Down at heart the meaning and implications of social inequalities in cardiovascular disease

The ubiquitous nature of health inequalities is reflected in the long history of their description. Differences in life expectancy between social groups have been demonstrated since the early days of industrialisation. In 1845 Frederick Engels presented such data, using both area-based and individual indicators of socioeconomic position (Table 1). Engels went on to quote from the Report on the Sanitary Conditions of the Working Class: 'In Liverpool in 1840 the average lifespan of the upper classes, gentry, professional men, etc, was thirty-five years; that of the business men and better-placed handicraftsmen, twenty-two years; and that of the operatives, day-labourers, and serviceable class in general, but fifteen years. Engels described the wretched housing conditions of the working people, the soul- and body-destroying work, the poor sanitary state of the city environment and the inadequate diet with which they had to contend. The direct contribution of such factors to poor health seemed obvious to Engels, as it would to any reader of his book today.

Engels was writing at a time when the death rate in some of the great cities was rising and the decline in overall population mortality rates that had been occurring in Britain had ceased. From the 1860s on, however, mortality rates started to decline, first for children and young adults and then for older adults. Infant mortality rates, interestingly, did not fall until after the turn of the century. With only occasional reversals – seen, for example, among middle-aged men and women between 1920 and 1940 – mortality rates have continued to decline. Against this background of generally declining mortality rates, socioeconomic differentials have persisted. During the depression of the 1930s, another period when the influence of environmental adversity on poor health and increased mortality risk seemed obvious, Dr G C M M’Gonigle, Medical Officer of Health for Stockton-on-Tees, analysed deaths in a group of local residents over a four-year period in relation to family income. Age-standardised death rates and family income were inversely related (Table 2) and there was a continuous gradient between family income and death rate.

Since the 1921 census (with a trial run around the 1911 census) statistics on mortality according to social class have been produced for England and Wales, providing at international level the best longitudinal series of such data. Figure 1 presents mortality rates for middle-aged men from the years around the 1921 census to the years around the 1981 census. Dramatic declines in mortality are seen for social classes I and II, while for social classes IV and V decreases in mortality are small and inconsistent. The relative and absolute differentials in mortality between the social class groups have widened since the early 1950s, a pattern that has continued throughout the 1980s.

Socioeconomic differentials in cardiovascular disease

Socioeconomic differentials in cardiovascular disease risk should be considered against the background of large and increasing differentials in all-cause mortality rates in the population. It is popularly thought that cardiovascular disease used to affect the rich more than the poor. This is true only if the category of ischaemic heart disease is considered alone. For overall mortality rates, the social class differentials in the 1930s were very small, and the increase in differentials over the subsequent 60 years has occurred mainly among the young and middle-aged. 

Table 1. Mortality ratios (number of living people for each death) in Chorlton-on-Medlock, Manchester

| Class of street | Class of house |
|----------------|----------------|
| 1st (best)     | 2nd            | 3rd (worst)   |
| 1st (best)      | 51             | 45            | 36          |
| 2nd             | 55             | 38            | 35          |
| 3rd (worst)     | -              | 35            | 25          |

* No data. Source: Engels, 1845.

Table 2. Death rate versus income in Stockton-on-Tees during the depression of the 1930s

| Income in £ per week | Death rate per thousand |
|----------------------|-------------------------|
| 1.25–1.75            | 26.0                    |
| 1.76–2.25            | 19.3                    |
| 2.26–2.75            | 19.2                    |
| 2.76–3.25            | 15.1                    |
| 3.26–3.75            | 13.5                    |
| Over 3.75            | 11.5                    |
all cardiovascular disease mortality the lower, manual social classes experienced higher rates than the higher, non-manual social classes when the statistics first became available, around the 1911 census\textsuperscript{10,11}.

Coronary artery disease was considered a disease of the affluent classes from the time it was first recognised. Dr Samuel Black of Newry, County Down, described many cases of angina pectoris and contributed to the development of the hypothesis that ischaemia underlay angina pectoris\textsuperscript{12}. He associated susceptibility to angina pectoris with male sex, stress and ‘full and plethoric habits,’ and protection from angina with female sex, hard labour, exercise, and being French. The congruence with current thinking is remarkable. However, he considered the better ranks of society to be more liable to the disease and the poor relatively exempt.

The perceived increased susceptibility of the ‘better ranks of society’ persisted during the first two decades of this century as the full spectrum of coronary artery disease, including myocardial infarction, was recognised. Large social class differences, with the non-manual groups at considerably greater mortality risk than the manual groups, were seen in the analyses around the 1911, 1921 and 1931 censuses\textsuperscript{13}. Writing in 1950, Dr Ian Stewart invoked George Orwell’s Nineteen eighty-four, stating that Orwell saw the workers of 1949 as the ‘proles’ of 1984 for whom ‘heavy physical work, the care of home and children, petty quarrels with neighbours, films, football, beer, and, above all, gambling, filled up the horizon of their minds. To keep them in control was not difficult.’ The proles, however, were the only happy people in Oceana, Stewart goes on, ‘and one can be sure that like their prototypes – the working class of 1949 – they were little troubled by coronary disease’\textsuperscript{14}.

Sir Robert Platt took issue with Stewart’s claim that ‘the better educated and those who work with their brains are more liable than their fellows to coronary disease’\textsuperscript{15}. Platt quoted data from the Registrar General’s Decennial Supplement for 1931 showing standardised coronary heart disease mortality rates being: agricultural workers 32; coal-miners below ground 40; banking and insurance officials 183; Anglican clergy 218; physicians and surgeons 368\textsuperscript{13}. There was a tenfold gradient in coronary heart disease mortality across these occupational groups. Platt went on, however, to suggest that the likelihood of persons dying between 1930 and 1932, after having seen a cardiologist, would be in the following descending order: physicians and surgeons; Anglican clergy; banking and insurance officials; coal-miners below ground; and agricultural workers. The clear implication was that varying diagnostic practices for members of different social groups, partly due to the types of physicians they consulted, generated the apparent differentials in morbidity from coronary heart disease. Coronary disease, in the early 1930s, would often be recorded as myocardial degeneration and myocarditis. Mortality from these categories was higher among manual than non-manual groups. Others shared Platt’s views\textsuperscript{16}.

Analyses using area-based measures of socioeconomic status did not show the expected coronary heart disease gradients in the early 1950s\textsuperscript{17}. Studies with objective measures of disease, or with standardised methods of classification across social groups, show much less evidence of higher rates of coronary heart disease amongst the better off than those studies where data were more liable to diagnostic bias and differential misclassification of disease according to socioeconomic position\textsuperscript{18}.

The geographical distribution of coronary heart disease, unlike the apparent social class differentials, has remained stable. Rates have always been higher in the North West of England than in the South East\textsuperscript{18-20}. This gradient, which existed earlier this century, is not congruent with the proposed positive association between coronary disease and socioeconomic position, since the North West has remained a less prosperous region than the South East over the century\textsuperscript{21}. In conclusion, the evidence for a marked positive social gradient in coronary heart disease earlier this century

\begin{figure}[h]
\centering
\includegraphics[width=\textwidth]{Fig1.png}
\caption{Death rates per 100,000 men aged 50–64 in England and Wales, 1921–1981}
\end{figure}
is considerably weaker than is generally supposed. The apparent social class cross-over cannot be considered as providing strong support for any aetiological theory that cites it in its support.

Currently there are large social class gradients in mortality from all the major cardiovascular diseases. Figure 2 displays social class differences in ischaemic heart disease, stroke and all-cause mortality for men of working ages between 1976 and 1989\textsuperscript{10}. The socioeconomic distribution of mortality due to cardiovascular disease is reflected in morbidity rates. In a large survey of more than 20,000 people aged 35 and over in Somerset and Avon, histories of angina, myocardial infarction and stroke were all more common amongst individuals living in deprived than in affluent areas. (Table 3)\textsuperscript{22}. Socioeconomic position is also related to the early stages of developing cardiovascular disease. For example, low income, manual occupation and little education are all related to an increased degree of carotid atherosclerosis in a Finnish study\textsuperscript{23}.

**Factors contributing to socioeconomic differentials in health**

The first Whitehall study of London civil servants demonstrated considerable differences in mortality risk according to two socioeconomic measures – employment grade in the civil service and car ownership. Mortality rose steadily from 4.4 to 11.0/1,000 person years in car owners and from 4.9 to 18.8 in non-car owners from the higher to lower employment grades. Car ownership was a good indicator of available income in the late 1960s, when this study was established\textsuperscript{24}, and within each employment grade the non-owners had higher mortality. Although the lower grade and non-car owning civil servants were more likely to smoke than the higher grade and car owning ones, the pattern of mortality differentials among men who had never smoked was identical to that of the whole cohort\textsuperscript{25,26}. The above data are for all-cause mortality, but cardiovascular disease mortality showed similar associations with employment grade and car ownership to all-cause mortality (Fig 3).

Cholesterol levels were greater among higher than among lower grade civil servants in the late 1960s, when this study was established. Differences in cholesterol levels could not, therefore, account for the higher rates of coronary heart disease among the lower grade employees. This can be taken to suggest that differences in dietary fat intake between grades were not responsible for the coronary heart disease mortality differentials. Indeed, simultaneous consideration of a range of risk factors – smoking, blood pressure, cholesterol levels and prevalent cardiorespiratory disease – failed to account for the differences in cardiovascular and non-cardiovascular mortality between employment grades\textsuperscript{24}. Similar findings have emerged from a study in the West of Scotland conducted about the same time as the first Whitehall study\textsuperscript{27}. Large differentials in cardiovascular disease mortality according to both educational attainment and social class existed at a time when blood cholesterol levels were highest in those with the most education and in professional and managerial occupations. Adjustments for a wide range of risk factors failed to explain the considerable mortality differentials from cardiovascular disease in this study. These findings are not limited to British studies. A prospective study of a third of a million men screened for the Multiple Risk Factor Intervention Trial between 1970 and 1973, with 16 years of mortality follow-up, found a strong association between the income level of the men’s area of residence and their risk of mortality from coronary heart disease and stroke\textsuperscript{28}. While adjustment for smoking, cholesterol levels, blood pressure and diabetes somewhat attenuated these associations, it did not remove them.

As a result of the finding that conventional risk factors fail adequately to account for the social distribution of cardiovascular disease, the Whitehall II study was initiated in 1985 to explore additional psychosocial, behavioural, dietary and metabolic factors that
could contribute to the socioeconomic differentials in health. Both male and female higher grade civil servants – with higher incomes – had lower prevalence of cardiorespiratory disease. Average cholesterol levels were similar in each grade, but concentrations of serum apolipoprotein AI, the main structural protein of high density lipoprotein (HDL) cholesterol, showed an association with grade and suggested that characteristic disturbances of lipid metabolism associated with lower occupational status were potentially identifiable.

An opportunistic study using data from the Whitehall II civil servants cohort assessed the effect of job insecurity on health status. When the baseline examinations were carried out in the mid-1980s the civil servants thought they had secure jobs for life. Privatisation of some civil service function was discussed and then implemented. The first civil service department into which these changes were introduced was the Property Services Agency (PSA), for whom it became clear from 1988 on that changes were to be made and that jobs were therefore insecure. By 1993 the PSA was fully privatised. The rest of the civil service remained a relatively secure employer, at least until 1990. Thus the health of the group of people undergoing the stress of anticipation – and then the actuality – of their employment being rationalised could be examined in relation to a control group. At the time of the repeat examinations in 1990 PSA workers, who had generally better health at the time of the baseline examinations than the rest of the cohort, were reporting more symptoms of ill health and worse overall health over the previous year. Two years later repeat clinical examinations were made, when the PSA employees were actually experiencing rationalisation, privatisation and loss of secure employment. Cholesterol levels and body mass index (BMI) in men and women had increased in the PSA compared to the other civil service departments. There was an increase in blood pressure for women and a non-significant increase in ischaemia for men and women combined. These effects suggest higher cardiovascular disease risk among members of the civil service agency first to experience privatisation. This study demonstrates how a particular form of social stress can increase cardiovascular disease risk. Further work of high methodological quality on the effects of

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**Table 3. Age standardised prevalence per 100 of self-reported illness by deprivation category**

| Condition               | 1st fifth | 2nd fifth | 3rd fifth | 4th fifth | 5th fifth | p value (test for trend) |
|-------------------------|-----------|-----------|-----------|-----------|-----------|-------------------------|
| **Men**                 |           |           |           |           |           |                         |
| Angina                  | 4.4       | 5.5       | 5.5       | 5.5       | 6.9       | < 0.001                 |
| Myocardial infarction   | 3.2       | 3.7       | 4.0       | 4.5       | 4.8       | < 0.001                 |
| Stroke                  | 2.0       | 1.8       | 1.3       | 2.3       | 2.6       | 0.03                    |
| **Women**               |           |           |           |           |           |                         |
| Angina                  | 3.8       | 4.4       | 4.6       | 4.4       | 5.8       | < 0.002                 |
| Myocardial infarction   | 1.5       | 1.9       | 1.7       | 1.8       | 2.5       | 0.03                    |
| Stroke                  | 1.6       | 2.0       | 2.1       | 2.2       | 2.4       | 0.04                    |

Source: Eachus et al. 1996.

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**Fig 3.** Cardiovascular disease mortality by employment grade and car ownership in the Whitehall study. Source: Davey Smith et al, 1990.
psychosocial stress on risk of disease is required, since much research in this area is difficult to interpret.

**Life course influences on socioeconomic differentials in cardiovascular disease risk**

Until recently the debates on inequalities in health concentrated on socioeconomic circumstances in adulthood. There has recently been a revival of interest in the effects of poor social circumstances in early life on health in adulthood\(^5\)\(^-\)\(^3\)\(^3\). The UK Department of Health report, *Variations in health*\(^4\), has recognised the importance of a life course perspective on inequalities in health. It concludes that it is likely that accumulative differential lifetime exposure to health-damaging or health-promoting physical and social environments is the main explanation for observed variations in health and life expectancy. Few empirical data regarding such cumulative effects exist, however. In a cohort study in the West of Scotland\(^5\) in which men have been followed for over 20 years, it was possible to relate death rates to the social class of the fathers of the cohort members; to the social class of the first occupation of the men on entering the labour market; and to the social class of their occupation at the time of screening, when aged 35–64. Table 4 demonstrates that cumulative social class — indexed simply by summing the manual and non-manual social class locations at the three stages of the life course — together with other indicators of socioeconomic position at the time of screening, are strongly related to mortality risk. When social class at different periods of the life course is related to mortality from specific causes, social class of the fathers of the men and their own social class at the time of screening independently contribute to all-cause and cardiovascular disease mortality. This indicates that there are some long-lasting influences of socioeconomic circumstances in childhood on mortality in adulthood.

The suggestion that the risk of dying reflects the accumulation of environmental insults across the individual's life or the cumulative effects of unfavourable behavioural or psychological factors that progressively increase susceptibility to disease\(^5\)\(^7\)\(^-\)\(^8\) receives further support from a study based on record linkage of the 1960, 1970 and 1980 census records for Norway, in which particularly high mortality risks are seen among men with limited education who then went on to work in manual occupations and live in poor housing. Similar findings have come from the US National Longitudinal Study of older men. The particular dependence of cardiovascular disease risk on childhood socioeconomic circumstances in comparison to other causes of death has been observed in area-based studies from Finland\(^4\)\(^1\)\(^,\)\(^4\(^2\)\). Socioeconomic circumstances in childhood and adulthood have been examined with respect to a variety of cardiovascular disease risk factors in the above-mentioned Scottish cohort. Cigarette smoking was commoner amongst the men in manual than in non-manual occupations, with father's social class contributing little to the distribution of smoking behaviour when examined in addition to the social class of the men themselves. This suggests that smoking behaviour is determined by current social environment rather than by any particular influences of childhood environment. Height is associated both with father's social class and with own adulthood social class. This could reflect environmental – particularly nutritional – effects from early life, together with the possible contribution of height-related upward social mobility. Body mass index was more strongly associated with father's social class than own social class, the reverse being the case for blood pressure and cholesterol levels. Thus, experiences at different stages of life make different contributions to particular cardiovascular disease risk factors. Fibrinogen was not measured in the Scottish cohort, but was included in the Whitehall II study. In this study fibrinogen levels were associated with indicators of both parental social class and current socioeconomic position, with poorer social circumstances being related to higher plasma fibrinogen.

**Table 4. All-cause mortality by cumulative social class, car and deprivation category (age-adjusted relative rates)**

| Cumulative social class | All 3 non-manual | 2 non-manual/1 manual | 2 manual/1 non-manual | All 3 manual |
|-------------------------|-----------------|----------------------|----------------------|-------------|
| Car                     | 1               | 1.28 (1.01–1.63)     | 1.36 (1.08–1.73)     | 1.57 (1.27–1.95) |
| No car                  | 1.22 (0.91–1.64)| 1.52 (1.19–1.95)     | 1.76 (1.40–2.21)     | 2.00 (1.64–2.44) |
| Deprivation category 1–4| 1               | 1.25 (1.01–1.56)     | 1.37 (1.09–1.72)     | 1.70 (1.39–2.09) |
| Deprivation category 5–7| 1.06 (0.74–1.52)| 1.41 (1.10–1.82)     | 1.54 (1.25–1.90)     | 1.74 (1.45–2.09) |

Source: Davey Smith et al, 1997\(^4\).
A nutritional factor that may contribute to the accumulation of disease risk throughout life is that of fruit and vegetable intake\(^6\). In a study of Finnish men\(^6\), intakes of fruit, non-root vegetables and vitamin C were related to childhood socioeconomic circumstances, implying that this adult dietary behaviour may become established early in life.

Of particular current research interest are the long-term effects of development during fetal and early infant life on disease risk in adulthood. While many previous examples can be found\(^5,33,17,48\) interest was regenerated in early influences on adult cardiovascular disease mortality by the work of Forsdahl in Norway\(^49\), who related infant mortality rates earlier this century to present day coronary heart disease mortality rates. He demonstrated that in areas where infant mortality rates had been high in the past, and where, by implication, children had been nutritionally deprived both in early infancy and in childhood, mortality from coronary heart disease 70 years later was also high. Whilst these data are suggestive, it is precisely those places that had high infant mortality rates at the beginning of this century that remain the most deprived places today. If current deprivation levels are taken into account in the analysis, there is essentially no residual association between past infant mortality rates and present coronary heart disease mortality rates (Fig 4)\(^50\). Studies are needed with adequate data throughout life if the separate effects of early and later life exposures are to be ascertained.

Since the pioneering work of Forsdahl a series of ecological and prospective studies has demonstrated that birth weight and weight at one year of age are inversely related to cardiovascular disease, diabetes and blood pressure in later life\(^51\). These findings support the proposition that there are important persisting influences on cardiovascular disease risk from early life into adulthood, and encourage the establishment of studies with adequate data across the life course, to take this area of research forward.

In the recent period, little research has been carried out on the effects of childhood nutrition on later disease, although earlier this century it was considered obvious that such effects did exist\(^5,33\). Preliminary data are now available from a mortality follow-up of the children included in surveys of poverty, nutrition and child health carried out under the auspices of Lord John Boyd Orr immediately before the Second World War\(^52\). At the time this survey was carried out, one of the investigators recognised that leg length was a particularly good indicator of childhood socioeconomic and nutritional circumstances:

> "When the Carnegie UK Dietary and Clinical Survey was planned at the Rowett Research Institute in 1937, cristal height as a measure of leg length was included in the measurements. . . it was found that cristal height was consistently better than total height for indicating expenditure group . . . we find the longer-legged children suffered less bronchitis than the short at all ages. Since there is neither complicating immunity mechanism nor specific cure for bronchitis, we might argue that constitution built up when the complete harmonious pattern of growth is unfolded is, in some way, superior to that associated with inhibition of growth, however slight.\(^35\)"

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**Fig 4.** Infant mortality rates 1905–8 and female ischaemic heart disease mortality age 65–74 in 1969–73 before (a) and after (b) control for measures of adult deprivation. Source: Ben-Shlomo and Davey Smith, 1991\(^50\)
In a re-analysis of these data this is clearly the case\(^\text{54}\) (Table 5). Age-standardised indicators of total height, leg length and trunk length reveal differential associations with nutritional and socioeconomic factors. In particular, the negative correlations between overcrowding and social class of head of household (scored from 1 for professional groups to 5 for unskilled manual workers) are considerably stronger for leg length than for trunk length, while the positive correlations between weighted per capita food expenditure and relative family per capita calorie consumption are also stronger for leg length than trunk length. This applies to men and women equally.

Leg length in childhood is associated with mortality over the subsequent 60 years (Table 6). These data suggest that there may be important long-term consequences of childhood nutrition on health in later life. They do not, however, paint a one-sided view of rapid growth in childhood. In line with evidence from animal studies and some epidemiological findings\(^\text{55,56}\), they suggest that cancer risk may be increased by greater calorie intake and growth in early life. Thus reductions in cardiovascular disease mortality in response to socioeconomic and nutritional conditions that encourage growth in childhood may, in part, be counterbalanced by increases in cancer mortality.

The ratio of leg length to total height changes throughout childhood, but those children with a high leg length to total height ratio are likely to become adults with a high leg length to total height ratio. Thus it is possible to investigate the association between leg length in adulthood and cardiovascular disease risk, using the former in part as a proxy for exposures during childhood. In the Caerphilly study, data on sitting height and leg length were obtained and ten-year follow-up of coronary heart disease incidence and mortality were recorded. Coronary heart disease risk is inversely associated with leg length and with leg length to sitting height ratio, whereas the association with overall height is weak. The components of syndrome X – insulin resistance, high fasting triglyceride levels and obesity – are commoner amongst the men with shorter legs and with low leg length to sitting height ratios. This suggests that development during child-

| Anthropometric, dietary or socioeconomic index (n) | 'z' score for height | 'z' score for leg length | 'z' score for trunk length |
|--------------------------------------------------|----------------------|-------------------------|---------------------------|
| Birth order (1,397)                              | -0.14                | -0.14                   | -0.06                     |
| No of children (1,394)                           | -0.25                | -0.24                   | -0.14                     |
| Weighted per capita food expenditure (1,394)     | 0.31                 | 0.33                    | 0.14                      |
| Social class of head of household (1,287)        | -0.18                | -0.21                   | -0.05                     |
| Overcrowding (1,220)                             | -0.19                | -0.20                   | -0.08                     |
| Relative family per capita calorie consumption (1,394) | 0.23                | 0.26                    | 0.08                      |

Source for data: Gunnell et al, 1997\(^\text{54}\).

| Quintile | CHD mortality | Cancer mortality |
|----------|---------------|------------------|
|          | Fully adjusted relative risk (*95% CI) | Fully adjusted relative risk (*95% CI) |
| Males    |               |                  |
| 1 (shortest) | 2.8 (1.1, 6.9) | 0.4 (0.1, 1.1) |
| 2         | 2.5 (1.0, 6.0) | 0.4 (0.2, 1.1) |
| 3         | 2.2 (0.9, 5.2) | 0.6 (0.2, 1.5) |
| 4         | 2.5 (1.1, 5.7) | 0.8 (0.4, 1.8) |
| 5 (tallest)| 1.0           | 1.0              |
| Linear trend test | \(p = 0.09\) | \(p = 0.06\) |
| Females  |               |                  |
| 1 (shortest) | 4.2 (0.8, 22.2) | 1.0 (0.4, 2.3) |
| 2         | 3.5 (0.7, 17.8) | 1.1 (0.5, 2.4) |
| 3         | 1.9 (0.3, 10.6) | 1.0 (0.4, 2.2) |
| 4         | 0.9 (0.1, 6.6)  | 0.8 (0.4, 2.0) |
| 5 (tallest)| 1.0           | 1.0              |
| Linear trend test | \(p = 0.006\) | \(p = 0.37\) |

* Adjusted for age and indices of childhood and adult socioeconomic circumstances, calorie consumption, and birth order. CHD, coronary heart disease; CI, confidence interval. Source: Gunnell et al, 1997\(^\text{54}\).
hood may be related to the risk of syndrome X, which in turn increases cardiovascular disease risk.

Writing in 1951, Isabella Leitch discussed what happened to animals stunted at birth due to poor nutrition, but well fed during later life. She called the particular animal model she used in her work the low–high pig, stunted in growth and becoming obese ‘rather than finished’ when it is then well fed53. She postulated that this is what would happen to children who were poorly nourished in childhood but given a more calorific diet in later life. Data showing that obesity is more strongly related to childhood than adulthood socioeconomic circumstances give support to this hypothesis45.

Studies of birth weight and cardiovascular disease risk factors suggest that interactions may occur between early life and later life exposures. In a study from Uppsala, Sweden, low birthweight babies who became obese adults had elevated blood pressure and high rates of insulin resistance57. In the Caerphilly study, birth weight was inversely related to risk of coronary heart disease in adulthood58. This association is robust to adjustment for a wide range of conventional cardiovascular disease risk factors. The relationship between birth weight and coronary heart disease risk is, however, restricted to men in the highest body mass index tertile in adulthood59. Similarly, the association between leg length and coronary heart disease risk is strongest among the men who became obese in adult life.

Recent research has demonstrated important interactions between socially patterned exposures in early life—such as low birth weight and poor growth—and later life exposures, reflected in obesity levels. Studies of how factors accumulate and interact throughout life to generate cardiovascular disease risk are in their infancy, but offer to advance our understanding of how social phenomena are translated into socioeconomic differentials in cardiovascular disease risk.

Implications of inequalities in cardiovascular disease risk

The existence of socioeconomic differentials in cardiovascular disease risk provides an important model with which to study the basic causes of cardiovascular disease. Attempts to explain the social patterning of cardiovascular disease should therefore advance our understanding of fundamental issues related to the aetiology and possible prevention of these diseases.

Socioeconomic differentials in cardiovascular disease risk also provide a challenge for social and health policy. Recent trends in coronary heart disease mortality for Scotland according to level of deprivation in the area of residence are shown in Fig 562. Between 1980–2 and 1990–2 the gap has increased. People living in the most affluent areas showed a 40% decline in coronary heart disease (CHD) mortality over this period, while people living in the most deprived areas experienced only a 20% fall. A large increase in the relative and absolute difference in CHD mortality between the poor and rich areas has therefore occurred.

Using data from this Scottish study it is possible to calculate that if, in a district of 300,000 people, everyone had the same mortality rates as people living in the least deprived areas, there would be 600 fewer deaths per year. Calculations based upon the best implementation of the results of randomised trials of treatment of acute myocardial infarction suggest there will be 74 fewer deaths per year in a district of 300,000 people where these treatments are implemented than in a district where nothing is done63. These rough calculations illustrate the relative magnitude of potential benefits from health care interventions and broader social change.

Equitable distribution of health service activities is an important goal. A comparison of angiography rates with coronary heart disease mortality rates provides a way of ascertaining whether people in different areas are being investigated to the same degree for a given level of need. In Scotland, people living in areas of high deprivation are less likely to be investigated with angiography than those living in more affluent areas, once coronary heart disease mortality rates—on an index of need—are taken into account64. The same applies to England and Wales65. Evidence of an inequitable distribution of cardiovascular investigations in relation to need would be greater if private procedures were taken into account.

Inequities in health service interventions generate inequalities in cardiovascular disease risk. These inequalities cannot be simply ascribed to behavioural and lifestyle differences between social groups. Furthermore, even where differences in smoking behaviour, dietary patterns and exercise participation are seen, they should not be considered as simply due to ignorance or fecklessness on the part of people living in materially less favoured circumstances.

To take dietary practices as one example, those least able to purchase a healthy diet due to financial constraints are those most likely to be disadvantaged with regard to access to healthy micronutrient-dense food. Thus a shopping basket survey in Glasgow demonstrated that households in a poorer area paid more for a healthy basket of food than households in a more affluent area, while there was no difference in the cost of an unhealthy basket of food. It was also noted that several items of the healthy food basket were simply not available in the poorer area66. A similar survey was carried out in London in 1988 and repeated in 1995. At both times healthy food was more expensive in the deprived area while unhealthy food was slightly cheaper in the deprived area67. Poorer families have been disadvantaged by changes in food retailing. Between 1980 and 1992 the number of food retail outlets decreased by 35%68. This reflects a decline in the number of small grocery retailers and
specialist shops, including butchers and greengrocers, and an increase in large supermarkets. These tend to be based outside towns and customers require transport to them. The low rate of car ownership among poorer households makes it difficult for them to utilise these generally cheaper outlets. In essence, the transfer of food retailing from smaller local retailers to large out-of-town superstores represents a transfer of costs from the food wholesaler who is required to transport food to fewer outlets to the customer, who must travel further to purchase food. This transfer represents a disproportionate burden to poorer households and contributes to widening inequalities in material circumstances.

Low income households, living in less affluent areas, are disadvantaged in other ways with respect to food, diet and nutrition. Such households may especially value the personal nature of local shopping, given that they have fewer alternative social opportunities. Shopping can become a demoralising experience for those whose choice is constrained by a lack of income. The costs of cooking and of stocking essential items required for food preparation represent additional expenditure which may not be available in less well-off households. Thus the use of convenience foods or items such as sandwiches that require no cooking is encouraged.

Over the recent period, when inequalities in health have been widening, many indicators demonstrate increasing social polarisation. By 1993, one in three children in the UK lived in households with less than 50% of average UK income after housing costs; in 1979 this was less than one in ten. Inequalities in income have increased enormously over the same period, with the income after housing costs of the lowest decile group in 1991 being lower than the equivalent income of the lowest decile group in 1979. This growth in income inequality has gone hand-in-hand with growth in socioeconomic differentials in mortality which includes widening inequalities in deaths from cardiovascular disease. The only economic argument in support of allowing income inequalities to widen is that the incentive of large increases in income for the already wealthy in some

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**Fig 5.** Age- and sex-standardised death rates per 100,000 for coronary heart disease in Scotland, 1980–2 and 1990–2, by deprivation category. Source: McLoone and Boddy, 1994

**Fig 6.** Income inequality around 1980 and labour productivity growth between 1979 and 1990. Source: Glynn and Miliband, 1994
way drives overall economic performance. This doctrine, strongly associated with the Thatcherite agenda of the 1980s, has recently been exploded. Figure 6 plots labour productivity growth between 1979 and 1990 against income inequality in 1980. It is clear that countries with lower levels of income inequality in 1980 had greater labour productivity growth over the following decade. In 1979, the UK lay at around the average of the countries under consideration for both income inequality and labour productivity growth. Since then, inequality has increased in the UK and it is now vying with the USA for the unfavourable title of most unequal country. Tackling socioeconomic inequalities in cardiovascular disease risk involves addressing the processes leading to increasing social inequalities more generally and making firm decisions about the sort of society in which we would like to live.

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