

General explanations for social inequalities in health

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Life expectancy has always differed according to status in society, with a higher mortality among those of lower social status. Although cancer and cardiovascular diseases are more common as causes of death in rich than in poor societies, in industrialized countries the major causes of death are more common in those of lower social status. In this chapter, the magnitude of socioeconomic differences in health is examined using different measures of socioeconomic status, and methodological issues relating to these measures are discussed. Much of the discussion about social inequalities in health has been focused on the health disadvantage of those of lowest socioeconomic status. However, data from the Whitehall studies show that the social gradient in morbidity and mortality exists across employment grades in British civil servants, none of whom is poor by comparison with people in developing countries, suggesting that there are factors that operate across the whole of society. A number of potential explanations are considered here. The magnitude of socioeconomic differences in health varies between societies, and over time within societies. This suggests that identification of factors that influence socioeconomic status and health, and the pathways by which they operate, is an important public health task that could lay the basis for a reduction in inequalities in health.

Inhabitants of poor countries have a shorter life expectancy than those of rich countries. There are exceptions, but low levels of gross national product (GNP) are associated with a high toll from infectious diseases – in childbirth, in the early years of life, in adulthood and in old age. The change from predominantly communicable to non-communicable diseases as causes of death in rich countries led to the notion of diseases of affluence. We now know that this view is misleading. Although cancer and cardiovascular diseases are more common as causes of death in rich compared with poor countries, in industrialized countries the major causes of death are all more common in those of lower social status. Thus, as far as we can tell, life expectancy has always differed according to status in society, even though the causes of death that make up that higher risk have changed. In the past, the task was to explain, and take action to relieve, social inequalities in deaths from communicable disease; now the public health task is to explain, and lay the basis for action on, social differentials in cancer, cardiovascular disease and accidents.

There are at least three reasons to be concerned by inequalities in health. First, governments have

set targets for improvements in health. If improvements occur at a slower rate in large portions of the population, then the remainder must show much greater improvement to keep up the average for society as a whole. Second, reduction in inequalities in health has traditionally been a goal of public health for ethical and moral reasons. Third, the level and distribution of health is an important tangible indicator of the level of well-being in society.

Geoffrey Rose taught that the proportion of 'deviants', or high-risk people, is predicted by the mean (Rose & Day, 1990). Applied to a behaviour such as alcohol intake the implication is that we should concentrate not only on the high-risk people like heavy drinkers, but on shifting the whole distribution. Might this be taken as implying that we should ignore the problem of social variations in health, but concentrate on shifting the distribution? The argument might be that there will always be socioeconomic differences within a society, and so we should not attempt to change the immutable. The response to this is twofold. First, the magnitude of socioeconomic differences in health varies between societies, and over time within societies. This might be either because the

Table 1. Mortality of men aged 15–64 by social class in England and Wales, 1930–1982^a (SMRs)

Social class		Year					
		1930–1932	1949–1953	1959–1963 ^b	1970–1972	1979–1980 ^c	1982–1983 ^c
I	Professional	90		86	75	77	66
II	Intermediate		94	92	81	81	76
III-NM	Skilled non-manual	97		101	100	99	94
III-M	Skilled manual						106
IV	Partly skilled		102	104	103	114	116
V	Unskilled	111	118	127	137	165	

^aSMRs; standardized mortality ratios, all men = 100.

^bAdjusted figures; occupations reclassified according to 1950 classification.

^cMen aged 20–64.

magnitude of socioeconomic differences varies or because the factors influencing the link between socioeconomic status and health vary. This leads to a second response: an aim of research in this area is to understand the reasons for the link between socioeconomic status and health, in order to inform action to change them.

Inequalities or social inequalities?

Differences between individuals in genetic endowment may well contribute to differences between individuals in life expectancy. In the unlikely event that all individuals were subject to the same set of environmental influences they would not all flourish, age and die at the same rate. As Rose has made clear, however, the determinants of individual risks of disease – why one individual gets sick and another remains healthy – may be different from the determinants of population rates of disease (Rose, 1992). We are concerned with social inequalities in health, the reasons for them, and ways of reducing their magnitude. The causes of these social inequalities are likely to be different from the causes of individual differences, where genetic factors will play a bigger role.

Le Grand and Illsley, for example, have focused on individual differences in life expectancy (Le Grand, 1989; Illsley & Le Grand, 1987). They have shown that variability (using Gini coefficients) around age at death has become less, and argue therefore that there is less inequality in society.

This focus on individuals, however, answers a different question from the one we address of differences in health among social groups. If the determinants of variations in health and disease between individuals are different from the determinants of variations among social groups, it is quite possible for there to be no diminution in the relative differences in health between social groups at the same time as there has been a general improvement in health. If fewer people die prematurely, variation in age at death will be less, but this may still be distributed unequally across social groups.

There are other sources of inequality in society, among them gender and race/ethnicity. Gender differences are of great interest and importance but are treated by others. In England and Wales at least, ethnic differences in mortality cannot easily be explained on the basis of conventional social class descriptions (Marmot *et al.*, 1984a). There must be other explanations of ethnic differences; however, these are not reviewed here.

Persistent social differences in mortality within countries

Routine health statistics in England and Wales have been used to demonstrate socioeconomic differentials in mortality since they were first collected. Chadwick reported that in 1842 the average ages at death in several occupationally defined groups were as follows: 'gentlemen and persons engaged in professions, and their families... 45 years; tradesmen

and their families... 26 years; mechanics, servants and labourers, and their families... 16 years' (Chadwick, 1965). Since 1921, mortality data have been available for broad occupation-based social class groups (Pamuk, 1985), providing a unique historical series that demonstrates the persistence of socioeconomic differentials in mortality risk against the background of overall improvement in life expectancy.

Table 1 shows mortality in England and Wales according to the Registrar General's social classes,

for men of working age (Blane *et al.*, 1992, *pers. commun.*). This is the classification that has been standard in British statistics, and is based on classifying occupations according to status and level of responsibility. The standardized mortality ratios (SMRs) allow for comparisons of relative differences in death rates at one period. They show trends over time in relative differences, not in absolute rates. What these data do not show is that the apparent widening of mortality differentials between the 1930s

Table 2. Annual age-adjusted rate of years of potential life lost per 1000 population for deaths from principal causes in England and Wales, 1971 and 1981

Social class	All causes	Ischaemic heart disease ^b	Malignant neoplasms ^c	Accidents and violence ^d
1971 (Men aged 15-64)				
I	48	14	12	10
II	52	15	12	11
III-NM	63	19	14	11
III-M	66	18	16	13
IV	77	19	17	19
V	101	21	19	28
1981 (Men aged 16-64)				
I	37	12	11	10
II	42	14	12	11
III-NM	53	18	14	11
III-M	58	20	16	16
IV	68	21	17	20
V	103	29	24	35
1971 (Women aged 15-64)				
I	29	2	13	4
II	30	2	13	5
III-NM	35	3	14	5
III-M	39	4	15	3
IV	42	4	15	5
V	53	5	17	8
1981 (Women aged 16-59)				
I	13	1	8	3
II	15	1	8	3
III-NM	18	1	9	4
III-M	18	2	9	3
IV	22	2	10	4
V	28	3	12	6

^aFigures were standardized to total population of England and Wales, 1981.

^bICD (International Classification of Diseases) codes 410-414, 9th edn.

^cICD codes 140-209, 9th edn.

^dICD codes 800-999, 9th edn.

Table 3. Age-adjusted mortality in 10 years (and number of deaths) by civil service grade and cause of death

Cause of death (ICD code)	10-year mortality % (number of deaths)				Relative mortality ^a				X-test for trend	
	Administrators	Professional/ executive	Clerical	Other	Administrators	Professional/ executive	Clerical	Other		
Lung cancer (162-1)	0.35 (3)	0.73 (79)	1.47 (53)	2.33 (59)	0.5	1.0	2.2	3.6	54.62	
Other cancer (140-239) (162-1)	1.26 (12)	1.70 (195)	2.16 (73)	2.23 (46)	0.8	1.0	1.4	1.4	7.08	
Coronary heart disease (410-414)	2.16 (17)	3.58 (399)	4.90 (160)	6.59 (128)	0.6	1.0	1.4	1.7	38.24	
Cerebrovascular disease (430-435)	0.13 (1)	0.49 (51)	0.64 (23)	0.58 (14)	0.3	1.0	1.4	1.2	1.70	
Other cardiovascular (404, 420-429, 440-458)	0.40 (4)	0.54 (58)	0.72 (24)	0.85 (24)	0.9	1.0	1.4	2.0	6.95	
Chronic bronchitis (491-492)	0.0 (0)	0.08 (8)	0.43 (15)	0.65 (13)	0	1.0	6.0	7.3	21.01	
Other respiratory (460-490, 493-519)	0.21 (2)	0.22 (24)	0.52 (18)	0.87 (15)	1.1	1.0	2.6	3.1	11.99	
Gastrointestinal disease (520-577)	0.0 (0)	0.13 (15)	0.20 (7)	0.45 (15)	0	1.0	1.6	2.8	6.26	
Genitourinary disease (580-607)	0.09 (1)	0.09 (10)	0.07 (2)	0.24 (6)	1.3	1.0	0.7	3.1	2.46	
Accident and violence (800-949, 960-978)	0.0 (0)	0.13 (17)	0.17 (5)	0.20 (3)	0	1.0	1.4	1.5	1.36	
Suicide (950-949, 980-989)	0.11 (1)	0.14 (18)	0.15 (4)	0.25 (4)	0.7	1.0	1.0	1.9	0.97	
Other deaths	0.0 (0)	0.16 (18)	0.26 (9)	0.40 (6)	0	1.0	1.9	2.0	4.18	
Causes not related to smoking^b										
Cancer	0.86 (9)	1.24 (145)	1.53 (50)	1.57 (33)	0.8	1.0	1.3	1.4	4.70	
Non-cancer	1.00 (10)	1.94 (216)	2.76 (93)	4.19 (82)	0.6	1.0	1.5	2.0	31.83	
All causes	4.73 (41)	8.00 (892)	11.67 (393)	15.64 (326)	0.6	1.0	1.6	2.1	144.05	

^aCalculated from logistic equation adjusting for age.
^bAll causes less 140-141, 143-149, 150, 157, 160-163, 188-189, 200, 202, 410-414, 491, 492.

and the 1980s took place against a background of falling death rates.

One of the problems with the SMR as a way of controlling for age is that it weights more heavily mortality at older ages. Higher relative differences at younger ages would be masked by this technique (see below). An alternative weighting strategy is to use years of potential life lost (Blane *et al.*, 1990). This gives more weight to deaths occurring at younger ages. Table 2 shows the striking social gradient in potential years of life lost. For men up to age 64, neoplasms and ischaemic heart disease account for about the same number of years of potential life lost. For women, neoplasms are more important.

A second implication from Table 2 is that there is a social gradient in the three major causes of death shown. This is shown in more detail with data from the Whitehall study of British civil servants (Table 3) (Marmot *et al.*, 1984b). Grade of employment is used as the marker of socioeconomic status. There is a social gradient for all the major causes of death. The magnitude of the gradient may vary from one cause to another; it is steeper for chronic respiratory disease and lung cancer, which are strongly related to smoking, but the gradient is also present in diseases not related to smoking.

Potential problems in interpretation

Apart from the difficulty of international comparisons, there are several potential problems in interpreting the national data from England and Wales: lack of comparability between numerator and denominator; changing class composition; doubt about the appropriateness of the Registrar General's social classes; doubt about the appropriateness of the classification for women; and whether these differences in mortality are an effect of belonging to a certain class or whether degree of healthiness, or lack of it, determine both the occupational class and mortality risk – that is, there is selection on the basis of health.

The numerator/denominator argument arises because the death records are not linked to census data that supply the estimates of population at risk (Office of Population Censuses and Surveys, 1978). The recording of occupation, and hence assignment to social class, at death may differ from that at the census, leading to biased estimates of mortality ratios. While this argument may apply

to data in the Registrar General's supplements on occupational mortality, it cannot apply to longitudinal studies such as the Whitehall study data shown in Table 3 (Marmot *et al.*, 1984b), or the Office of Population Censuses and Surveys (OPCS) longitudinal study, which follows a 1% sample of the 1971 census. Comparison between the longitudinal study and the 'cross-sectional' approach to examining social inequalities showed similar relative differences between the classes (Office of Population Censuses and Surveys, 1978; Goldblatt, 1990).

A version of the numerator/denominator bias was raised as casting doubt on time trends. It was argued at the time of production of the 1979/83 *Decennial Supplement* that the size of the numerator/denominator bias may have changed between 1971 and 1981 (Office of Population Censuses and Surveys, 1986). If, for example, some people whose occupation would have been classed as unskilled manual (class V) in 1971 had their occupation classified as semiskilled (class IV) in 1981, this would reduce the apparent size of the denominator in class V. If a similar change did not apply to coding of occupation at death, the numerator in class V would not be reduced in the same way. This could account for the apparently increasing relative mortality disadvantage in class V, with the SMR increasing from 137 to 165.

One way of addressing this issue was simply to combine the three non-manual classes and the three manual into two groups (Marmot & McDowall, 1986). It is quite unlikely that there would be misclassification from manual to non-manual. The results are shown in Figure 1. They confirm that for total mortality, and for lung cancer, coronary heart disease, and stroke, the social differential in mortality has widened. Unlike the SMRs shown in Table 1, these have been calculated using the 1979–1983 rates as standard. This allows comparison of absolute as well as relative changes in mortality over time. It shows that the mortality of both nonmanual and manual classes has declined over the 10-year period, at the same time as the gap between them has widened.

Similar changes have been shown in Finland (Valkonen *et al.*, 1990). Using linked data – that is, death records linked to census information – Valkonen showed that there was a clear inverse association between an occupation-based measure of social class and mortality. Furthermore, these

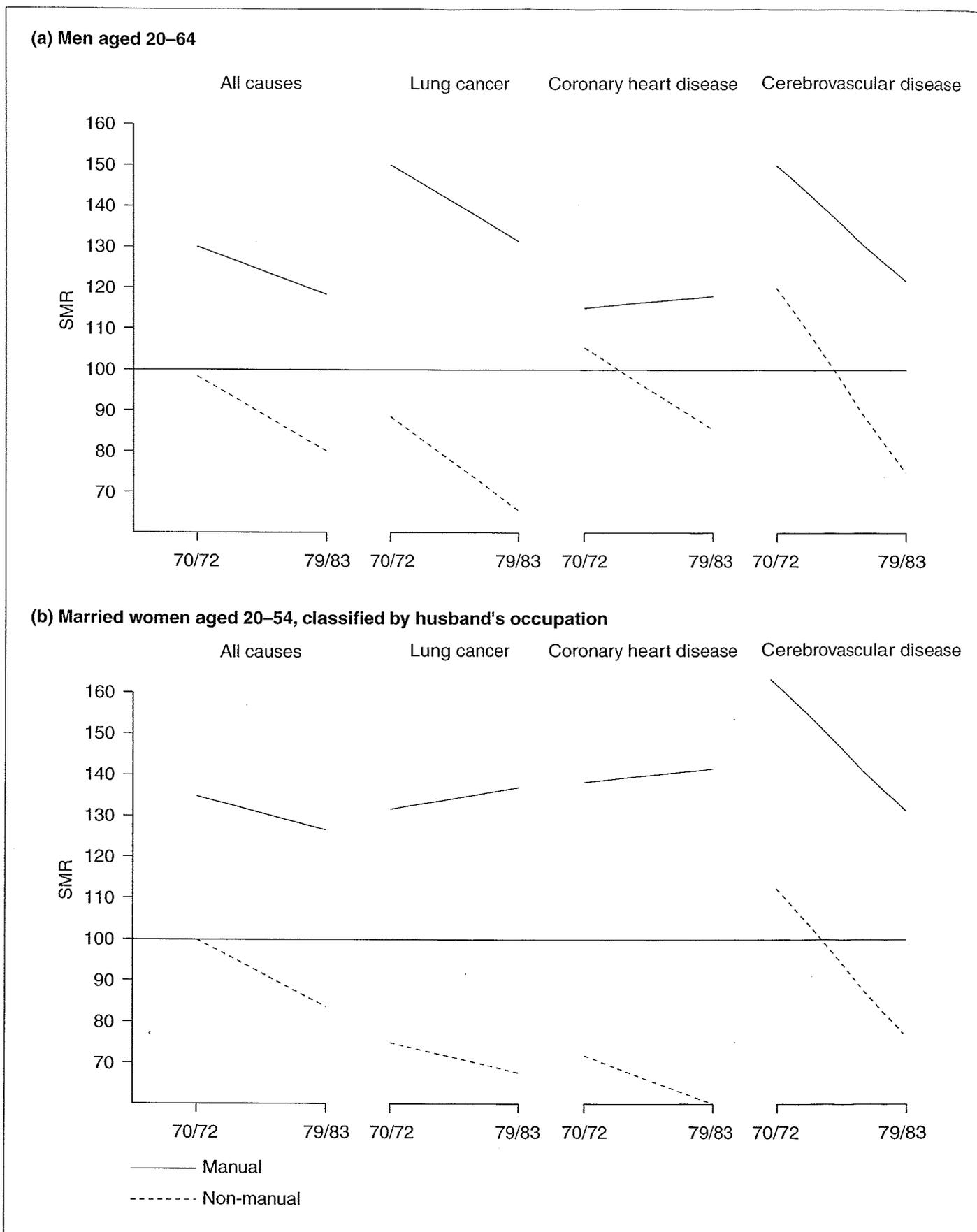


Figure 1. Changes in SMRs by manual and non-manual groups for selected diseases in Great Britain from 1970–1972 to 1979–1983 (Marmot & McDowall, 1986).

differences increased over the period 1971–1985 (Figure 2).

Figure 1 also addresses the question of changing class composition (Illsley, 1986; Strong, 1990). Illsley argues that the relatively high death rates of social class V apply to a decreasing proportion of the population and the low death rates of class I to an increasing proportion (Illsley, 1986). It is therefore difficult to state that inequalities have increased or even changed in magnitude over time. This applies less to a comparison of manual and non-manual classes. In 1971, 60% of men were in manual occupations; in 1981, 55% were. The widening gap in mortality applies therefore to groups of nearly similar size.

Pamuk dealt with changing class composition and changes in classification over a longer period by reclassifying occupations and constructing an index of inequality that takes into account both relative mortality and relative size of classes (Pamuk, 1985). Her conclusion was that class inequality in mortality in England and Wales narrowed in the 1920s and increased again during the 1950s and 1960s, so that by the early 1970s it was greater than it had been in the early part of the century both in absolute and relative terms.

The issue of the extent to which this social gradient in mortality could be produced by selective social mobility will be dealt with more fully below under 'Explanations'. The issue of the appropriateness of the classification for women is part of a more general discussion on measures of social position.

Measures of social differences in mortality

Occupation-based measures of class

The usual British approach to social class analysis, using the Registrar General's classification of occupations, has come in for criticisms additional to those discussed above (Bunker *et al.*, 1989; Illsley, 1986; Strong, 1990). The measure is said to lack theoretical content (it is not clear what it signifies); it may not apply to people not at work; people younger than working age have to be classified by parents' occupation, and those older, or otherwise unemployed, by their previous employment; and women may not be well classified, especially housewives.

These issues have been comprehensively reviewed by the OPCS longitudinal study (Goldblatt, 1990). Goldblatt reminds us that the classification was

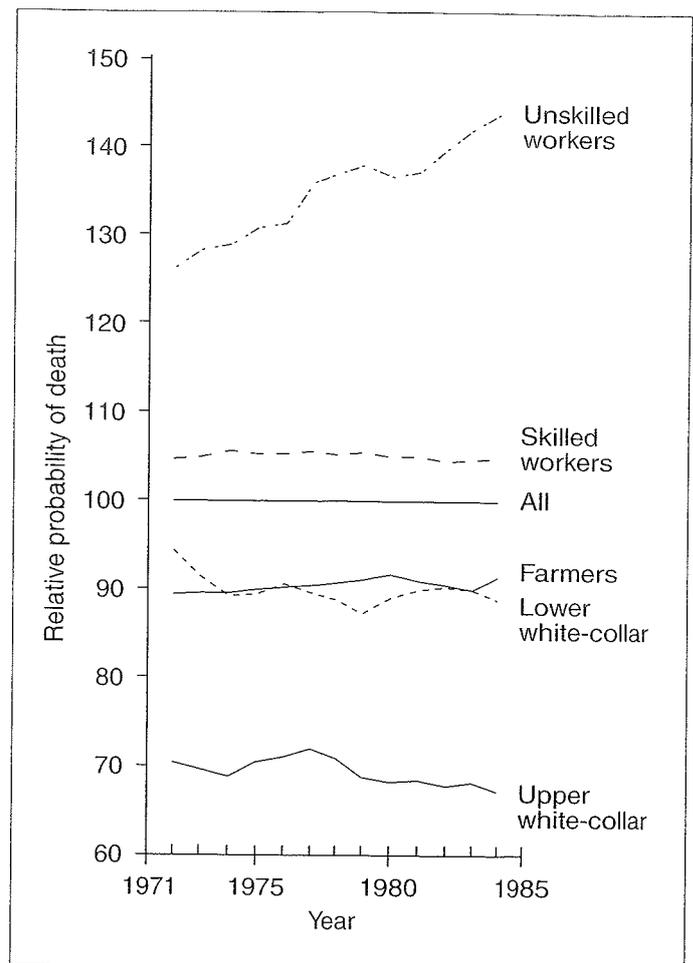


Figure 2. Relative probability of death by occupational class in Finland of men aged 35–64 (all men = 100; three-year moving averages) (Valkonen *et al.*, 1990).

developed, by Stevenson for the *1921 Decennial Supplement on Occupational Mortality* (Stevenson, 1928), to indicate way of life. He quotes Stevenson (Goldblatt, 1990):

Classification of individuals by income was not possible under present conditions in this country, though it had been employed on a very limited scale in America. Estimation of poverty by housing conditions was very unsatisfactory, as bad housing was only one of the handicaps of poverty, so that it was impossible to determine how far the excess of mortality associated with bad housing was due to poverty and how far to the direct effects of overcrowding, etc. Even if full details of income were available, these in themselves would not provide an ideal basis for classification, as it was probably the cultural associations of wealth which promoted longevity rather than wealth itself... The method

Table 4. Mortality in 1976–1981 of men aged 15–64 by alternative social classifications

Social classifications	SMR	(%) ^a	SMR	(%) ^a	SMR	(%) ^a
Occupation-based						
Social class						
I	67	(5)	75	(24)	84	(35)
II	77	(20)				
III-NM	105	(10)				
III-M	96	(37)				
IV	109	(17)	114	(24)	103	(61)
V	125	(7)				
Other	189	(4)				
Household-based						
Private households						
Tenure						
Owner-occupied	85	(51)	114	(47)		
Privately rented	108	(16)				
Local authority	117	(31)				
Car access						
Two or more	77	(15)	87	(65)		
One	90	(50)				
None	122	(33)				
Non-private households	162	(2)				
All men aged 15–64	100	(100)				

^aFigures in parentheses are the percentage of expected deaths attributed to each group.

advocated for meeting the conditions to be considered was that of inferring social position from occupation. By this means regard could be paid to (average) culture as well as income.

In this discussion are the seeds of arguments that continue to flourish: the extent to which we should be seeking measures that reflect material well-being and those that reflect lifestyle – ‘culture’ in Stevenson’s terms; and the degree to which we want a measure that reflects poverty or one that is related more generally to social position that includes those not in poverty.

Social class and household measures of social circumstances

The classification based on occupation has served well the task of predicting differences in mortality across the spectrum of society, as shown in Table 1. The OPCS longitudinal study has explored the use of this and other methods of social classification

based on the material conditions of households: housing tenure, and access to cars. All of these measures are strongly related to household income. The relation of these measures to mortality for men aged 15–64 is shown in Table 4 (Goldblatt, 1990). Social class, housing tenure and access to cars all predict mortality.

One of the criticisms of current analyses of social class is that the extremes of mortality apply to small groups of the population. Indeed, Table 4 shows that the SMR of 67 in social class I applies to only 5% of the expected deaths; and the SMR of 125 in class V applies to only 7%. The analysis by access to cars deals with this criticism. The SMR of 122 in those with no access to cars applies to 33% of deaths.

The longitudinal study was also able to deal with the question of applying social classification to people of different ages. Each of the three measures – social class, housing tenure, and access to cars – makes an independent contribution to

the prediction of mortality at working age and each continues to predict beyond working age (Goldblatt, 1990). Lack of access to a car is less predictive at older ages, which is in accord with the decline in the percent of people with access to a car beyond age 75.

Applying these measures to women

Using the Registrar General's social classes to examine mortality of women is more problematic. Married women who are not employed outside the home are, provocatively, classed as 'unoccupied' and are therefore difficult to assign to a social class. In addition, if occupational class, in Stevenson's terms, is supposed to provide a guide to culture as well as income, a married woman's occupation will provide only part of the picture. It will more often be the case that the family circumstances are misclassified if only the woman's occupation is known than if only the man's is known. Figure 3 shows, for married women, that social class based on husband's occupation predicts mortality better than social class based on the woman's own occupation (Moser *et al.*, 1990). As can be seen from the figures showing the proportion of expected deaths, approximately 41% of deaths are expected in women who are 'unoccupied' (mostly housewives). Among these women, husband's social class predicts mortality more strongly than among other women. For single women, own social class is a powerful predictor. For example, the SMR among women in non-manual occupations is 81, and among manual occupations it is 156.

Housing tenure and access to cars predict mortality at working ages, as well as among older women. One advantage of these measures for use among women is that they do not require the distinction between married and unmarried women (Goldblatt, 1990). They also, as with men, distinguish mortality differences among groups that make up large proportions of the total population.

Education

Another approach to measurement of the social gradient in mortality is to take education as a predictor. Valkonen (1989) has used this to compare socioeconomic differences in mortality in different European countries in the 1970s (Figure 4). For men, the slope of the relation is remarkably similar in Scandinavian countries, in England and Wales, and

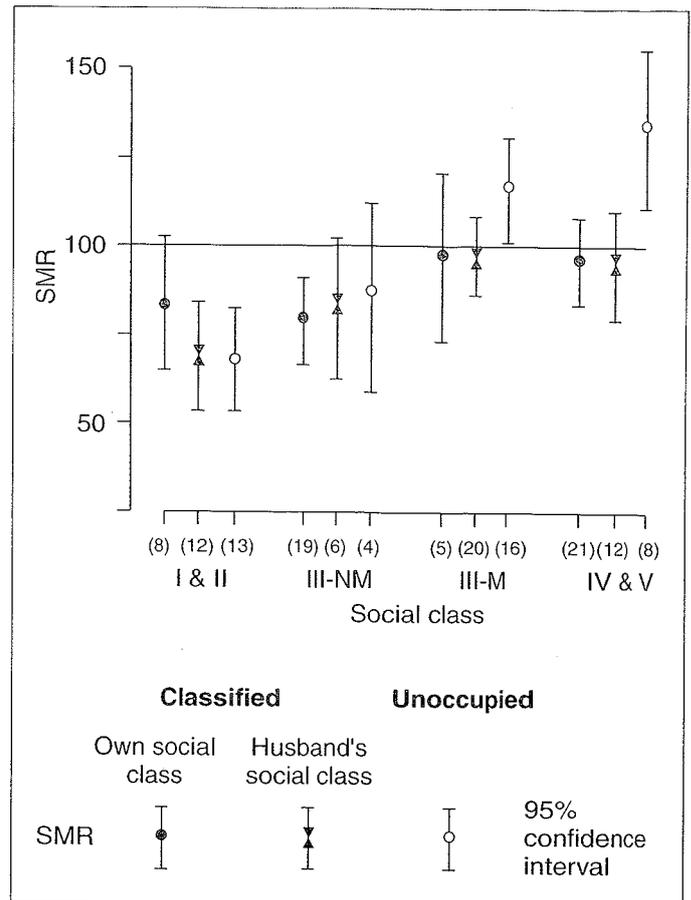


Figure 3. Mortality in 1976–1981 among married women aged 15–59; by own social class and by husband's social class for those classified to an occupation, and by husband's social class for those who were 'unoccupied' in 1971. Figures in parentheses represent expected deaths associated with each SMR (standardized mortality ratio) (as a percentage of the total for all married women). Data are from the OPCS longitudinal study (Moser *et al.*, 1990).

in Hungary. The greater the number of years of education, the lower the mortality rate. The general relationship is similar among women, but the slope of the lines varies. As with the discussion on women's social class based on occupation, so with education; a woman's material income and wealth may be determined not only by her own characteristics but also by those of her husband. The degree to which this is so may vary from country to country and hence account to some extent for the variations in slopes.

Valkonen points out that the apparently fixed nature of the relation between education and mortality for men should not be taken as a general rule. The slope became steeper in England and Wales from 1971–1975 to 1976–1981. This is consistent with the findings for occupational social class shown in Figure 1.

Similar findings come from the United States of America. Kitagawa and Hauser (1973) showed a strong and consistent inverse association between education and mortality in the 1960s in the USA. More recently, Pappas *et al.* (1993) showed that the decline in mortality in the USA varied substantially by education: the greater the number of years of education, the steeper the decline.

Area-based measures of material circumstances

A quite different approach to measuring inequalities in health is to classify not individuals but areas. To some extent this has been used as a proxy for individual-based measures, where these may not be available. The use of these measures has also been justified on theoretical grounds. Townsend, one of the authors of the Black report, developed census-based measures of social deprivation precisely to examine the effect on mortality of material circumstances (Townsend *et al.*, 1988). His index of deprivation comprised the proportion of households that had access to cars, the percentage of unemployed, the percentage of owner-occupiers, and the degree of crowding. In the Northern Region of England, this measure was strongly related to mortality – the greater the deprivation the greater the mortality (Townsend *et al.*, 1988). A similar measure of deprivation in Scotland was strongly related to area differences in mortality (Carstairs & Morris, 1991).

We have applied the Townsend measure of deprivation to census tracts (population 7000, approximately) throughout England (Eames *et al.*, 1993). The relation of deprivation scores to all-cause mortality is shown in Figure 5. This illustrates the continuous relation between deprivation and mortality – the more deprived tracts have higher mortality. There are alternative ways of interpreting this finding of a gradient in the relationship of deprivation to mortality. One is that deprivation is a misleading term. As with data on occupation-based social class, we are dealing not with the effects of poverty but relative position – a gradient in mortality. Most people living in wards that are classified in the second or third quintiles in Figure 5 are not deprived – yet their mortality rate is higher than that of people in the least-deprived quintile. This interpretation suggests relative deprivation rather than absolute material disadvantage.

An alternative interpretation would be of a dichotomy between deprived and non-deprived –

above the threshold of deprivation, mortality is raised compared with below. This would suggest that each successive quintile contains a greater proportion of deprived households; hence the appearance of a gradient in mortality. It is not easy to distinguish between these two alternatives from ecological-based studies. The studies based on classification of individuals, reviewed above, suggest strongly that the relation between social position and mortality is graded and not a threshold effect.

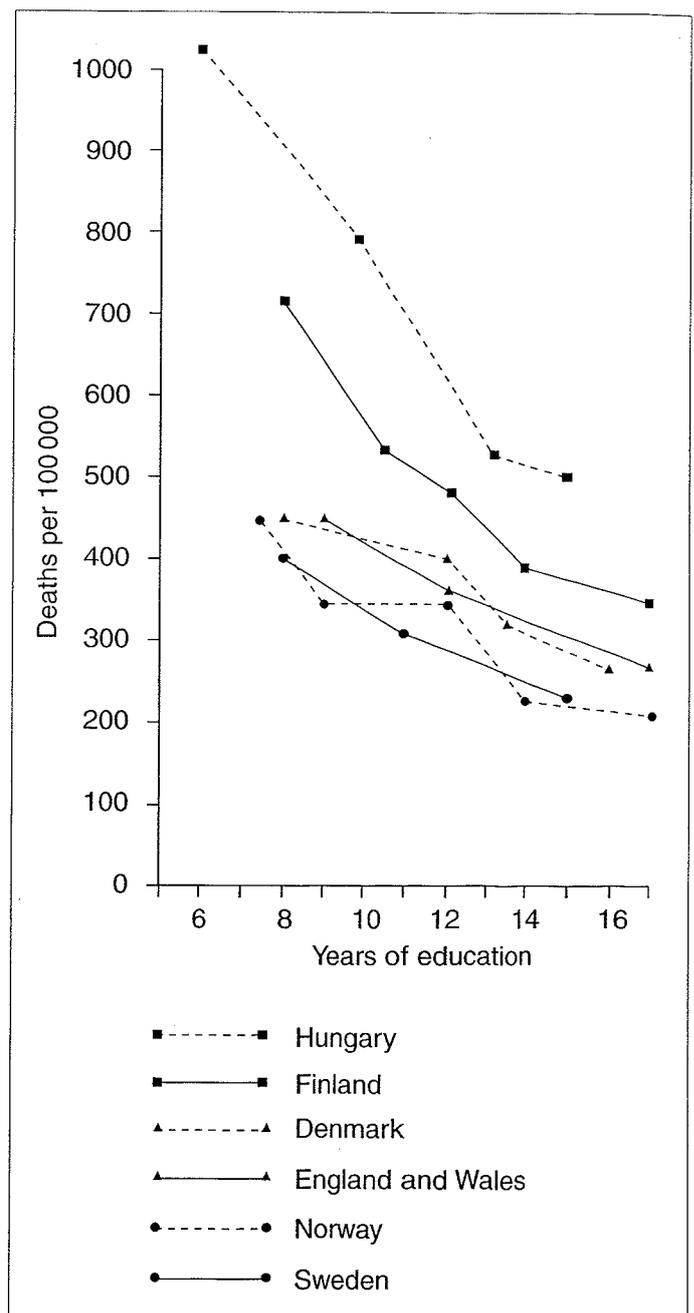


Figure 4. Age-standardized mortality (per 100 000) from all causes of death by years of education and country, for men aged 35–54 (log-scale) (Valkonen, 1989).

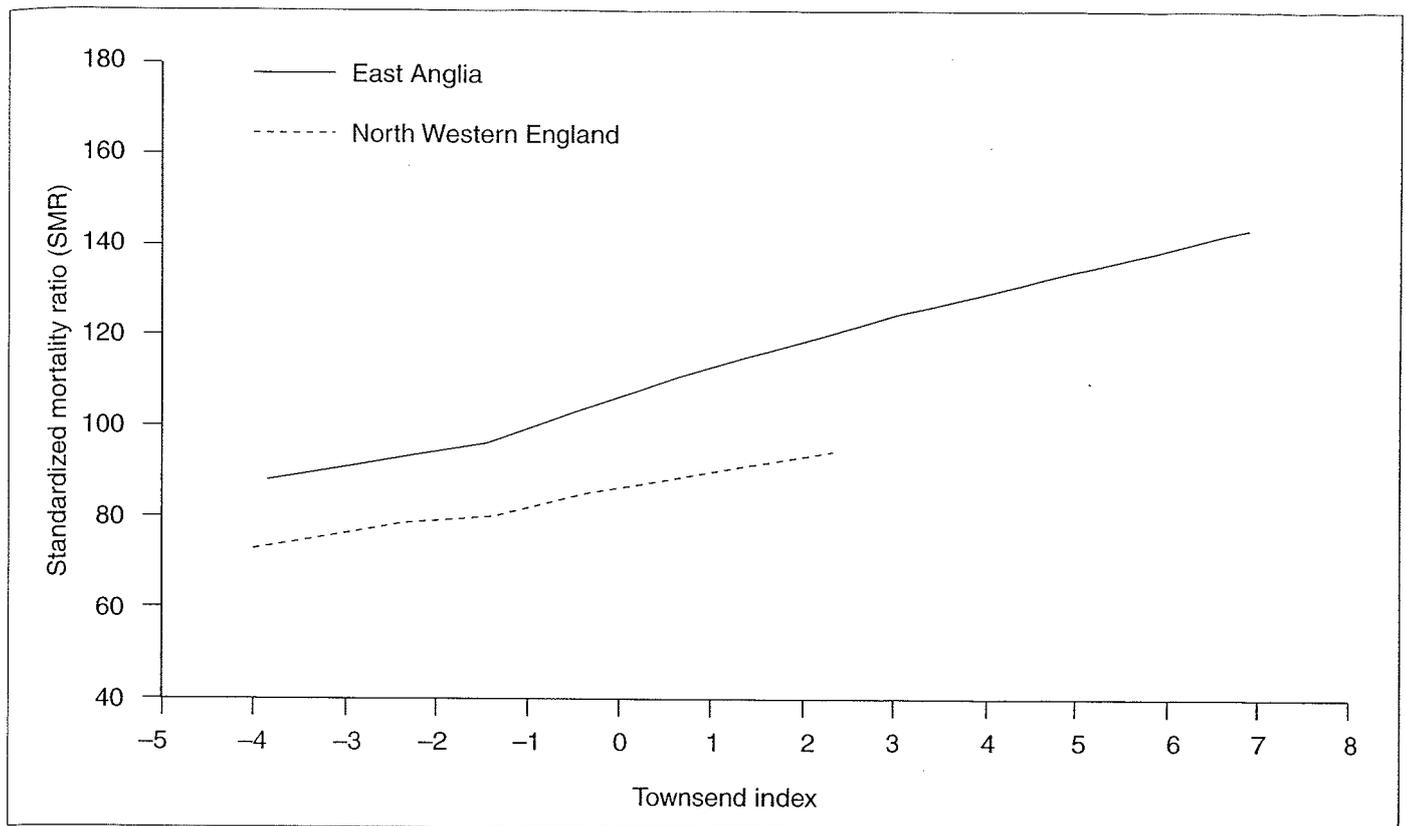


Figure 5. All-cause mortality in two regions of England: North Western England and East Anglia, with mean male mortality by quintiles of the Townsend index of deprivation. The higher the score, the greater the deprivation (Eames *et al.*, 1993).

A second observation from Figure 5 relates to regional differences in mortality within England. The North Western Region has higher mortality than East Anglia and a greater spread of deprivation, yet at comparable levels of deprivation, the regional difference in mortality persists. In addition, the slope of the relation between deprivation and mortality appears to differ between regions. This suggests that the 'meaning' of the deprivation index differs depending on the context, or that some other factor(s) modifies the effect of deprivation on mortality within regions, and contributes to regional differences in mortality independently of deprivation.

These area-based studies may be important not only because they provide a guide to the socioeconomic status of individual residents. The Human Population Laboratory from Alameda County, California showed that people living in a poverty area experienced a higher mortality rate than people living in non-poverty areas, independent of a wide range of personal characteristics including income and health behaviours (Haan *et al.*, 1987).

Choosing between measures

Stevenson based his social class measure on occupation because he wished it to represent status in the community: a mixture of material conditions and culture. Other methods of classifying social position have been discussed. To these we should add income as commonly used in the USA (Bunker *et al.*, 1989). How are we to choose between them? There are two types of criteria: prediction and explanation.

We consider prediction first. The work of the OPCS longitudinal study, discussed above, shows how household measures – housing tenure and access to cars – predict mortality independently of social class and are more appropriate for married women. Education also predicts mortality. As Valkonen *et al.* (1990) discusses for Finland, as the majority of the middle-aged population falls into one basic education category, it distinguishes better among higher status groups than it does among lower status. A similar consideration limits the use of education in census-based studies in Great Britain (Goldblatt, 1990).

The pragmatic question of the best socioeconomic predictor will be answered differently

depending on circumstances. In the special circumstances of the Whitehall study, for example, employment grade provides a precise social classification that is a powerful predictor of mortality. This particular measure is highly correlated with salary and material conditions such as housing tenure and access to cars (Davey Smith *et al.*, 1990) and with 'culture', as well as with years of education.

This raises the more difficult question of explanation. One should be wary of the temptation of using a standard multivariate analysis as a way of determining which of a number of socioeconomic indicators is most important in the causal network (Marmot, 1989). For example, suppose mortality were analysed in a statistical model in which the predictors (independent variables) included education, social class (based on occupation), housing, cars, income, and area-deprivation score. Years of education might show the strongest prediction

simply because it was measured more precisely than the others. One should be wary of concluding that education holds the best prospect for intervention to reduce social inequalities. Education may be a precise marker of social position and hence good as a predictor, but may not itself be a determinant of health status. The question of explanations will be considered at length below.

Poverty or inequality

Much of the discussion about social inequalities in health has related to the health disadvantage of those at the bottom (M'Gonigle & Kirby, 1937). This is analogous to seeing social problems as a particular problem for a disadvantaged minority. There is little doubt that poverty, or deprivation, is likely to be bad – for health among other things. The Whitehall data on mortality (Table 3) suggest that something other than absolute poverty is at

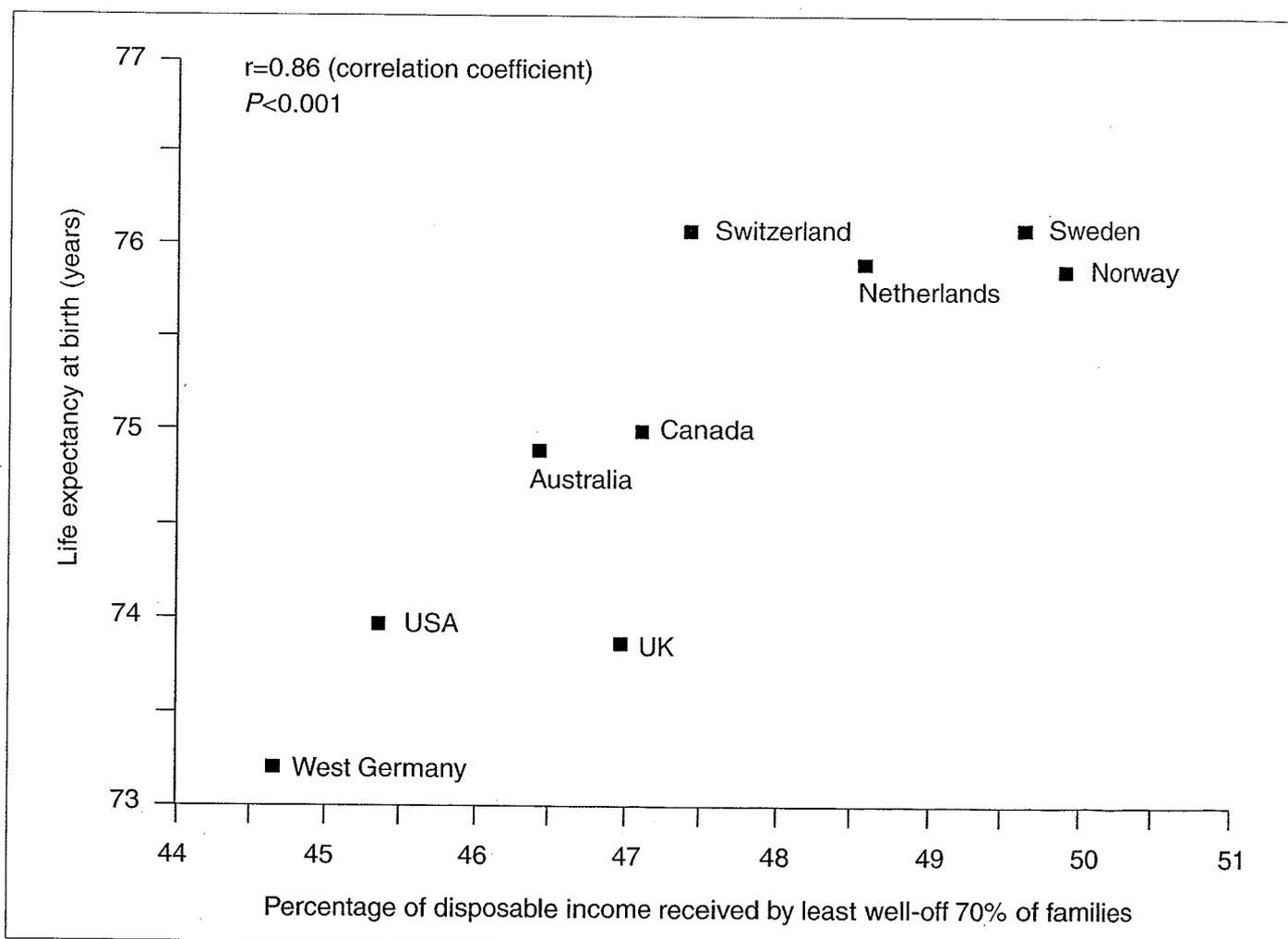


Figure 6. Relationship between life expectancy at birth (male and female combined) and percentage of post-tax and post-benefit income received by the least well-off 70% of families in a group of OECD countries in 1981 (Wilkinson, 1992).

work here. Each grade has worse health and higher mortality rates than the grade above it. Executive-grade civil servants are not poor by any absolute standard yet they have higher mortality rates than administrators. Even clerical officers who are far from well-off, with earnings at or below the national average, are not poor by comparison with people in England at an earlier period in history, or with those in developing countries.

This social gradient in mortality suggests the operation of factors across the whole of society. Whether they are relative deprivation or relative lack of access to the fruits of a wealthy society, it is clear that explanations for socioeconomic differentials in Britain in the 1990s must be broader than the notion of poverty advanced earlier in the century (M'Gonigle & Kirby, 1937).

As with the above discussion of social inequalities in mortality, international data suggest conclusions that are congruent with those from within-country comparisons. Wilkinson (1992) argues that, comparing countries, there is a relation between GNP per head and life expectancy at birth only for poor countries: in 1984, below a threshold of GNP per head of about US\$ 5000, few countries had inhabitants with a life expectancy of 70 years or more. Beyond that level, however, there is little relation between GNP and life expectancy (Wilkinson, 1986). The relation of life expectancy with measures of income dispersion is much closer. Figure 6 uses as a measure of equality of income distribution the share of total post-tax household income received by the least well-off 70% of families. There is a striking correlation with life expectancy. Wilkinson (1992) tried several measures of income distribution – the share of total income received by the least well-off 10%, 20%, 30% and so on. He found that the correlation with life expectancy increased progressively until the bottom 60–70% was reached – that is, the higher the share of total income enjoyed by the bottom 60–70%, the longer the average life expectancy.

As a further test of the income inequality hypothesis, Wilkinson plotted changes in life expectancy against changes in income distribution for six countries for which these data were available. Japan had the greatest increase in equality of income distribution and the greatest increase in life expectancy; and Japan now has the most equitable distribution of income and the longest life ex-

pectancy of any country within the Organization for Economic Cooperation and Development (OECD). By contrast, in the United Kingdom the bottom 60% of households had a declining share of total income and the population had a relatively small increase in life expectancy. (World Bank, 1992).

How are we to explain the relation between income inequality and life expectancy? One possibility is that greater income inequality implies a greater proportion of the population in poverty. If there were a non-linear relation between income and life expectancy – stronger at lower incomes than at high – the apparent relation between income inequality and life expectancy could possibly be explained by the differing proportions of those in poverty. Wilkinson (1992) points out that this is not the most likely explanation. If the criterion of income inequality is taken as the share of income received by the bottom 10%, the relation with life expectancy is very much weaker than the criterion of the share received by the bottom 60 or 70%. Even if increased income inequalities did result in more households in poverty, the proportion of absolute poor in OECD countries is still too small to account for the size of the relation between income inequality and life expectancy. There must be an effect of income inequality on the large bulk of the population. In other words, the main influence on life expectancy among rich countries is likely to be of relative rather than absolute deprivation.

Explanations of social inequalities in health

Medical care

A first attempt at explanation for social inequalities in health might be inequity in the distribution of medical care. Indeed, the establishment of the Black Committee in the United Kingdom was in response to the apparent failure of social inequalities in health to have disappeared 30 years after the establishment of the National Health Service. Congruent with the conclusion of McKeown (1979) on the limited role of medical care in contributing to improvements in life expectancy, the Black Committee did not attribute inequalities in health to inequity in the distribution of medical care.

One way this issue has been examined is to look at mortality from causes judged to be amenable to medical care. Figure 7 extracts data from a report by Mackenbach *et al.* (1989). It shows that the

relative decline in mortality from causes amenable to medical care may have been slightly greater in social class I than in class V – consistent with inequity in medical care. Amenable causes are, however, a small proportion of the total. In absolute terms the widening gap in mortality between the classes is clearly the result of the decline in non-amenable causes in higher classes and the lack of such decline in lower classes.

Within Great Britain, an analysis of data from the OPCS longitudinal study shows that social class differences in cancer mortality are largely the result of differences in the incidence of cancer. Differentials in survival make a minor contribution to overall cancer mortality (Kogevinas *et al.*, 1991).

Analogous to the limited role of differences in medical care in generating social inequalities in health is the limited role of variations in medical care in generating international differences in health. When comparing Japan with England and Wales, for example, we noted that Japan, like the United Kingdom, spends a relatively small proportion of GNP

on medical care. The decline in mortality in Japan was observed for both amenable and non-amenable causes of death (Marmot & Davey Smith, 1989).

Access to good-quality medical care is a right that should be enjoyed equally by all members of society. It is hard to make the case that it is differential access to or provision of medical care that is responsible for inequalities in health in European countries.

Health selection

The argument here is, in essence, that health may determine social position rather than vice versa. This was one of the possible explanations considered by the Black report and rejected as a major cause of social inequalities in health (Black *et al.*, 1988). There are several periods during the life course when selection could operate and also several potential mechanisms, with a varying degree of plausibility and evidence supporting these interpretations (Blane *et al.*, 1993).

The most straightforward suggestion is that the sick drift down the social hierarchy, producing

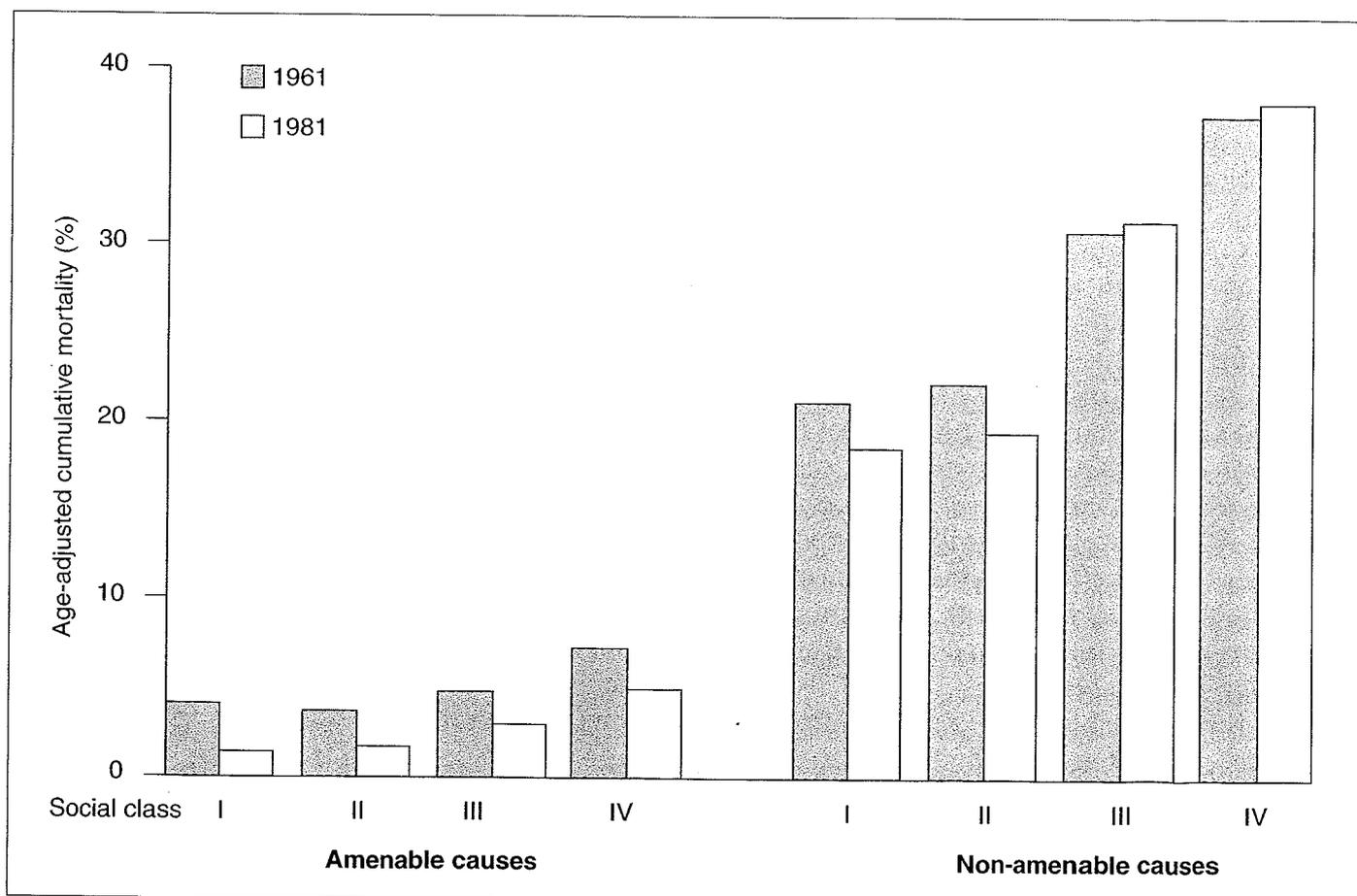


Figure 7. Mortality from amenable and non-amenable causes by social class in England and Wales in men aged 15–64 in 1961 and 1981 (Mackenbach *et al.*, 1989).

social groups at the bottom containing a disproportionate number of individuals at high mortality risk. Three pieces of evidence bear on whether such intragenerational selection is an important contributor to mortality differentials. First, in the OPCS longitudinal study, social class mortality differentials after 1981 for subjects who were in the same social class groups in 1971 and 1981 – and could therefore not have experienced health-related social mobility – were identical to those for the whole population (Goldblatt, 1989). Second, in this study, mortality differentials according to the Registrar General's social classes persisted beyond age 75, long after retirement (Fox *et al.*, 1985). Here social class is based on last occupation. By definition, health after retirement cannot cause downward social drift as the classification is fixed. Third, in participants free of manifest disease at entry to the Whitehall study, mortality differentials by employment grade were essentially the same as in the whole study population. As no reclassification of social position after study entry was made, mobility caused by differences in health status, at least as measured, cannot account for the mortality gradient (Marmot *et al.*, 1978).

Recently, there has been much interest in the possibility that selection occurring at an earlier age – between early childhood and labour market entry – is an important determinant of health inequalities (Illikey, 1986; West, 1991). This could occur in two ways: if health status in childhood determines both health and social class in early adulthood; or if a common antecedent determines both adult health status and future adult social class. This second possibility is considered in the next section. There is evidence that ill health in childhood is associated with downward social mobility (Wadsworth, 1986), but the effect is of minor importance. Other studies that can relate health in childhood to health in early adulthood similarly suggest that this cannot account for class differences in health in adulthood (Power *et al.*, 1990; Lundberg, 1991).

Factors operating early in life

A different version of the selection hypothesis suggests that while social selection based on health status is not a crucial contributor to health differentials, common background factors determine both social position and health in adulthood.

This process has been termed 'indirect selection' (Wilkinson, 1986), and recognizes that people bring with them into adulthood the results of influences from their earlier days: genetic factors, biological results of early experiences, and educational, cultural, psychological and social factors.

An interesting body of work suggests that non-genetic factors operating *in utero* or in the first year of life may have long-term effects on risk of cardiovascular and other diseases (Barker, 1989). Barker's work showed that birth weight, weight at one year and thinness at birth were related to a variety of indicators of cardiovascular disease and diabetes. These associations appear to be independent of parents' social class. It is possible that both social position and health in adulthood are determined by common early life influences. The focus on the childhood origins of adult disease has been criticized precisely because influences from early life shape the lives people lead and the social environments in which they live and work (Ben-Shlomo & Davey Smith, 1991). It may be these conditions of adult life that are related to ill health, and the importance of childhood conditions may therefore be indirect. It is clearly not easy to separate the direct effects on health of early and later life experiences.

One indicator that may help is height. In his studies in Aberdeen, Illsley (Illsley, 1955) showed that women who were upwardly mobile – their husband's social class was higher than their father's – were taller than women who married within their class. More recent work, analysing data from the 1958 birth cohort in the United Kingdom, confirms that social mobility between birth and age 23 was selective with respect to height but mobility did not account for the social gradients in height (Power *et al.*, 1990, 1991).

Whitehall data relating height to mortality provide some insight into the possible separate effects of current and past environment. Height is influenced by environment as well as by genes and is related to social status as measured by employment grade. Short height predicts adult mortality independently of grade of employment (Marmot *et al.*, 1984b), and it is reasonable to speculate that this may in part be a reflection of a persisting influence from early life. Grade of employment, which is to some extent an index of current social influences, predicts mortality independently of height. Thus, two sets of influences may affect mortality risk:

factors from early life and current influences. As indicated, this is to oversimplify: people's current social situations are influenced by their prior experiences. Nevertheless it is important to attempt to distinguish these two sets of influences as their relative importance is crucial to determining the appropriate locus for interventions that may both improve overall adult health and reduce the socioeconomic differentials. Research is currently ongoing that may reduce the degree to which views are polarized between those who think future health status is virtually programmed in early life (Barker, 1990) and those who support the current mainstream focus on influences acting in later life (Elford *et al.*, 1991).

It is interesting that short height is a stronger predictor of cardiovascular disease than of all-cause mortality. If short height predicts cancer mortality less strongly than it does cardiovascular disease, might this mean that cancer is less influenced by factors operating early in life?

General susceptibility or specific causes?

A striking feature of social class differences in health is the generalizability of the findings across

diverse pathological conditions. In the Whitehall study, the higher risk of death among lower grades applied to deaths from lung cancer, other cancers, coronary heart disease, cerebrovascular disease, other cardiovascular disease, chronic bronchitis, other respiratory disease, gastrointestinal disease, genitourinary disease, accidents and violence (Marmot *et al.*, 1984b). Findings such as these, suggesting that there may be common factors operating that cut across known causes of disease, have prompted speculation that there may be factors that increase general susceptibility to ill health (Berkman & Syme, 1979; Cassel, 1976).

An alternative to a general susceptibility hypothesis is that a variety of specific factors are operating to explain social class differences in mortality. Support for this view comes from the fact that some cancers, notably those of the colon, brain, prostate, haematopoietic system and breast, and melanoma, do not show the same social class variation as the causes of death listed above (Davey Smith *et al.*, 1991).

When posed with two conflicting alternatives such as these, a reasonable working hypothesis is that they are both correct. A general susceptibility

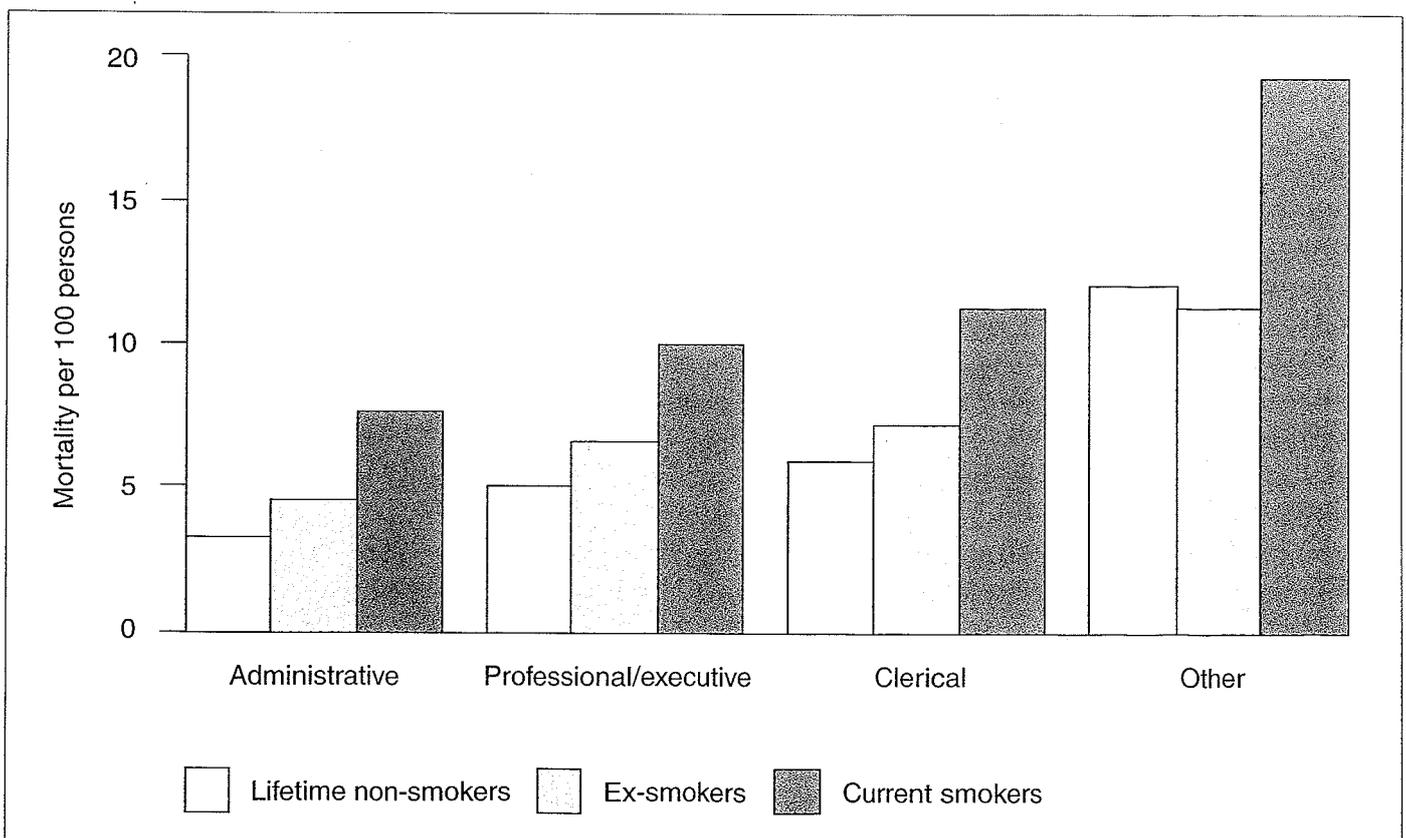


Figure 8. Ten-year mortality risk (age-standardized) by smoking behaviour and employment grade (Davey Smith & Shipley, 1991).

hypothesis implies that certain groups will be at higher risk of death whatever causes are operating. It does not deny the operation of specific causes. Diseases linked to smoking, such as chronic bronchitis and lung cancer, show a particularly strong social class gradient – stronger than cancers not linked to smoking. But the latter do show a social class gradient, as do other diseases not linked to smoking. Put another way, the general susceptibility hypothesis means that there are factors operating that cut across our current system of classifying diseases. These will increase risk of death in addition to the effect of known factors such as smoking. This can account for the fact that an administrator who smokes 20 cigarettes a day has a lower risk of lung cancer mortality than a lower-grade civil servant smoking the same amount, even after pack-years and tar content of cigarettes are taken into account; and for the gradient in mortality that occurs for coronary heart disease even among nonsmokers. Figure 8 illustrates this point (Davey Smith & Shipley, 1991).

The production of this apparently increased susceptibility may well be operating at a social level. The whole life course of people in different social locations is different, and insults to health may accumulate over the entire period from birth to death. That these influences on health cluster in such a way as to produce social groups at differing degrees of disadvantage with respect to most diseases is undeniable. Our current level of knowledge regarding this general susceptibility allows us to go little beyond this empirical observation, however.

Health-related behaviours and biological risk factors

The influential review by Doll and Peto (1981), which became the basis for the USA National Cancer Institute's goals, suggested that much of cancer occurrence could be attributed to behaviours – smoking, diet, alcohol consumption and behavioural response to screening. There are two questions here: how much of the social differential in the occurrence of cancer can be attributed to social differences in behaviours; and why are there social differences in behaviours?

The Whitehall II study established a clear social gradient, using grade of employment as an indicator of socioeconomic position, in a number of health behaviours and established biological risk factors (Marmot *et al.*, 1991). The most striking risk factor difference among grades was in smoking (see

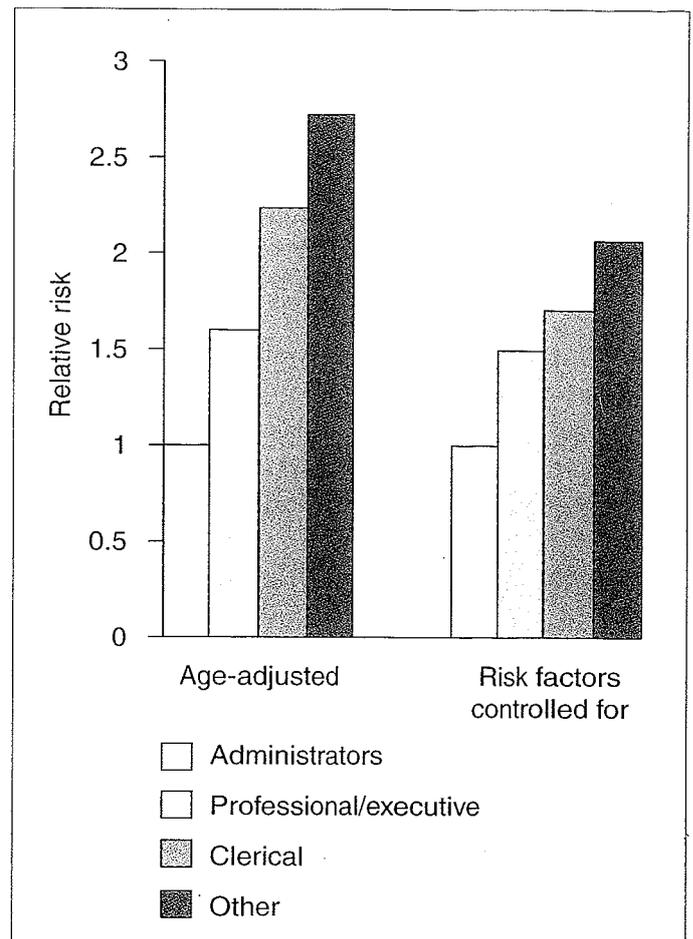


Figure 9. Relative risk of coronary heart disease death in 10 years, controlling for age and risk factors (smoking, systolic blood pressure, plasma cholesterol, height, and blood sugar) (Marmot *et al.*, 1984b).

also Figure 10). Women had a higher prevalence of smoking than men in all but the lowest (clerical and office-support) grade. As a rough indicator of dietary pattern, consumption of skimmed and semi-skimmed milk, wholemeal bread and fresh fruit and vegetables was higher in higher grades. To the extent that dietary intake of non-starch polysaccharides and of antioxidants is protective against cancer, this might provide part of the explanation for the social gradient, along with smoking.

In addition, the proportion of men and women not taking moderate or vigorous exercise in their leisure time was higher in lower grades. Possibly related, there was a significant inverse trend of mean body mass index (weight/height²) by grade but, especially in men, the differences were small. The distribution is different, however. The prevalence of obesity (body mass index >30) was greater in lower grades, strikingly in the clerical grade.

Interestingly, average alcohol consumption was higher among the higher grades of men and, more strikingly, of women. In fact, there were more non-drinkers in the low grades, no difference by grade in the proportion of heavy drinkers, and more moderate drinkers in the high grades (Marmot *et al.*, 1993). This is similar to results from other non-industrial populations – in the Netherlands, for example, women from higher socioeconomic groups were more likely to report higher weekly alcohol consumption than women from lower socioeconomic groups, although negligible differences were observed for men (Mackenbach, 1994). On the face of it, therefore, differences in alcohol consumption cannot be playing a major role in social differentials in cancer risk. It might be that among industrial workers, alcohol consumption may be higher and hence contribute more to higher cancer risk.

Plasma cholesterol levels did not differ by grade, and the small inverse association between grade and blood pressure level in men observed in the first Whitehall study was still present in the second study but was even smaller.

It is easier to quantify the effect these risk factor differences may have on cardiovascular disease than on cancer. The grade differences in smoking were insufficient to account for differences by grade in mortality from smoking-related diseases (Marmot *et al.*, 1984b). There were only small differences in blood pressure between grades, and plasma cholesterol levels were higher in higher grades. The main coronary risk factors could account therefore for little of the gradient in mortality by grade of employment (Figure 9).

To the extent that behaviour does account for social gradients in health, this raises a new question: why the social gradient in behaviour? It is worth dwelling on the implications of the smoking rates in Figure 10. In the baseline examinations for the first Whitehall study, conducted between 1967 and 1969, there was a clear social gradient in smoking. Twenty years later, the prevalence of smoking has declined across the whole of society and in a new cohort of civil servants, different people from those examined 20 years earlier, the social gradient is reproduced.

Social class should not be treated as a confounder. It is reasonable to 'control' for smoking if one wishes to examine the extent to which

smoking accounts for social class differences in disease. Similarly, an analysis of the health risks of smoking will be flawed if it fails to take into account that smoking is associated with social position which, in turn, is associated with adverse health for reasons in addition to smoking (Davey Smith & Shipley, 1991).

To understand the pathways by which social inequalities in health are generated, one needs to examine the links in the chain – for example, between social position and smoking – and not to control for their effects.

Material conditions

The Black report (Black *et al.*, 1988) emphasized the importance of material conditions as an explanation for social inequalities in health. In fact, Black referred to materialist or structural explanations: emphasizing hazards to which some people have no choice but to be exposed given the present distribution of income and opportunity. These can be interpreted as broader than simply material conditions, to include psychosocial influences that are inherent in position in society.

As shown above, in the OPCS longitudinal study, measures other than social class based on occupation predicted mortality, including housing tenure (ownership) and household access to cars. These are clearly a guide to material conditions. In the Whitehall study, car ownership predicted mortality, independently of grade of employment (Davey Smith *et al.*, 1990). In addition, men who reported they engaged in gardening had lower mortality than other men. The link between gardening and lower mortality is open to a variety of interpretations; among them is that possessing a house with a garden is a measure of wealth. These data are consistent with the link between deprivation and mortality shown in the geographic-based studies reviewed above.

The difficulty in understanding material explanations is to know how they operate. At a time when poor living conditions meant polluted water, crowded unsanitary housing with high rates of cross-infection, and appalling conditions of employment, it was not difficult to see how these could be responsible for worse health among the socially deprived. This would be additional to the effects of inadequate diet, which is part of the material conditions of life. As conditions improved,

mortality of all social groups improved. But why do the social gradients persist? Are we to understand that there are residual effects of bad housing with damp and infection, as well as air pollution and other material conditions that, although they affect the lower social groups less than they used to, still affect them more than the higher social strata? If there are such residual effects it is no surprise that they do not affect all social strata equally. Can this be the whole explanation?

The Whitehall data and Wilkinson's data on income inequalities are relevant here. In both Whitehall studies, morbidity and mortality varied linearly with grade of employment. It is possible that the worse health of the second-highest grade compared with the highest could be the result of worse housing, poorer diet for children, or greater pollution, but it seems unlikely that comfortable 'middle-class' people in Great Britain are suffering from the effects of material deprivation. Similarly, comparing rich countries, Wilkinson showed that it was not differences in wealth that predicted differences in life expectancy, but differences in inequality of income distribution.

One is drawn to the view that, in addition to the multiple influences already discussed, there must be an influence of relative position in society. What may be important is not absolute deprivation but relative deprivation. This would account for a social gradient in ill health, because each group, while not necessarily suffering from greater effects of bad housing and so on, will have 'less' than the group above it. This would account for the widespread finding of social inequalities in health in societies with very different levels of health. The social gradient in ill health will vary in magnitude depending on the magnitude of the relative differences in 'deprivation'.

In a society that has met the subsistence needs of its members, what do we mean by 'less' or 'relative deprivation'? In addition to the factors explored above, we should look to psychosocial factors for part of the answer.

Psychosocial factors as potential explanations

This is not the place to review the whole field of psychosocial factors and health. There are a number of ways such factors could influence social differentials in health: characteristics of the psychosocial work environment, low control, and low

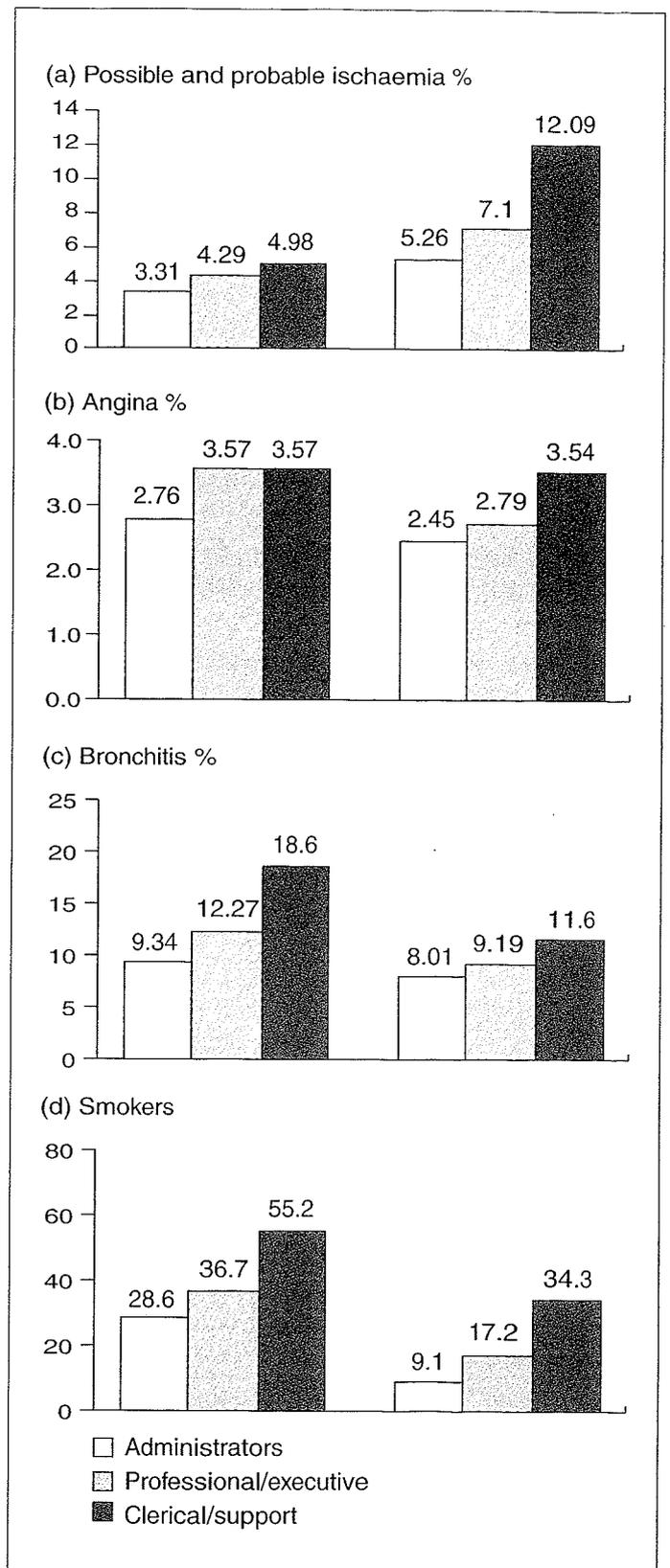


Figure 10. Prevalence of cardiorespiratory disease and smoking among men aged 40-54 in the Whitehall I (1967-1969) and Whitehall II (1985-1988) studies: age-adjusted percentages of (a) probable and possible ECG ischaemia; (b) angina pectoris; (c) chronic bronchitis; and (d) current cigarette smokers (Marmot *et al.*, 1991).

variety and low use of skills show a social gradient; there may be differences in the frequency of life events or other sources of life stress; and there may be social gradients in social supports. In the second Whitehall study, as one measure of perceived control over their health, fewer of the lower grades reported that they believed it was possible to reduce the risk of heart attack.

There is here a richness of potential explanations for social inequalities in health that tie in with other evidence on psychosocial factors and health. Three major hypotheses concern job strain, low social supports, and low control, respectively. There is a large body of evidence linking high psychological demands at work and low control to cardiovascular and other diseases (Karasek & Theorell, 1990). The relative disadvantage of lower grades with respect to control could be a factor in their higher rates of disease (Marmot & Theorell, 1988). Similarly, the evidence on low social supports and ill health suggests a further contributor to the gradient in ill health (Berkman, 1984; House *et al.*, 1988).

The concept of 'control' may provide a common link to many of the psychosocial explanations. Perceived lack of control over health (external health locus of control) may account for social differentials in health behaviour. Perceived lack of control may also be the crucial factor in the workplace that explains why jobs of lower status are associated with higher disease risk. Syme has suggested that increasing lack of control may be a linking factor that accounts for increasing health disadvantage as the social scale is descended (Syme, 1989). Poverty, whatever else it represents, is lack of control.

The evidence linking these factors to social differentials in disease is again stronger for cardiovascular disease and mental illness than it is for cancer. It remains an open question how important these may be in cancer. They may potentially act by influencing health behaviours such as diet and smoking or by some more direct pathway such as those now being explored under the heading of psycho-neuro-immunology.

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