities in CHD incidence for two reasons. First, improvements in risk factors have been patchy. Smoking continues to show a clear social gradient. Emberson et al. acknowledge that in this area, at least, there needs to be special attention to the social distribution of smoking. Second, the established coronary risk factors do not account for the major part of the social gradient in CHD mortality. In the first Whitehall study, the 25-year follow up showed that adjusting for coronary risk factors reduced the relative mortality of low grade men compared with high grade men from 1.77 to 1.52—a reduction of 27%. In Whitehall II, with the reduction in smoking prevalence, we still see a social gradient in CHD, but the contribution of the risk factors to the gradient is marginally less. We have taken measurement imprecision into account by assessing smoking on three occasions over 5 years and calculating pack years (unpublished data).

If being of manual class accounts for 22% of all CHD among middle-aged men, its contribution is far from modest and requires attention. Control of risk factors and attention to social inequalities should not be thought of as alternative strategies. We do indeed need to pay attention to the causes of inequalities in health, and the Acheson report suggested 39 ways that could be done, starting from the beginnings of life and carrying through to old age. We also need to pay attention to risk factors. The evidence of the last 30 years suggests that unless we build into our programmes specific concern with inequalities we shall be fortunate if our attempts to reduce disease are spread equally among all social groups.

References