'INTERPRETING AND ADDRESSING INEQUALITIES IN HEALTH: FROM BLACK TO ACHESON TO BLAIR TO...?'

Professor Robert Evans

7th Annual Lecture

OHE
Office of Health Economics

CIAR
The Canadian Institute for Advanced Research
L'Institut canadien de recherches avancées

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OFFICE OF HEALTH ECONOMICS

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'INTERPRETING AND ADDRESSING INEQUALITIES IN HEALTH: FROM BLACK TO ACHESON TO BLAIR TO...?'

'The Health Department of a great commercial district which encounters no obstacles and meets with no opposition, may safely be declared unworthy of public confidence; for no sanitary measure, however simple, can be enforced without compelling individuals to yield something of pecuniary interest or of personal convenience to the general welfare.' [*Second Annual Report of the Metropolitan Board of Health of the State of New York, 1867* (New York, 1868) p. 7.]
Quoted in Duffy, John (1974) *A History of Public Health in New York City, 1866-1966* New York: Russell Sage Foundation, p.1.

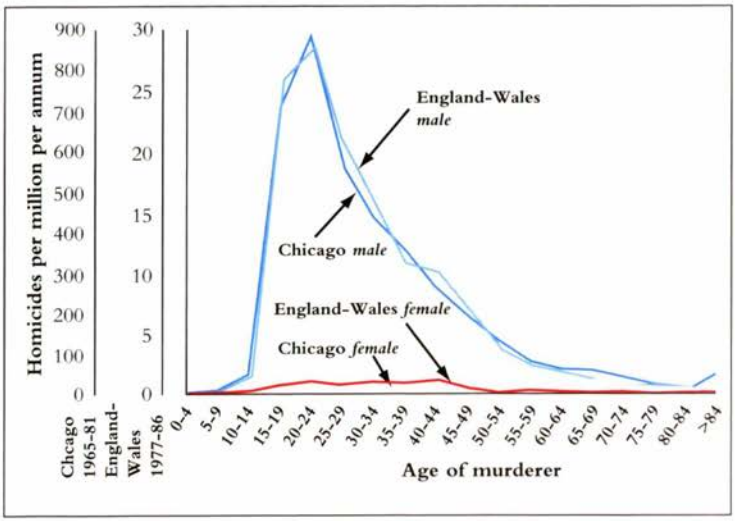
'Money is like muck; not good unless it be well spread.' [Sir Francis Bacon]

INDIVIDUAL VARIATION IN SOCIAL CONTEXT

In a recent paper, Michael Marmot (2000) makes graphically a point fundamental to the understanding of the determinants of health of populations. Figure 1, originally from Cronin (1991), plots homicide rates by age and sex of perpetrator for Chicago and for England and Wales. What first strikes the eye is the remarkable similarity of the age-sex patterns. In both jurisdictions, males are much more likely to commit homicide, and their propensity to do so first rises and then falls with age along virtually identical curves. The probability of committing a homicide clearly varies systematically with individual characteristics, as well as with the chances of time, place and circumstances. Individual personality differences undoubtedly also play a role; there might indeed be a 'gene for violence', or a genetic cluster, that predisposes some individuals to more potentially homicidal behaviour.

But then one notices the vertical scale. The probability that a male, of any age, will commit homicide is *thirty times* higher in Chicago than in England and Wales. The effect of individual characteristics, real though it is, is swamped by the overwhelming impact of the environment, of the social context (which of course includes the availability of firearms and attitudes toward their use). The age and

Figure 1 **Who kills, and where: A robust invariance**



Source: Cronin H, The Ant and the Peacock, 1991, Cambridge University Press.

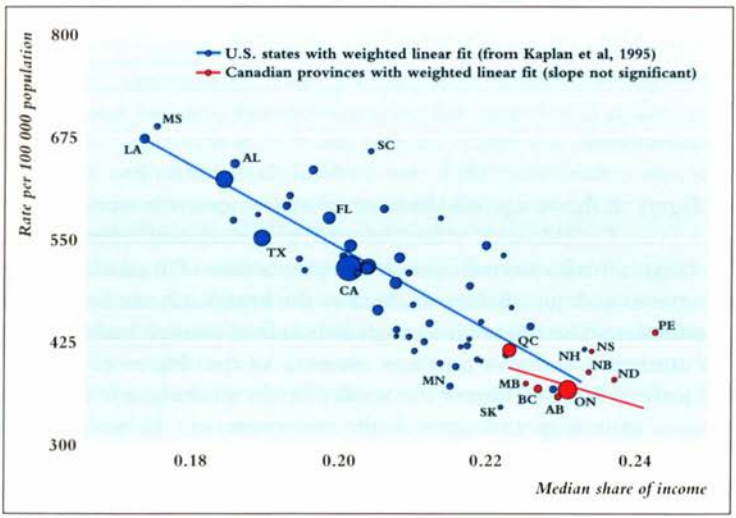
sex effects are peanuts; the elephants are lurking in the environmental background.

This is a powerful demonstration of a general epidemiological principle emphasized in a classic paper by Geoffrey Rose (1985): the causes of individual cases are not the same as the causes of overall incidence. The factors that tell us which individuals *within* a population will commit homicide – or more generally, become ill or injured – are not the same as those that determine the differences in rates *between* populations. If we were to study homicide only within Chicago, for example, we might be led to focus on the characteristics and circumstances of individual perpetrators. We might go beyond age and sex to look at personality differences, perhaps looking for genetic correlates. But if we were seriously interested in the causes of homicide we would be better advised to look at the differences between the social environments of Chicago, and of England and Wales. Searching for a 'gene for violence' is unlikely to lower the murder rate, however much it may advance research (and researchers) in molecular biology.

This advice can be generalized to more jurisdictions and more comprehensive indicators of health. Figures 2 and 3, reproduced from Ross *et al.* (2000b), embody a set of core ideas and current issues in the

understanding of health inequalities. They are part of a large and rapidly growing body of research with quite controversial policy implications.[1] A close analysis of these data thus provides a useful starting point for a discussion of the much broader question of why some people are healthy and others are not, and what could or should be done about that – the essential question addressed by the Black Report (Black *et al.*, 1980), the Acheson Report (Independent Inquiry, 1998), and the flurry of responses to the latter by the new Labour government in the U.K. (Department of Health, 2001). Focusing on income inequality, these figures raise the question of whether this is the *sine qua non* for reducing health inequalities? Or would a direct assault on inequality of income, with its obvious political difficulties, dissipate energy and public support that might better be devoted to more promising, less directly confrontational strategies?

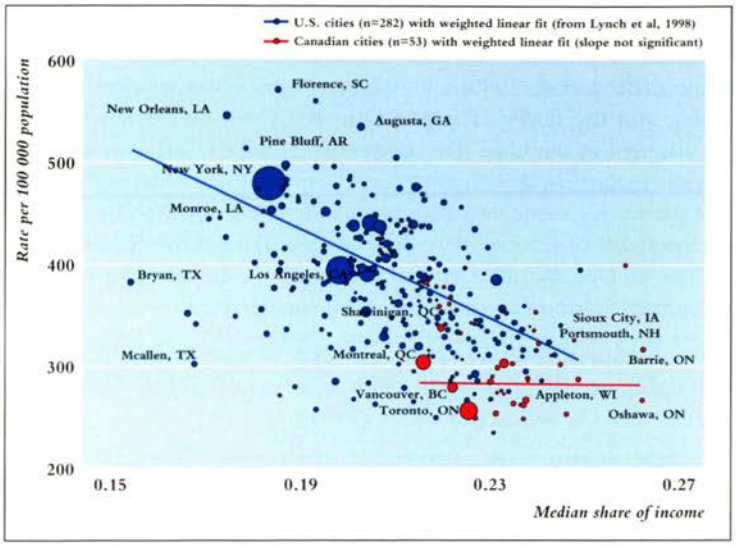
Figure 2 **Mortality in working age men by proportion of income belonging to the less well off half of households, U.S. states (1990) and Canadian provinces (1991)**



Source: Ross et al. BMJ, 2000, Vol 320, p. 898-902. With permission from the BMJ Publishing Group.

1 The relationship between income inequality and health was, for example, a dominant theme at the biennial meeting of the International Health Economics Association meetings in York, England in June, 2001. Surveys of the related literature are provided in Wagstaff and van Doorslaer (2000) and Mellor and Milyo (2001).

Figure 3 **Mortality in all working age people by proportion of income belonging to the less well off half of households, U.S. (1990) and Canadian metropolitan areas (1991)**



Source: Ross et al. BMJ, 2000, Vol 320, p. 898-902. With permission from the BMJ Publishing Group.

Figure 2 shows age-standardized mortality rates for working-age men in each of the states of the United States (blue), and the provinces of Canada (red), plotted against the proportion of total disposable income in each jurisdiction received by the lowest-income half of the population. The former is a rough indicator of average health status; the latter is a compact summary measure of the degree of income inequality. Figure 3 shows the same plot for working-age men and women in metropolitan areas in the two countries. In both figures there is a clear negative correlation. Jurisdictions (in North America) in which the division of income is more unequal, have higher average mortality rates. And the range of mortality rates associated with differing degrees of inequality is *large* – roughly two to one. Some places are much healthier than others.

These data illustrate the *heterogeneity* or patterning of health status within populations. There is systematic variation between *groups* of people defined by particular characteristics – in this case jurisdiction

of residence. That individual people differ in their health experience is a matter of common observation. Health, like homicidal behaviour, varies with individual characteristics such as age and sex, and the trajectory of individual health over the life course is influenced by random misfortunes, misjudgements, and misbehaviour as well as the luck of the genetic draw. But patterning – systematic differences in average health experiences among sub-groups defined by area of residence, occupation, income, education, or other variables of interest – carries information. '...[T]here is a tremendous potential to exploit heterogeneity in populations as a wedge for greater understanding.' (Sapolsky, 1993).

Correlation is not in itself causality, and the relationship between any particular partitioning characteristic and (some aspect of) health status may not always be clear. What exactly is it about Chicago, for example? But correlation (repeatedly observed) is evidence of *some* underlying causal process. The patterning of health status carries information about the determinants of health. If we knew why people are, on average, significantly healthier in some jurisdictions than in others, we might be able to use that information to reduce the differences among individuals.[2]

The explanation apparently implicit in Figures 2 and 3 has two important dimensions. First, our attention is drawn to disposable income as a key determinant of health. But second, and in fact more important, differences in mortality rates are related to a characteristic of the states and provinces themselves, not of the individuals comprising them. Disposable income can be measured for individuals (or families), but individual measures of income inequality are undefined.

Moreover the authors report that, once account is taken of differences in the degree of income inequality, aggregate mortality rates are largely unrelated to differences in average income levels. It is the socioeconomic environment in which people live and work – as reflected in these data by the degree of income inequality – not the income level of the community, that is important for health.

This observation again exemplifies Rose's (1985) distinction between the causes of incidence and the causes of cases. *Within* each jurisdiction, mortality may be associated with various measures of

2 One thing we know about such cross-regional differences is that they are not the result of genetic variation. Studies of migrants between regions with very different disease patterns show that within one or two generations the migrants take on the patterns characteristic of their new social environment (Marmot et al., 1984).

socioeconomic status, and in particular with income level, as well as with various forms of misconduct and mischance. But the large differences across jurisdictions are associated, as in the case of homicide in Chicago, with characteristics of the jurisdictions themselves.

ISOLATING THE INDIVIDUAL AGAIN: BEHAVIOUR AS 'CHOICE'

The point is fundamental. More traditional explanations offered for health inequalities, by contrast, have focused attention on individuals, rather than on their environments. It could be argued – and has been, often – that the average health experience of groups differs only because they contain different proportions of people with particular characteristics or behaviours. 'Heterogeneity' reflects merely the aggregation of different mixes of individual characteristics in different regions – or social classes – rather than the shared experience of a particular social or physical environment, differing from those of other groups.

One of the clearest (and most extreme) expression of this alternative view was provided by Satel (1997, p.12). '... [D]ifferences are largely explained by ... poor access to health care, inferior nutrition, obesity, smoking, alcohol and drug abuse, and reckless sexual behaviour. Various genetic differences may play a part as well.' If there are 'class' differences in health status, it is simply because the proportions of people with high-risk behaviours or characteristics increases as one goes down the socioeconomic spectrum. (Income itself does not appear in Satel's list.)

The heavy behavioural content of Satel's list invites the interpretation that 'it's really mostly their own fault'. The emphasis on individual characteristics leads directly away from concern with the social environment as a contributor to health or illness, and towards the view that there is something 'wrong' with particular individuals. What, if anything, could or should be done to 'fix' those individuals? Preaching good behaviour and penalizing bad? And if 'they' do not respond appropriately, then they deserve their fate. Just say 'No'. The message of Figure 1 is thus lost – as is the matter of income.

There is, of course, an incontestable relation between individual behaviours and health status – smoking, obesity, drug abuse, reckless sexual behaviour (!?), and so on. But this in no way justifies dismissal of the social context. If these behaviours were merely matters of personal choice – the 'unhealthy choices' of the health promotion movement – one might expect to find them randomly distributed across

the population like choices of vanilla or chocolate ice-cream. They are not. 'Unhealthy behaviours' are generally highly concentrated at the lower end of the socio-economic spectrum. One has to ask: 'What is it about the social experience at different bands in that spectrum, particularly in childhood, that predisposes to or protects against smoking?'[3] The same question must be asked of any other form of 'individual' behaviour that shows a clear pattern with social context. Individual behaviours are included among the pathways through which aspects of the social environment are translated into health outcomes.

More generally, since illness and particularly death are ultimately biological events, experienced by the individual, all explanations of health differences must eventually be associated with differences among individuals. In the case of homicide, one individual kills another. But this is the end of a pathway; the more relevant question is what happened higher up that pathway, what were the 'upstream' circumstances emphasized in the Acheson report? Why does this process begin so much more frequently in Chicago? Why do so many more people smoke, and suffer the adverse health consequences, among those of lower socioeconomic status? Why are death rates so much higher in some American states than in others (and in all Canadian provinces)?[4]

3 The correlation between education and health has led some to argue that increased education leads ('empowers') individuals to make healthier choices. Unhealthy individuals can thus be 'fixed' by more education. The inverse correlation between education level and smoking behaviour, for example, could be interpreted as showing that education is 'preventive'. But Fuchs (1986) demonstrated, using California data, that differences in smoking behaviour emerge in high school, well prior to differences in eventual educational attainment. One could argue that smoking causes lower education; but a more plausible explanation is that differences in social context – class or family background – influence both smoking and education.

4 One may suspect that, if pushed hard enough, some of those who favour individualistic explanations for health differences would ultimately fall back on genetics. Top people are simply genetically superior to those farther down; that's why they are on top. That superiority shows up in healthier choices, as well as in healthier constitutions. The causal link runs not from status to health, but from genetic predisposition to both status and health. Such an explanation has a natural appeal to those at the top; implying as it does that such differences are no one's fault, and nothing much can be done about them. But evidence is accumulating to show that the expression of a genetic predisposition depends upon the environment, particularly in early life (see below). Genetic endowment is not predestination.

WHICH SOCIETY? WHOSE CONTEXT?

The answer implied in Figures 2 and 3, however, is much less clear than might appear at first sight. Taken as a whole, North American jurisdictions show a very clear positive relationship between mortality and inequality. The Canadian provinces fit within the overall pattern in Figure 2, placing a lighted tip on the American cigar. Incomes are more equal in Canada and mortality is lower (though a correlation between *individual* income and health is found *within* regions in both countries). But this relationship disappears if the Canadian provinces are viewed in isolation. Similar data for Australian cities (as yet unpublished) fit into Figure 3 in exactly the same way as those for Canada, lying at the lower right of the overall scatter of points, but showing within themselves no relation between inequality and mortality.

These observations strongly reinforce the view that differences in the socioeconomic context exert a powerful influence on health. Canada, and Australia, appear to be quite different from the United States in having not only more egalitarian income distributions but also a social environment that buffers the influence of income inequality on health. It may be that the United States is quite unusual in the extent to which income inequality does translate into increased mortality. Rather than being a general social phenomenon, a relationship between income inequality and health status may emerge only in certain types of societies – perhaps (in the developed world) only in one (see below).

MONEY CAN'T BUY ME HEALTH... CAN IT?

And perhaps not even in one. In their review of the burgeoning literature on inequality and health, Wagstaff and van Doorslaer (2000) argue that data from aggregate-level studies of population averages, such as those represented in Figures 2 and 3, are insufficient to discriminate between alternative hypotheses as to exactly how health depends, causally, on income. They focus therefore on studies of the relationship between income and individual health status, all done with American data. They find in these studies little or no support for the hypothesis that individuals' health is related to the degree of inequality in their societies, and none for the hypothesis that individual health is related to relative position in the income distribution. Instead, they find strong support for the more traditional view that individuals' health is related to their own income levels,

independently of the pattern of incomes of those around them – the so-called 'absolute income' hypothesis.

Such a conclusion could have extremely important implications. It suggests that policies to redistribute income more equally, while they would tend to improve the health of the gainers, could on average *reduce* that of those at the upper end of the spectrum. The same would be true if health status depended on one's relative position. Only if health status depends directly on the degree of inequality of the income distribution itself, can everyone's health be improved by a more egalitarian distribution of incomes. (If the effect of absolute income on individual health status disappears, above some income threshold, then the health of those above that threshold is not threatened by redistribution. But it is not improved either.)

Redistributive policies would nonetheless lead to a reduction in health inequalities and an increase in average population health status, because the evidence indicates that the health benefits of increasing income are smaller at higher income levels. The health of gainers from income redistribution improves by more than that of losers deteriorates. But economic policies that require those toward the upper end of the income distribution to give up health as well as wealth are likely to be political non-starters. Those who, in all countries, control or powerfully shape the political agenda, will have trouble with that degree of self-sacrifice. 'There must be some other way.'

The absolute income hypothesis appears to offer that other way – strong encouragement of economic growth. Equi-proportionate increases in individual incomes, across the income spectrum, will improve everyone's health *and* reduce health inequalities, because the health of those with the lowest incomes improves most. Everybody wins, and political consensus can replace class war. Forget the income distribution, go for growth.[5]

The only trouble with this comfortable economic scenario is that it cannot possibly be true. The 'absolute income hypothesis' focuses attention, yet again, back on individual characteristics as determinants of health. In essence, health is something individuals can 'buy',

5 This is, of course, something of a simplistic leap. Everyone's health improves, and inequalities narrow, *if* all incomes rise in equal proportions. If the benefits of growth are all received at the top end of the income distribution, while those farther down face static or falling incomes, then health inequalities will widen and population health status may even fall.

presumably through the purchase of particular health-enhancing goods and services, independently of their social context. But we know from a wealth of other research – and general information – on the determinants of health that no matter what the econometric studies show, this is wrong.

Sen (1999), for example, makes this point quite simply in noting that the average African American man in New York, or San Francisco, or the District of Columbia has a lower life expectancy than the average Indian or Pakistani. The effects of '...lack of guaranteed health care...blighted educational arrangements, and other problems of social malaise and disruption.' far outweigh any benefits of a much higher per capita income. Social context overwhelms the effects, if any, of the individual characteristic – as in Chicago.

The same point is made more comprehensively by the experience of Cuba, whose population enjoy a health status similar to that of 'first-world' countries, despite 'third-world' income levels (Jatar-Hausmann, 1999). Cuba is a kind of society very different from those elsewhere in Latin America – poorer (on average), but much healthier.

Less dramatically, Canadians are on average healthier than Americans despite considerably lower average incomes – as indeed are most western Europeans.[6] The striking contrast between the Canadian and American results shown in Figures 2 and 3 is, however, in itself a clear indication that there are significant differences in the social environment in the two countries that cannot be explained by differences in income.

At a lower level of geographic aggregation there is a substantial international research literature (reviewed by Pickett and Pearl, 2001) reporting the effects of neighbourhood characteristics on individual health, after adjusting for individual characteristics such as income,

6 This observation does, however, illustrate Wagstaff and van Doorslaer's point (*ibid.*) about the difficulty of drawing causal conclusions from aggregate data. While average incomes are lower in Canada, the incomes of those in the lower half of the income distribution are actually absolutely higher, in terms of purchasing power, than those of their American counterparts (Wolfson and Murphy, 1998). It is in the extreme upper reaches of the income distribution that Canadians fall far behind. The absolute income hypothesis would lead one to interpret Canada's health advantage as an advantage in the bottom half of the distribution that outweighed a disadvantage among those with higher incomes. But there is no evidence of such a disadvantage among upper income Canadians.

education, and occupation. Within the U.S. itself, for example, Haan *et al.* (1987) demonstrated from the Alameda County (California) study the powerful effects on mortality of living in a designated poverty neighbourhood, after adjustment for a broad array of individual characteristics. Most recently Diez Roux *et al.* (2001) report similar results for coronary heart disease. In the U.K. Duncan, Jones and Moon (1993, 1996, 1999) have been pursuing a long term research program using multi-level modelling to explore the relation of location to health, and particularly to individual health-related behaviour.

More generally, the much broader literature on the determinants of health indicates that, at least among citizens of wealthy societies, any relationship between individual income level and health status has to arise from the effects of the social context in which people at different levels find themselves. The debate over the significance of income inequality concerns its relative importance as a dimension of that context, and correspondingly as a possible target for those trying to reduce health inequalities.

BACK TO BLACK: THE SOCIAL GRADIENT IN HEALTH

To reconnect with the broader literature, it will be helpful to go back and trace out briefly how that debate has reached its present form and focus. In the process we may perhaps also suggest why a discussion whose centre of gravity has historically been in the United Kingdom and among epidemiologists and sociologists has recently flared up among American economists.

There is a long-established and very sophisticated research tradition in the United Kingdom focusing on the relationship between class and health. (The British front up to the reality of class; the term 'socioeconomic status' seems preferred in the 'classless society' of the United States.[7] But the shift in terminology serves to blur the fundamental distinction between differences in shared environments, and differences in individual characteristics.) A grouping of individuals by social class consistently reveals a clear gradient – the higher the healthier. This gradient may be displayed by relating various different measures of health to various different measures of status – income, say, or occupation, or education, or conceivably (in

7 '...Americans see class all around them – they just don't always call it that.' (Gamson, 2001, p. 36).

the U.K., and if the data were available) accent. But these variables are markers of status; they are not necessarily directly causal of good or poor health. Income, in particular, has been understood as an important indicator of class status, of lifestyle in the original meaning of the term, as a whole cluster of ways of living rather than a description of individual behavioural choices.

Figure 4 reproduces the now-classic historical data on social gradients in mortality in the United Kingdom (OPCS, 1978, extended in Marmot, 1986) that were drawn on extensively in the Black Report (Black *et al.*, 1980). There is a clear gradient across the social spectrum; there is no evidence of a threshold above which mortality rates converge. Even more significant, the observations span a long period of time during which there were dramatic improvements in both health status and living standards among the British population. The occupational hierarchy corresponds, more or less, to differences in income. If individual health status were causally linked to individual levels of absolute income, with a threshold or in a curvilinear relationship, then the slope of the gradient should have declined as overall incomes rose. An increasing proportion of the population would be above the

Figure 4 **Mortality by social class 1911-1981 (men, 15-64 years, England and Wales)**[a]

	Social class				
Year	*Professional*	*Managerial*	*Skilled manual and non-manual*	*Semi-skilled*	*Unskilled*
	I	*II*	*III*	*IV*	*V*
1911	88	94	96	93	142
1921	82	94	95	101	125
1931	90	94	97	102	111
1951	86	92	101	104	118
1961[b]	76 (75)	81	100	103	143 (127)
1971[b]	77 (75)	81	104	114	137 (121)
1981[c]	66	76	103	116	166

Source: Marmot (1986) and OPCS (1978).

a Figures are SMRs, which express age-adjusted mortality rates as a percentage of the national average at each date.

b To facilitate comparisons, figures shown in parentheses have been adjusted to the classification of occupations used in 1951.

c Men, 20-64 years, Great Britain.

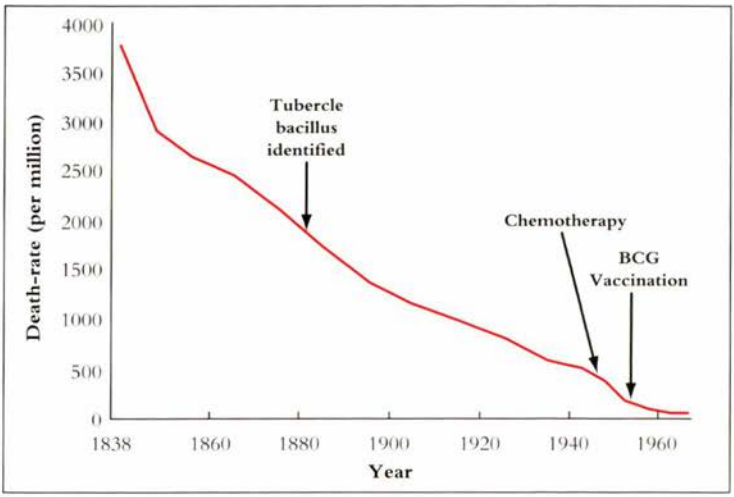
threshold, or more generally into ranges of income in which the health effects of further increases were small. This has not happened.

Also, over this long time span, medical care increased enormously in both quantity and effectiveness, and became much more equally available across the population. If access to effective medical care were a significant contributor to the social gradient in health, that gradient should have been shrinking. Over the same time period the predominant causes of death have changed radically from infectious diseases and other acute illnesses and injuries, to the chronic and degenerative illnesses of later life. Yet throughout this major demographic transition the gradient has been preserved and perhaps even grown steeper. The causes of death have changed, but on average the 'lower classes' still die sooner, indicating that the sources of the gradient lie, not in the relative incidence or virulence of particular diseases, but in underlying patterns of differential vulnerability – or 'host resistance' – by social class. Average life expectancy has greatly increased, but the mortality rates of the different social classes have moved up in line. Whether or not the host resistance of the whole population has improved, the relative positions of the social classes have not changed.

HISTORICAL DECLINES IN MORTALITY: PUBLIC HEALTH OR PRIVATE WEALTH?

The increases over this period both in living standards and in the quantity and quality of medical care are of particular interest. McKeown demonstrated over thirty years ago that the major declines in mortality from infectious disease in the U.K long predated the introduction of effective medical therapy (McKeown, 1979). He noted that the decline in tuberculosis mortality was particularly significant (Figure 5). Tuberculosis is not water-borne, so would also not have responded to public sanitary measures to dispose of sewage and supply clean water. Moreover, most of the population continued to show evidence of exposure to the bacillus, long after the death rate had fallen. On the basis of this evidence, McKeown concluded that improvements in diet and general living standards, by increasing host resistance, were the crucial factors for improving population health. This would imply that health was causally linked, at least historically, with individual levels of absolute income – a conclusion that, as noted above, tends to support policies to promote general economic growth rather than income redistribution or public programs to improve the quality of the physical or social environment.

Figure 5 **Respiratory tuberculosis: mean annual death-rates (standardized to 1901 population): England and Wales**



Source: McKeown T, The Role of Medicine, 1979, Basil Blackwell Ltd.

More recent assessments, however, have noted that while TB is not itself waterborne, it is an opportunistic infection that can take advantage of reduced host resistance caused by diseases that are (Szreter, 1988). Waterborne gastrointestinal illnesses, in particular, can weaken the host both directly and by reducing the nutritional uptake from a given diet. Moreover, improved historical income data have revised previous beliefs as to the closeness of the association between reduced mortality and improved living standards during the nineteenth century, leading Szreter (2000) to conclude that McKeown was right about the minimal role of medical care, but wrong to dismiss the effects of sanitary measures and to place exclusive emphasis on the rise in individual living standards. Dramatic improvements in health status were associated with improvements in the material environment, which in turn depended on the socio-political context for their achievement.[8] We shall meet this idea again below as 'neo-materialism' (Lynch *et al.*, 2000).

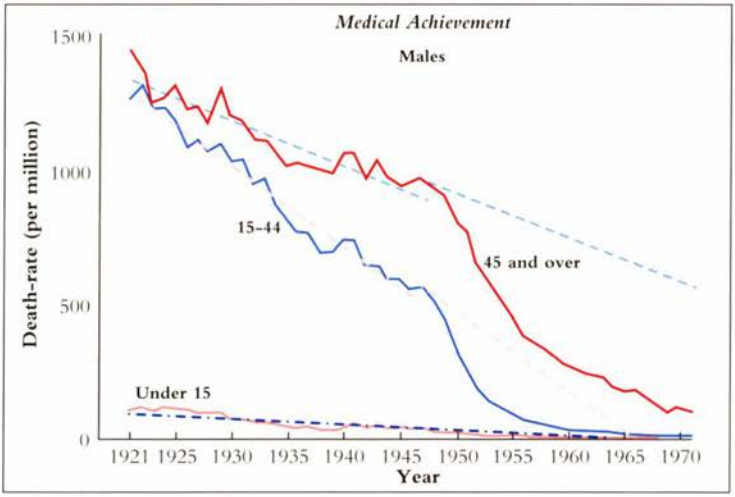
8 Szreter's revision makes intuitive sense. Drinking sewage is probably unwise even for Bill Gates.

HEALTH IMPROVEMENTS AND HEALTH INEQUALITIES: WHAT ROLE FOR MEDICINE?

While McKeown's conclusion as to the historical insignificance of personal medical care appears to stand up, it is worth noting the decline in mortality from tuberculosis following the introduction of effective therapy in the late 1940s. A very large proportionate decline in mortality emerges when these years are isolated from McKeown's longer term historical data (Figure 6); the new drugs did save lives. While medicine cannot in general claim credit for the dramatic historical declines in deaths from infectious diseases, this is no argument for medical nihilism. Medical care does matter; it is just that social conditions matter more. And the fact that the gradient has persisted through both advances in medicine, and radical shifts in the causes of death, suggests that its roots lie beyond the reach of medical therapy.

The fact that advances in medicine in this century are making a contribution to general improvements in health, raises the potential for unequal access to contribute to health inequalities. The United States, lacking a universal public system of health care finance, is unusual among developed countries in the degree of inequality of

Figure 6 **Respiratory tuberculosis: annual death-rates of males: England and Wales**

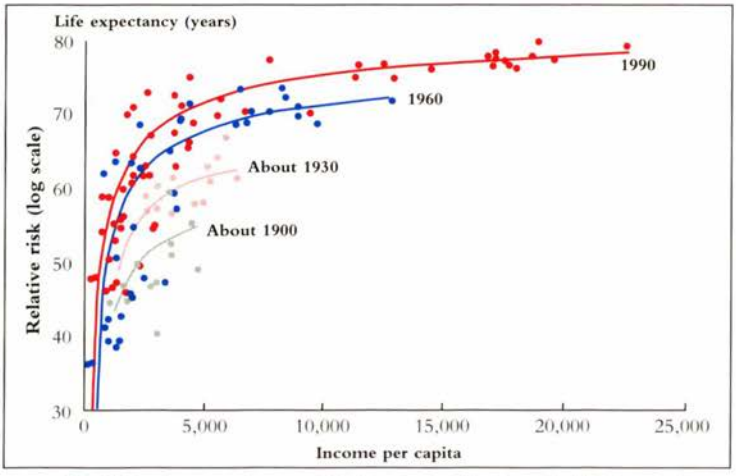


Source: McKeown T, The Role of Medicine, 1979, Basil Blackwell Ltd.

access and this undoubtedly does contribute to health inequalities. It is not clear, however, how much or whether differential access matters for general health inequalities in other developed nations.

It is striking that even the introduction of the National Health Service in 1948, with the explicit intent of making care more equally accessible across the income classes, had no discernible effect on the gradient in Figure 4. The use of care may have become more equally distributed, and that may have contributed significantly to the sum of human happiness and well-being. Certainly public support for the institution has always remained very strong. But at least on the measures in Figure 4 there is no evidence that it influenced the inequalities in health. There may be good arguments for further efforts to increase access or use by those lower down the income scale, but no basis for assuming that this would necessarily reduce inequalities in health. A *fortiori* there would seem little basis for any

Figure 7 **Life expectancy and income per capita for selected countries and periods**



Source: World Development Report 1993 *Investing in Health*, Oxford University Press.

Note: International dollars are derived from national currencies not by use of exchange rates but by assessment of purchasing power. The effect is to raise the relative incomes of poorer countries, often substantially. For illustrative country comparisons and a more detailed explanation, see Table 30 in the World Development Indicators.

presumption that general increases in the quantity and quality of care provided in the future would reduce health inequalities, however desirable they might be on other grounds.

The long term stability of the social gradient, and the absence of any sign of threshold effects from rising income, are not mirrored in comparisons of countries with very different levels of average income. Figure 7 is reproduced from the World Development Report (World Bank, 1993, Fig. 1.9), extending work by Preston *et al.* (1972). Average life expectancies in lower and middle-income countries are strongly correlated with national income per capita. There are some very suggestive exceptions, in addition to Cuba, that demonstrate the independent effect of social or cultural context for good or ill (Caldwell, 1986), but the general relationship is clear.

The benefits of increasing income are, however, subject to diminishing returns, flattening out as average incomes increase. Somewhere out beyond $10,000USD ($1991) per capita – depending upon who is looking at the Figure – average incomes reach a threshold at which the relationship disappears. Significant mortality differences remain among high-income countries, but they are unrelated, or very little related, to average incomes. [It is also notable that there have been substantial upward movements in these curves over time, reminding us that there are other determinants of health besides income, and these seem to be improving.]

INCOME INEQUALITY AS A THREAT TO HEALTH

But if international variations in average health status among wealthy countries do not depend upon income levels, what do they depend on? The answer given by Richard Wilkinson did not originate with him – he has been thorough in identifying his precursors – but he more than anyone else placed income inequality firmly in the spotlight. In a series of publications (1992, 1994, 1996a) he explored the relationship between inequality and health, drawing on cross-national comparisons of both levels and changes over time in measures of income inequality with levels and changes in measures of health status. He argued that there was a clear relationship across countries linking greater inequality with lowered health status.

Wilkinson's work was not without its' critics. Their concerns included selectivity in the choice of measures used to represent income inequality (Judge, 1995) and about the countries included in

his comparisons. Wilkinson tended to rely on the measures that most clearly made his case, and his results have turned out to be quite sensitive to the particular set of countries compared. There were also serious problems with the comparability of cross-national data on income inequality because income distribution data in most countries are much more fragmentary than aggregate economic statistics, and are much less consistently defined and compiled across countries.

It was therefore something of a triumphant vindication when, in 1996, two different research groups in the United States, using unimpeachably comparable data for the American states, confirmed Wilkinson's hypothesis. The original version of Figure 2, including only the American observations, was published by Kaplan *et al.* (1996).[9] A companion paper by Kennedy *et al.* (1996) found essentially similar results using a different measure of inequality, the 'Robin Hood Index' or the proportion of aggregate income that would have to be transferred from above- to below-average income recipients, in order to achieve complete equality of incomes. Both studies also found that the effect of inequality on health persisted, after allowing for differences in per capita incomes. In an accompanying editorial George Davey Smith (1996) hailed Wilkinson's hypothesis as a 'Big Idea', a breakthrough in understanding of the determinants of health and the sources of health inequalities.[10]

It *is* a Big Idea, not only intellectually intriguing – through what pathways does inequality *per se* 'get inside the body' to cause illness and even death? – but also potentially politically transformative. At a time when market forces in a number of countries have been generating significant increases in economic inequality, and governments have been treating this trend with either 'benign neglect' or active assistance, the finding that economic inequality was hazardous to health, not just for the losers but for the whole population, offered a powerful basis for opposition. As Ross *et al.* (2000a) put it with charming directness: 'What better way to promote income distribution than to demonstrate that population health is compromised, in societies which tolerate a large gap between rich and poor?'

9 The American data in Figure 3 were presented in Lynch *et al.* (1998).

10 The American findings, like Wilkinson's, might still have been a statistical artefact arising from a (negative) relationship betwen individual income and mortality risk that weakens with rising income (Duleep, 1995; Gravelle, 1998). Wolfson *et al.* (1999), however, have shown that while this effect is present, it is not sufficient to account for the aggregate rekationship.

THE ECONOMETRICIANS AND THE WILKINSON HYPOTHESIS

Ironically, however, the American data that provided by far the clearest evidence for Wilkinson's hypothesis also shifted the centre of gravity of the debate to the United States, and have drawn in more economists. Their professional preconceptions lead them to view 'income' as a discrete variable in a system of causal connections, rather than as one indicator of a social context, a world-view has been influenced much more by physics than by sociology.

For tracing out these hypothesized causal connections, multivariate regression analysis is the 'methodology of choice' and its results are treated by many economists as an informational gold standard, rather than as one piece of evidence, possibly helpful, possibly misleading, in a much larger puzzle. But since the results of such statistical exercises are so diverse, depending upon the data set chosen, the formulation of the variables, and the assumed functional form of the relationship, there is endless opportunity for debate over which findings are the real gold, and which the fool's gold. Participants generally accept implicitly, however, that 'the truth' is to be found somewhere in the growing blizzard of regression equations.

In this intellectual environment, several cross-currents have emerged that have pulled understanding both forward and back. Lynch *et al.* (2001) have analyzed cross-national income distribution data generated by the Luxembourg Income Study from a set of OECD countries, that is of a quality and comprehensiveness not available to Wilkinson ten years ago. They find that his hypothesis is not sustained in this more extended cross-national analysis.

A literature survey by Judge and Paterson (2001) concludes that '...the relative effect of income inequality *per se* as a determinant of population health has been greatly exaggerated... (p.1)' And a recent set of papers from several countries in the British Medical Journal (Osler *et al.*, 2002; Shibuya *et al.*, 2002; Sturm and Gresenz, 2002; Muller, 2002) finds little or no support for this relationship. In an accompanying editorial Mackenbach (2002) concludes that '...the evidence for a correlation between income inequality and the health of the population is slowly dissipating, with supporting evidence reduced almost entirely to (still inconclusive) analyses in the United States'.

But in fact the 'strong form' of his hypothesis, that average health status is always and everywhere (among wealthy populations) causally related to income inequality, had already been undermined by the

findings of Ross *et al.* (2000b). If the relationship exists in the United States but not in Canada, then its' presence or absence is conditional on other features of a society. Inferences drawn from American experience may thus have little or no general validity. Since it was the American data alone that rode so strongly to the support of Wilkinson's hypothesis in 1996 House (2001) seems to be on good ground in arguing that this bald statistical relationship is perhaps the weakest and least defensible aspect of the more general relationship between social inequality and health.

This does not mean that it is any less important to understand the variations in population health within and among countries, or that economic inequality is irrelevant to this understanding. The really interesting questions arise from consideration of what features of a society might predispose to, or buffer, a relationship between income and ill-health, or indeed operate independently of income. Lynch *et al.* emphasize the fundamental role of history and culture in shaping the experiences of different populations. Universal relationships holding across large numbers of observations, each characterized by a parsimonious set of measures, are unlikely to exist.

Yet it is precisely these sorts of historical and cultural factors that multivariate statistical analyses are least well adapted to identifying, let alone elucidating. By focusing on the interactions of a small number of variables defined as, or assumed to be, common to a number of observations, this particular methodology encourages its users to ignore broader issues of context. The variables used tend to take on a life of their own; like the entities of physics they have their effects, and can be manipulated, independently of what is happening around them.

BACK TO BLACKS? WHAT IS AN 'INDIVIDUAL' CHARACTERISTIC ANYWAY?

Deaton and Lubotsky (2001) provide an example, with their finding that the mortality differences among states in the United States, shown in Figures 2 and 3, are correlated with the proportion of the state population that is black. When this is taken into account, they find that the cross-state correlation between income inequality and mortality disappears. Just as Satel *(ibid.)* asserted that class differences were simply a result of 'classes' containing different mixes of people with healthy or unhealthy behaviours, so states differ in their average health status depending on the proportion of blacks they contain. Such a finding

appears to leads directly away from issues of income inequality or any other aspect of the social environment, and to point instead toward 'What's wrong with blacks?' and perhaps 'How do we fix them?'[11]

But in fact the matter is not so simple. 'Being black' would indeed appear to be (subject to questions of definition) a quintessentially individual characteristic. But how the wider society reacts to the fact of an individual's blackness is not. Racial questions are an ideological minefield. But as it happens there is an example of a remarkable 'exercise' in an American school classroom thirty years ago that powerfully illustrates the point (Coronel, 1996; California Newsreel, 1998; Kral, 2000).

In 1968, Jane Elliot was a schoolteacher in Riceville, a small Iowa town of about 1000 people, all white. When Martin Luther King was assassinated, her students could not understand why, and she devised a classroom exercise to try to communicate the experience of racism. The class were divided, purely arbitrarily, according to eye colour (brown vs. blue) and Elliot began systematically to discriminate in favour of the brown-eyed children and to denigrate and humiliate the blue-eyed, in numerous ways. She told the students that brown-eyed people were inherently superior, while assigning various negative traits to the blue-eyed.

She describes as 'horrifying' the speed – minutes, not days or weeks – with which both groups of children began to live their assigned roles. Brown-eyed children began to mistreat the blue-eyed, just as Elliot did, and to flaunt their 'superiority'. Both school performance and personalities began to diverge; the 'superior' group became more out-going and competent while the inferiors regressed emotionally and scholastically and showed signs of significant stress. The experience has apparently never been written up in the scientific literature, but Elliot has continued to conduct, for organizations and other groups, workshops that demonstrate the powerful impact of discrimination based on a distinction *known* by all concerned to be purely arbitrary. Apparently it works just as well for adults.

The phenomenon that Elliot triggered with her exercise dramatically illustrates the ambiguity of an 'individual' characteristic, and the complexity of inferring causality. In particular, it emphasizes that establishing a statistical relationship at the individual level does not

11 More research is probably necessary.

necessarily prove that causality originates at that level.[12] An individual characteristic such as eye colour – or skin colour, or income – may trigger reactions in the social environment, and responses in the individuals themselves, that are the real pathways to ill-health.[13]

Elliot's exercise clearly demonstrates the potential strength of such environmental responses to otherwise innocuous individual characteristics, but could that process be operating in Deaton and Lubotsky's results? It is well known that the prevalence of hypertension in the United States is markedly higher among blacks than among whites, and that this is one source of their higher mortality. Yet hypertension is not universally linked with 'blackness'; there is in fact a geographic gradient in prevalence rising from residents of west Africa, to the Caribbean, to the United States (Cooper and Rotimi, 1997). The reported range is roughly 2:1. It is being black *in the United States* (and to a lesser extent in the Caribbean) that is associated with hypertension. Similarly Fang *et al.* (1996) find cardiovascular mortality among blacks in New York City to be highest among those born in the American south, lower among those born in the north, and lowest among those from the Caribbean, suggestive of the role of the social environment in earlier life in 'embedding' longer term vulnerability or resilience. (This embedding process will be addressed in some detail below.)[14]

12 Interestingly, Deaton and Lubotsky (2001) find that the average health status of whites is lower in states with a large black population. This suggests that these are less healthy societies; Kawachi and Blakely (2001) found that state-level inequality correlate with average mortality rates for both blacks and whites. Deaton and Lubotsky point out, however, that this result could reflect out-migration by the healthier whites.

13 Most graduate students have had the experience of having very little money, but not of poverty. They are very different things.

14 Remarkably, having shown the geographic gradient, Cooper and Rotimi turn firmly away from exploration of environmental questions, social or otherwise. 'A focus on what causes hypertension in blacks, rather than the distraction of why blacks have more hypertension than whites, is likely to be a more productive strategy.' (p. 810). Or in short, what's wrong with blacks, and how could or should they be fixed? Similarly Gillum (1996) in an editorial comment on Fang *et al.*, suggests that that the geographic gradients correspond to the sequential acquisition of behavioural characteristics that are risk factors for cardiovascular disease. But if blacks are simply converging to the behaviour patterns of their new social environments, why are they so much more at risk

In such a complex and interactive environment, statistical tournaments between the 'Absolute Income Hypothesis' and the 'Relative Income Hypothesis' are likely to be productive of much publication and little understanding. A logically prior question has turned out to be more fruitful. How could income inequality *per se* influence health in the first place?

GETTING INSIDE THE BODY: FROM SOCIAL ENVIRONMENT TO PHYSIOLOGICAL RESPONSE

There is a perfectly plausible causal chain from absolute income to health – at very low income levels. Starvation, exposure, and various physical or psychological assaults have health effects that can be readily observed and understood. Absolute poverty – insufficient income to buy adequate nutrition, or shelter, or personal security (which would include necessary medical care) – is closely associated with illness. For those in poverty one would expect to find a direct link between rising incomes and improving health status, as indeed is found cross-nationally (Figure 7).

But such absolute poverty, sheer material deprivation, is also relatively rare in modern, developed societies (again the United States may be something of an outlier), nor could it account for a gradient in health status across the whole population. As noted above, if the link from income to health ran through (absolute) deprivation, the gradient would show a threshold income level beyond which the relationship disappeared – just as is observed in the international data – and the gradient itself should be disappearing over time as average

than whites in those environments? And if their higher mortality is to be explained by a more risky average 'portfolio' of behaviours and exposures (than that of whites), why do blacks in the United States preferentially adopt this more risky pattern? (Why is smoking now predominantly a lower-class activity? Why is homicide so much more popular in Chicago?) To dismiss the black-white differential as 'a distraction' is to throw away a crucial piece of evidence as to the role of differing social environments in generating good or bad health. Combined with the geographic gradient, this differential demonstrates that it is not 'being black,' but where a black person lives and works, that is associated with higher risk. Unlike the Japanese migrators studied by Marmot *et al.* (1984, note 2 *supra*.) blacks in the United States do not share the same social environment as the majority population.

incomes have risen. But this is not what the data show. The prevalence of absolute poverty is certainly not irrelevant as a source of health inequalities, but for most of the population of developed societies, a link from income to health, in whatever form, must operate along other pathways.

Roughly speaking, the explanations offered fall under two heads, the psychosocial and the neo-materialist. Wilkinson and Kawachi (E.g. Kawachi *et al.*, 1997) on the one hand, and Kaplan and Lynch on the other, have been leading exponents of these differing views. Both interpretations postulate a link from inequality to physiologically experienced stress as the route 'into the body'; the biological and behavioural ways in which individuals response to that stress then predispose to health or illness. The social environment will influence both the degree of stress experienced by different individuals, and the types of responses – healthy or unhealthy – that they muster (Cassel, 1976). (Both behavioural and biological responses are viewed as partly innate and partly 'learned' from the social environment and from past experience.) But the psycho-socialists and the neo-materialists differ over which features of the social environment, associated with income or income inequality, are primary sources of stress and/or conditioners of the stress response. These differing views have differing implications for the choice of appropriate policy responses.

The psychosocial view emphasizes processes going on within the mind of the individual – perceptions of self-worth (Wilkinson), for example, or degree of trust and confidence in others (Kawachi) – that may have direct physiological effects. Believing that one is inferior, inadequate, simply not as good a human being as those about one (raising obvious questions as to the reference group) and living in a society that constantly reinforces this belief, is a source of psychological stress. Greater inequality implies that more people are exposed to a greater degree of this form of psychological assault, so that average health status is lower. Alternatively, mistrust and suspicion of others increases the degree of stress and sense of vulnerability involved in the multiplicity of social and economic interactions that make up daily life. Shared environments and experiences, contact and communication, tend to reduce this mistrust; greater inequality on the other hand increases social distance and impedes mutual understanding.

The neomaterialists, by contrast, argue that while only a very small proportion of the population in wealthy countries suffers from sheer

material deprivation – basic diet and shelter inadequate to sustain good health – nonetheless the whole population is constantly exposed to any number of minor and sometimes more serious challenges, frustrations, and threats to well-being, just in going about their daily lives. Money is an extraordinarily effective 'all-purpose tool' for mitigating or avoiding these stresses, permitting one to mobilize extra resources to cope with them. Greater income permits one to fly first class, to hire a nanny or other home helps, to buy better quality equipment and to pay for preventive maintenance, to maintain a financial cushion to deal with unexpected crises from car breakdown to unemployment or serious illness. Higher income is associated with greater coping ability, or its converse, lesser self-perceived vulnerability.

'Money as all-purpose coping tool' provides the neo-materialist explanation for a social gradient across all income classes; a link between inequality and average health requires consideration of how that money is spent. The wealth of a society can be spent both individually and collectively; in a number of cases these are alternative routes to similar objectives. Individuals may buy more or less reliable and comfortable automobiles, or the community can spend on a high-quality public transportation system. The community can upgrade its water and sewage systems, or individuals can purchase bottled water and home water purification systems, and try to avoid pollution by residential choices. Individuals can purchase medical care and insurance privately, or the state can provide a universal, comprehensive health insurance or health care system financed from taxation. Public education and private schools have a long history of competition. Public libraries, concerts, parks, and recreational facilities are alternatives to private purchase or private travel. And so on.

In general, collective approaches to improving the physical and social environment tend to be both less expensive and more effective *for the population as a whole*.[15] But for the wealthy they may be less

15 Interestingly Anand and Ravallion (1993, quoted by Sen, *ibid.*) find that even among poor countries, where the strong positive link between health and average income per head is well established, average health levels are most closely related to the average incomes of the poorest in the population, and to the levels of public expenditure, particularly on public medical services. Indeed, once measures of these expenditures are accounted for, the cross-country link between average health status and average income disappears. Augmenting the private incomes of the better-off, even in poor countries, brought no health benefits.

effective, and are certainly more expensive. The taxation to finance public services is predominantly related to income, even if not always directly proportionate, so that higher income people pay more. At the same time they have less personal influence over the nature and amounts of the services they themselves receive than if they were purchasing privately.

It follows that if one's income is high enough one is better off (in purely financial terms) to oppose, politically, the provision or upgrading of public services. The more unequal are incomes in a society, the more pronounced will be the disadvantage to its better-off members from public expenditure, and the more resources will those members have to mount effective political opposition. The more inegalitarian the society, the stronger the economic motivation for 'private affluence and public squalor.' Gated communities, private schools, private medical insurance offer the best of both worlds, the opportunity for wealthy individuals collectively to purchase high-quality municipal services, education, and health care for themselves (often with substantial public subsidies) without having to contribute toward the provision of a similar standard for anyone else. The 'secession of the wealthy' could thus provide an explanation for deteriorating public services and falling health status associated with increasing income inequality.[16]

The neomaterialist interpretation seems, at least to this author, to have a more satisfying concreteness than the rather insubstantial hypothesized link from a perception of inequality to a psychological loss of self-esteem followed by physiological damage. That said, however, a remarkable recent finding by Redelmeier and Singh (2001a) seems impossible to explain in other than psycho-social terms. They find, in examining the careers of movie actors, that winners of academy awards

16 Szreter's re-evaluation of McKeown's work is significant in this context. McKeown's analysis seemed to suggest that growing private consumption, rather than public sanitary measures lay behind the dramatic improvements in health status in Britain during the late 19th. and early 20th. centuries. Szreter's analysis reinstates public expenditures in the central position. The clarification is particularly important, because McKeown's original findings could be subject to 'paleo-materialist' misinterpretation as supporting the promotion of economic growth *über alles*. If rising individual incomes per se were sufficient to explain past dramatic increases in life expectancy, one might argue that future increases – regardless of how they were distributed or used – would also be the most effective way of improving population health.

show a systematically longer life expectancy – by an average of 3.9 years – than those who were nominated but never received an award.

This is an extraordinarily large gap: 'about equal to the societal consequence of curing all cancers, in all people, for all time.' (*ibid.*, p. 961).[17] Yet actors at this level, whether winners or unsuccessful nominees, all have incomes far above any level at which more material resources might help to prevent or mitigate stress. And the average lifespan of non-winners was still much above that of the general U.S. adult population.

The group studied is hardly representative; one may suspect that they are rather more egotistical and self-absorbed than most of us. But Redelmeier and Singh's findings seem to provide a very clear demonstration that perceptions of '...fortune and men's eyes...' can have a direct effect on mortality, wholly independently of material circumstances. The mechanism that Wilkinson postulates seems to exist, and to be powerful, whether or not it explains mortality differences in the general population.[18]

What it does not appear to explain, however, is the large and contradictory finding for screen-writers (Redelmeier and Singh, 2001b). Oscar-winning writers have on average significantly shorter life expectancies than non-winners (including nominees), though they have longer and more successful careers. Davey Smith (2001) offers a highly speculative (and highly entertaining) editorial comment; but the only conclusion seems to be that some very powerful psychological forces are at work in Hollywood, operating through biological and/or behavioural channels.

Less dramatically, but possibly of more general relevance, it turns out that individual trustfulness varies across societies, and tends to be higher among those with higher socioeconomic status. In particular, it is strongly correlated, across American states, with both income inequality and mortality (Kawachi *et al.*, *op. cit.*) Lynch *et al. (ibid)* do not find, in their comparison of OECD countries, a relationship between health measures and measures of trustfulness. Nonetheless lack of trust in personal relationships, financial or otherwise, remains

17 For multiple winners, relative to multiply unsuccessful nominees, the advantage rises to 6.0 years.

18 Elliot's *(ibid.)* brown-eye/blue-eye exercise was not, of course carried through to mortality! But the blue-eyed 'inferiors' showed clear signs of increased stress and depression.

a plausible source of stress, operating at the psycho-social level – measures of social isolation, as reported below, appear to have a measurable impact on health. Attempts to identify such an effect in cross-national data may be confounded by the cultural and historical factors that Lynch *et al.* emphasize.

The question of mechanism, of how social inequality perceived and/or real finds its way into the individual body to generate inequalities in health and ultimately in (age at) death, is far more interesting and far more likely to advance understanding, than controversies over statistical correlations. Pertinent to this question are the findings from a number of other studies with very different methods and objectives. These not only confirm the strength of the gradient in health and its relationship to various measures of status or hierarchy and their associated social environments, but also point towards a better understanding of the causal pathways through which these differing social environment could emerge as health inequalities.

The relevant studies follow individuals rather than looking at aggregate statistics; they come from the laboratory as well as the field, and they involve animals as well as humans. The common feature is that they address the relation between social structure and individual health.

HIERARCHY, STRESS, AND HEALTH IN WHITEHALL AND THE SERENGETI

We begin with two completely independent but surprisingly complementary research programs studying free-living primate populations: Robert Sapolsky's studies of olive baboons in the Serengeti ecosystem in Kenya, and Michael Marmot's studies of civil servants in the Whitehall ecosystem in Britain (Evans, 1996). (Representative individuals from each ecosystem are shown in Figure 8.) Both programs have been evolving over more than twenty years, and both collect individual-level, longitudinal data. The Whitehall studies have included between twelve and fourteen thousand people at any point in time, and so generate only statistical observations. Sapolsky's smaller study population permits him to include individual case studies, and to observe the impact on individuals of specific episodes in the life of the group.

The first stage of the Whitehall studies classified individuals according to their positions in the civil service hierarchy and followed them over a decade. As shown in Figure 9, there was a pronounced

Figure 8 Representatives from two ecosystems

HOW SIR HUMPHREY APES THE WILD BABOONS

Going ape: Nigel Hawthorne as Sir Humphrey, and a male baboon

By JENNY HOPE
Medical Correspondent

Yes, primate minister, it's a jungle out there in Whitehall

AS Sir Humphrey showed us, there's a lot of monkeying about in the higher echelons of Whitehall.

But Nigel Hawthorne's hilarious portrayal of the wily civil servant in BBC TV's Yes, Prime Minister, may have — as he might put it himself — more than a scintilla of anthropological similarity with the wild baboon.

Scientists have discovered that the animals on the plains of East Africa and government staff exhibit remarkably similar ways of coping with the stress of climbing life's greasy pole.

Low-paid clerks are three times more likely than their bosses to fall sick and die prematurely, while junior members of monkey clans are more likely to buckle under to threats from aggressive pack leaders.

However, it is easier studying the primates in the wild than those in Whitehall, says Professor Robert Evans of the University of British Columbia. For one thing, you can't subdue a civil servant with a tranquilliser dart.

The crucial link is the place of a monkey or human in the pecking order, he told the British Association's science festival at Loughborough University, Leicestershire. But personality and emotional make-up also help determine how well they deal with stress.

By measuring stress hormones in apes, researchers have a much clearer idea of how they might affect health among people subject to a similar fight for survival.

Research involving 10,000 civil servants has given fascinating clues to the effects of stress at work. Yet a study by Robert Sapolsky, who has been investigating baboons in East Africa for the last 14 years, used methods that would be impossible in Whitehall.

For a start, most of the interesting activities such as mating, grooming, feeding, confrontation and conflict take place under cover, not on the open plain.

'Moreover,' said Professor Evans, 'Sapolsky's basic technique consists of anaesthetic darting of subjects, as unobtrusively as possible, so as to take physiological measures under particular circumstances.

'It is difficult to imagine a researcher being permitted to dart a senior, or even a middle-level, civil servant during a particularly stressful meeting in order to measure hormone levels.'

For the young inexperienced baboon, bullying by his elders can ruin his health in the same way that a junior clerk is more likely to fall ill under constant pressure from senior staff.

But the comparison does not always hold. The most powerful baboons can maintain their position with the minimum of effort simply by baring their teeth.

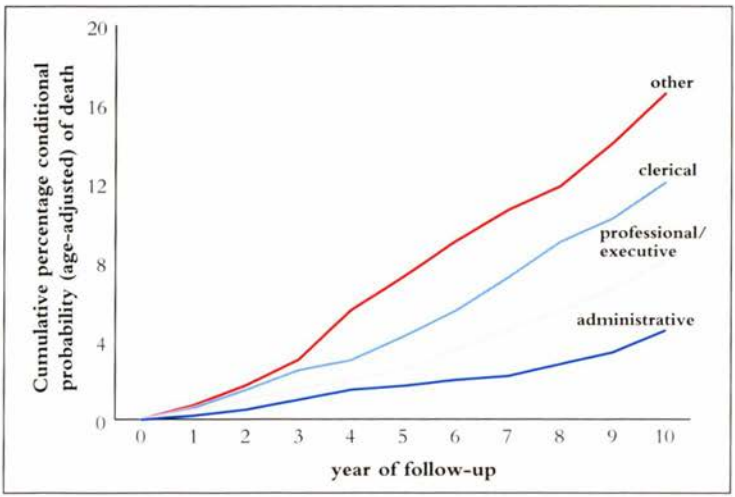
'It is doubtful if removing the canine teeth of adult males would greatly influence the hierarchical structure of the British civil service.'

But there is hope for humankind based on the monkey research, says Professor Evans. 'You don't have to remake society from top to bottom in order to mitigate the health effects of hierarchy. It may be enough to reduce the extent to which those of lower status have their faces rubbed in it.'

Source: Hope J, The Daily Mail, 6 September 1994.

gradient in (male) age-standardized mortality rates (Marmot 1986). The differences were very large, with working-age mortality roughly three times as high in the lowest as in the highest grades, and there was no sign of a threshold. The second-level professional and executive grades had higher mortality than those above them. All of the study subjects were employed; none were subject to absolute material deprivation or exceptional environmental risk.

Figure 9 **Whitehall study: all-cause mortality among total population by year of follow-up**



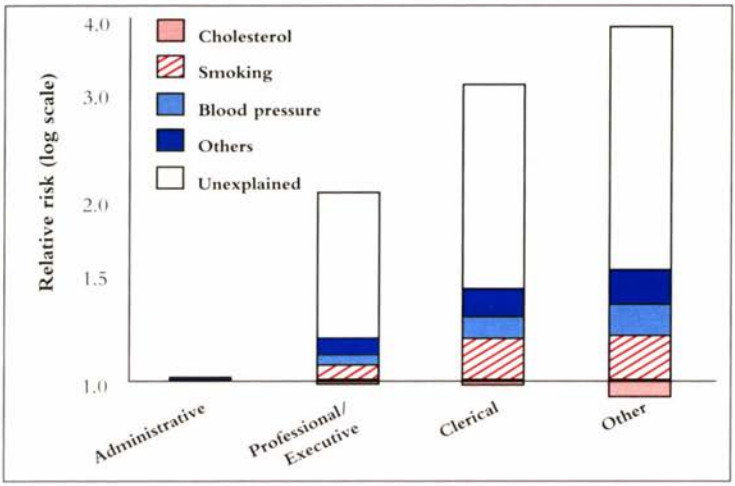
Source: Marmot M, in Wilkinson RG (Ed), Class and Health Research and Longitudinal Data, 1986, Routledge.

These findings were strong confirmation of the existence of a powerful effect on health status from factors correlated with hierarchical position. Those factors might be interpreted as psychosocial stress, though Kaplan (personal communication) has argued that the income differentials across civil service grades are large enough to yield very different levels of material resources for coping with the stresses of everyday life, and with its random shocks.

This mortality gradient was observed across most (though not all) causes of death, consistent with the data in Figure 4 showing persistence of the social gradient over time despite major changes in the dominant causes of death. Focusing specifically on deaths from heart disease, the Whitehall studies not only found a large mortality gradient, but found that little of the gradient could be explained by differences in the major individual risk factors – smoking, blood pressure, and blood lipid levels. Figure 10 displays the partitioning of their relative effects (Marmot *et al.*, 1978). These 'Framingham' factors[19] – individual

19 Named for the long-term prospective study of individuals in Framingham, Massachusetts that has identified these characteristics as predictive of heart attack.

Figure 10 **Relative risk of CHD death in different grades 'explained' by risk factors (age-standardised)**



Source: Marmot M, J Epidmiol Community Health, 1978; Vol 32: p. 244-249. With permission from the BMJ Publishing Group.

The figures on top of the histograms are the relative risk of CHD death (age-standardised), taking the administrative grade as 1.0. These figures are calculated from the equation and differ slightly from the 'crude' figures.

attributes – have received the lion's share of attention from health promoters, clinicians, and the public, and are the targets for massive and intensely marketed pharmaceutical interventions.[20] They are not irrelevant, but as in the homicide example above the individual attributes account for peanuts. The elephants lurk in the background of the social environment. (Even if the individual risk factors had been more powerful, one would still be left with the question of why 'exposures' were so clearly hierarchically graded. Attempts to reduce health inequalities could be addressed either to the risk factors themselves, or to their sources.)

20 This intense marketing has promoted the widespread use, in wealthy countries, of new and extremely expensive drugs for hypertension treatment in particular, that according to expert consensus are no better than, and in some cases worse than, older cheaper drugs (Morgan, 2000; Evans *et al.*, 2001). The resulting waste of public and private resources may well have had a negative impact on health.

Subsequent analyses have confirmed these earlier findings (Marmot, 2000), and have extended them to show similar gradients in both short and long-term sickness absence, for both men and women. More recent studies have also tried to identify the characteristics of the working environment that appear to be correlated with stress, and that vary across grade. These employ both surveys of workers and external evaluations of job characteristics; investigators are also examining various biological markers of individual strain that might be indicative or predictive of physiological damage.[21]

The pattern shown in Figure 9 also emerges from a much larger study by Davey Smith *et al.* (1996a, 1996b) who analysed mortality data from a sixteen-year follow-up of men screened in the early 1970s for the Multiple Risk Factor Intervention Trial (MRFIT) in the United States. Between November 1973 and November 1975, 361,662 men aged between 35 and 57 were screened at 20 different sites. This provided a massive amount of individual-level baseline data on factors believed to be associated with risk of mortality, including the Framingham factors, blood pressure, serum cholesterol, and smoking. For their study, Davey Smith *et al.* identified the zip code (postal code) of residence of these men at the time of the trial, and derived from 1980 census data the average income per capita in each area. Each individual screened for the MRFIT was then matched with the average income for his area of residence.

The investigators found a very clear, strong, and monotonic relationship between mortality and (residential area) income – higher income, lower mortality – a relation that persisted (though somewhat reduced) after adjusting for all baseline differences in individual risk factors. Most importantly, the effect did not weaken at higher incomes.[22] The size of the study population permitted the

21 The literature on stress tends to be quite confused as to whether 'stress' is a characteristic of the external environment, or of the organism's response to that environment. The neologism 'streessor' is quite widely used to refer to external forces. The engineering literature is, by contrast, quite plain and unambiguous. 'Stress' is the external force acting on a material or structure; 'strain' is the degree to which the material or structure is deformed – bent out of shape – by that force. We follow the engineering usage.

22 Or did it? The relative risk for each income category below the highest rose by an increasing amount with each (equal) drop in income. But the percentage increases in relative risk were approximately equal at each step.

investigators to use a large number of income steps, discriminating among income levels well above average.

These findings were strong confirmation that the relationship between income and health, whatever its source, cannot reflect simple material deprivation. They also confirmed the Whitehall findings of gradients in most, though not all, causes of death. And they arise from a very large sample of individual-level data on a broad general population (of middle-aged males). The fact that the measure of status used was ecological – residence-area income rather than individual income or as in Whitehall, employment grade – raises the issue of 'ecological fallacy' on the other hand as noted above there is ample evidence that regional characteristics have an influence on individual health independently of individual characteristics.[23] Davey Smith *et al.* refer to their income measure as an indicator of socioeconomic position, a marker rather than a proxy for individual income as a direct causal factor.

The Whitehall and related studies provide some of the most powerful evidence for a social gradient in health because they track the experience of individuals through time. They are not vulnerable to some of the technical criticisms that have been brought against the data in Figure 4, used in the Black Report and subsequently, that the composition and certainly the relative size of the social classes are not stable over time, for example, and the sources of information on the social class of decedents (death certificates) differs from those for the general population. But the question remains. What is this hierarchical effect and how does it work? How do differences in status, or their correlates, 'get inside the body' to influence physiological processes that lead to illness, and ultimately death?

Here Sapolsky's studies of baboons have been of particular importance (1990, 1993, 1999). As in the Civil Service, there is a status hierarchy among male baboons that is relatively transparent to the external observer. (There is also a hierarchy among females, but expressed somewhat more subtly.) Dominant animals are able successfully to challenge subordinates for access to food sources and mates, either by actual combat or by threat displays based on previously established status. (In the Serengeti, as in Whitehall, the food supply is relatively plentiful so that subordination does not imply

23 Recognizing this, Hertzman has suggested substituting the concept of 'ecological truth' for the traditional concern for 'ecological fallacy'.

absolute material deprivation.) But it turns out that associated with these behavioural patterns there are differences in physiological functioning that could be quite significant for health. Dominants and subordinates do not react the same way to threats or other challenges; their physiological responses to stress are different.

The stress response or 'fight or flight syndrome' is a collection of physiological changes in an organism that mobilize maximum energy transport to the large muscles and heighten alertness so as to deal with perceived immediate threats – the hungry leopard. What Sapolsky calls the 'optimistic functions' of growth and repair, and reproduction, are shut down. Investments in tomorrow are suspended in favour of ensuring that there *is* a tomorrow. This includes inhibiting the very energy-expensive immune system; infections are a less immediate threat than leopards.

But the suspension of investments has longer run costs for an organism, just as it does for an organization. The optimal stress response thus consists of very rapid mobilization, followed by rapid down-regulation once the threat is past. And accurate assessment of threats avoids unnecessary activation or prolongation of this highly adaptive but ultimately costly process.

Dominant baboons tend to show this desirable pattern of response, as evidenced by, among other things, the concentration in the bloodstream of the hormone cortisol that triggers subsequent physiological changes.[24] Subordinate animals, however, tend to show a slightly slower rate of elevation of cortisol in response to stress, and a substantially slower rate of decline. Furthermore, they tend to have an elevated basal level of cortisol, suggestive of a continuing state of low-level arousal (and corresponding inhibition of 'optimistic functions'). The explanation appears to be that subordinate status is associated with frequent threats that are, from the subordinate's point of view, not satisfactorily resolved. The physiological mechanisms that respond to the level of circulating cortisol and turn off the hormonal processes of the 'fight or flight' syndrome become blunted, and the animal is exposed to its deleterious effects on a more or less permanent basis (Sapolsky, 1990, 1999).

24 The process of immobilizing these powerful wild animals with anaesthetic darts and carrying out the necessary physiological measurements under field conditions is by no means a simple operation.

The immediate question is, could this mechanism be at work in the Whitehall primate population as well? Immobilizing civil servants with anaesthetic darts to measure their physiological responses to stress has not been a practical research intervention, but the search for biological markers of stress-response patterns *has* found that blood levels of one such marker, fibrinogen, are lower in the higher grades. And there is evidence that while blood pressure levels are elevated during working hours for all grades, blood pressure at home is substantially lower among the higher grades. This may reflect differences in home environments, but is certainly consistent with more effective down-regulation of the stress response (Marmot and Theorell, 1988).

Moreover the perceived circumstances and states of mind of human primates can be investigated through questionnaires, a form of self-report at which baboons are notoriously incompetent. The Whitehall studies are now generating findings showing a (negative) relationship between mortality and the sense of control, both self-assessed and evaluated by others, associated with particular jobs. This association is independent of other measures of social status, and appears more powerful in its effects.

This line of investigation has its roots in the work of Karasek and Theorell (1990), who classified jobs according to the degree of demand – the pace, effort and skill required – and the extent of control the individual worker has over the working process. 'Job strain' – and negative effects on health – were postulated to be generated by a mis-match between demand and control. They found that high demand/low control jobs were associated with higher mortality; low demand/high control jobs were healthiest. But the Whitehall results suggest that it is the dimension of control that is critical; with enough control one can cope with demand.[25]

These recent Whitehall findings from the work environment tend to confirm the work of Syme (1991) who has emphasized the more general relationship between health and the sense of control over ones' environment. Terms like 'self-efficacy' or 'mastery' carry the same idea – does the individual feel able to deal with whatever challenges or threats the environment is likely to throw up? Lack of

25 Presumably those who feel unable to cope with the demands of a job, do not feel a sense of control.

control implies continuing vulnerability, even if an immediate threat is not present. Sapolsky's subordinate baboons would probably also report a low level of control if they were asked.

The sense of control has an interesting link with some of the literature on the effects of placebo drugs – inert pills given to provide a comparison with the effects of an (hypothesized) effective drug. A randomized trial of the drug clofibrate, a number of years ago, tested its effects on mortality after five years among survivors of a first heart attack (Coronary Drug Project Research Group, 1980). The drug did not work; the five-year post-heart attack mortality rate of men assigned to take the drug was not significantly different from that of men given the inert pills. But what did emerge was a strong and statistically significant advantage among those who complied closely with their assigned therapeutic regimen. Mortality was about fifteen percent among those who took their pills, no matter what was in the pills. Those who did not, had mortality rates of 25% – 28%.[26] These differences could not be explained by any of the extensive measures taken at baseline of the characteristics of the individuals in the trial. Compliance with a long-term regimen might well be interpreted as reflecting a sense of control or 'self-efficacy' – the belief that what you do, matters.[27]

The Whitehall and Serengeti studies are in a sense starting from opposite ends of a possible bridge. While the baboons show hierarchically associated variations in physiological responses to stress that are consistent with health effects, the civil servants show hierarchical variations in health outcomes that must emerge from some physiological pathway. Laboratory studies, however, offer further clues as to the linkage from hierarchy to health, to the middle span of the bridge.

SOCIAL ENVIRONMENTS AND ARTERIAL PLAQUE

An extended program of research at Wake Forest University, using caged cynomolgus macaques (as models for the development of heart

26 Among the less compliant, there was a slightly lower mortality rate among those (sometimes) taking clofibrate, but the difference was not statistically significant.

27 The authors' comments are interesting: 'Obviously there must be characteristics differentiating between good and poor adherers (e.g. alcohol use and abuse, behavioural characteristics, or *socioeconomic status*) [emphasis added] not accounted for...' (p. 1040).

disease in humans) has shown a direct link from the social environment to the state of the coronary arteries (Hamm *et al.*, 1983; Manuck *et al*, 1988; Kaplan *et al.*, 1999). The animals are fed a diet moderately high in cholesterol in order to promote the development of arterial plaque. When they are 'sacrificed' (to the God of Science?) they have a measurable degree of occlusion of the coronary arteries. Diet does matter. And, as with humans, the males develop a significantly greater degree of occlusion. But the animals also develop a readily identifiable dominance hierarchy within each cage, and the subordinate animals show a markedly higher degree of occlusion.

The investigators found an average of 44 percent occlusion of the coronary arteries among subordinate males, twice that among dominants (23 percent). Subordinate females had about the same degree of occlusion as dominant males – 24 percent – but dominant females had less than one third that of subordinates – about seven percent occlusion (Hamm *et al.*, *ibid.*). It is worth emphasizing that these animals were fed identical diets, and shared the same physical environment. Their social environments, however, differed markedly depending upon their hierarchical position, differences that are recorded very concretely in the coronary arteries. The relative impact of status differences was greatest among females, though the overall degree of disease was greatest among males.

The investigators explored this relationship further by deliberately disrupting the social hierarchies (Manuck *et al.*, 1988). For a later set of experimental animals, the cage memberships were changed at periodic intervals. Each animal was then faced with a new set of associates. Dominance hierarchies reemerged, and the same animals became dominant in each environment. But in the unstable environments the dominant males developed a *greater* degree of atherosclerosis – about twice that of subordinates, and over twice the rate of those in stable environments. Rates of atherosclerosis among subordinates were actually lower, by about 15 percent, in the unstable environments.

Stable hierarchies appear beneficial to those on top, but having constantly to establish dominance in a new group may be hazardous to health.[28] (We will come back to this point again, in a different,

28 While the difference in degree of occlusion between dominants and subordinates was statistically significant in the unstable case, that in the stable environment was not. It was nonetheless quite large, and a finding of 'lack of statistical significance' is not in itself conclusive evidence that an effect does

human context.) One is reminded of Sapolsky's (1990) comment that baboons are relatively comfortably placed in their ecological niche in the Serengeti, with plenty of time to spend in stressing each other, an activity at which they are very skilled. Possibly the requirements of dominance competition leave less time and energy available for harassing subordinates.[29]

Female dominant animals, however, did not lose their advantage in the unstable environments, suggesting that the modes whereby female primates establish dominance are not only different from those of males, but less costly to their health (Manuck *et al*, 1988).[30] Furthermore the deleterious effects of status competition in males could be prevented by the administration of a beta-blocker – propranolol – demonstrating to those who understand such things that the mechanism of damage in males operates through the HPA (hypothalamic-pituitary-adrenal) axis. The propranolol did not modify the behaviour of dominant males in unstable environments – hyper-vigilant and aggressive – but it did prevent the coronary consequences.[31]

not exist. 'Insignificance' may be found either because an effect is not there, or because the power of the test is insufficient to establish its presence with a high degree of confidence. The investigators were working with only six cages. The observed differential between dominants and subordinates is in fact statistically the 'best estimate' of the effect size, and gains plausibility from its consistency with other studies. (Sapolsky (1992), for example, describes an episode in which the dominance hierarchy of the baboon troop became unstable, and the dominant animals lost their physiological advantage.) The convention that an effect size is accepted only if it is significant at the 5% level (and is otherwise to be set at zero) is just that, a convention, and can lead to quite contradictory results depending upon the way in which the research question is posed.

29 Sapolsky also observed that subordinates were at less of a disadvantage during a period of drought, when finding food and water took up more of the time of dominant animals.

30 The suppression of one 'optimistic function' in subordinates, however, showed up quite clearly; they ceased ovulating.

31 The behaviour of these macaques recalls the discussion of 'type A' behaviour among human males, as a risk factor for heart disease. It appears that it is not the energetic, time-pressured, achievement-oriented dimension of type A behaviour that is a risk factor, but associated feelings of hostility, anger and aggressiveness. The 'happy warriors' are not at risk, only those who are angry and unhappy – and presumably feel threatened.

Again there is an intriguing parallel in a study of drugs in humans. A randomized trial nearly twenty years ago of the same beta-blocker, propranolol, showed that, compared with a placebo, it did significantly reduce mortality rates in men three years after a first heart attack (Ruberman *et al.*, 1984). But the study also showed very significant differences in mortality among the members of the trial population according to their degrees of social isolation and life stress. Individuals classified at the beginning of the trial as both socially isolated and highly stressed had *four to five times* the mortality rate of those not so disadvantaged. This compares with a 38% higher mortality risk among those receiving a placebo, compared with those receiving propranolol.

These findings indicate that, while drug treatment was indeed effective in lowering mortality rates, its effect was dwarfed by the effects of social circumstances. Furthermore, social isolation and life stress were most prevalent among study subjects with lower education, which is a marker for, indeed a dimension of, socio-economic status. If levels of income and education are correlated with a sense of control over ones' environment, and a lowered sense of vulnerability, they may operate through the same HPA axis and mitigate the risk of a too-frequently stimulated stress response. Administration of a beta-blocker could have the same effect, but perhaps not to the same degree.

FROM STATUS TO HEALTH: A LONG AND WINDING PATH

Studies of non-human primates, like those of human primates, thus show a strong relationship between social status and health; the animal studies also provide considerable additional information as to the biological pathways that link health with rank. Yet further research has indicated that there is no rigid, mechanical relation between hierarchy and health. It is the experiences associated with hierarchy or status, and the way they are interpreted by the individual, that matter, rather than hierarchical position *per se*. To quote Sapolsky (1999, p.39):

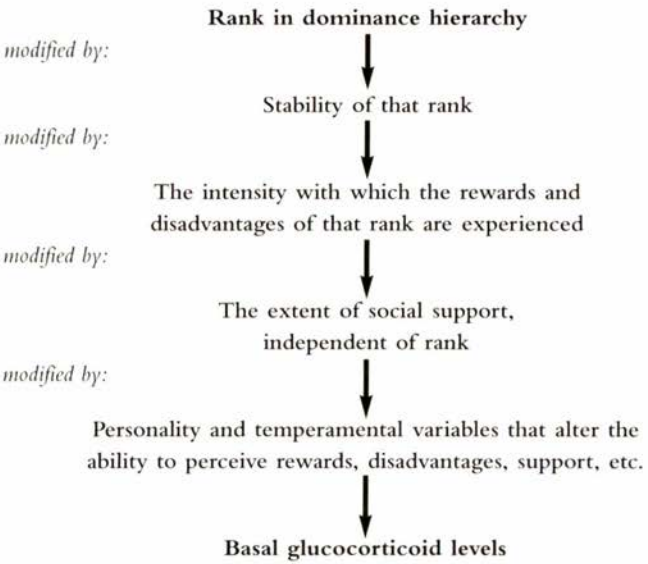
> 'A prime revisionist emphasis of this chapter has been how *little*, in fact, rank *per se* predicts any of those endpoints. [differences in physiology and stress-related disease] Instead, it seems virtually meaningless to think about the physiological correlates of rank outside the context of a number of other modifiers – the sort of society in which the rank occurs..., the individual's

personal experience of both the given rank and society,...and the role of personality as a perceptual filter of outside experience.'

Figure 11 from Sapolsky *(ibid.)* sketches out the links in a chain of effects leading from rank or social status to the level of basal glucocorticoid (*aka* cortisol) levels in the bloodstream. As noted above, a rise in this hormone mobilizes the transport of energy to the muscles and suppresses competing energy-using activities. Elevated basal levels indicate a prolonged and potentially harmful stress response – one that is never fully turned off.

First, as shown with the cynomolgus macaques, it is stability in rank rather than rank *per se* that is correlated with reduced stress and good health. Where rank must constantly be re-asserted through 'dubious battle,' or at least the expenditure of considerable energy and

Figure 11 **Social modifiers of the magnitude of the stress-response in male olive baboons, as measured by basal glucocorticoid levels**



Source: Sapolsky R, in Panter-Brick C, Worthman CM, (Eds), Hormones, Health and Behaviour; A Socio-ecological and Lifespan Perspective, 1988, Cambridge University Press.

attention, it may become – for males – a source rather than a reliever of stress, and a correlate of illness. But even a well-defined and stable hierarchy need not create radical differences in life experiences. R.H.I.P., but how great are those privileges, and how much, relatively, do those at the bottom suffer?

Coming back to the data in Figures 2 and 3, 'the intensity with which the rewards and disadvantages of...rank [are] experienced' in the United States may be greater than in Canada (or other industrialized countries) because income inequality is significantly greater (Wolfson and Murphy, 1998). But even more important, the wider range of public programs, and the narrower range of purely market transactions, also serves to lower the intensity of differential experience associated with income. The lack of guaranteed access to health care and blighted educational arrangements that Sen *(op. cit.)* pointed to as contributing to the high mortality of African Americans in particular, are generally associated with lower income in the United States, but are not experienced by Canadians at any income level. The more generous public programs of income assistance, unemployment insurance, severance provisions and minimum wages, and the job security and benefits associated with a higher rate of unionization, all serve not only to mitigate the extremes of poverty (relative to the United States, if not western Europe), but also to reduce the degree of uncertainty, the vulnerability, of individuals in the face of global or local economic fluctuations or other misfortunes.

Figure 12 illustrates the same point with Swedish data from Vagero and Lundberg (1989), who classified Swedish males according to the same set of social categories that are used in Figure 4. A social hierarchy is observed in Sweden, as it is in the U.K. or indeed everywhere in the known world. But the mortality differences associated with hierarchy are much less, again presumably because rank is experienced with less intensity. Moreover the mortality rate among the most disadvantaged Swedes is lower than that among the highest status British – raising the floor in Sweden has clearly not required lowering the ceiling.

Even if those on top do tread rather firmly on those farther down the scale, however, the resulting damage will depend upon how well those farther down are supported to bear the weight. Over a decade ago, House *et al.* (1988) assembled the results of several studies showing a negative relation between mortality rates and measures of social support (Figure 13). They noted that the evidence linking social

Figure 12 **Death rates by social class for men aged 20-64 years in England and Wales (1970-72) and Sweden (1961-79)**

	England and Wales			*Sweden*		
Social class	*Proportion of population*	*Deaths 100,000*	*RR**	*Proportion of population*	*Deaths 100,000*	*RR**
I + II	0.23	530.5	0.79	0.27	378.2	0.87
IIIN	0.12	644.1	0.96	0.11	454.7	1.04
IIIM	0.39	699.4	1.05	0.43	445.3	1.02
IV + V	0.25	784.2	1.17	0.19	482.0	1.11
I + II + IIIN	0.35	569.1	0.85	0.38	404.2	0.93
IIIM + IV + V	0.64	732.5	1.10	0.62	459.6	1.05
All	1.00	668.1	1.00	1.00	435.7	1.00
Ratio: $\frac{\text{IV + V}}{\text{I + II}}$	1.00	668.1	1.48	1.00	445.7	1.27
Ratio: $\frac{\text{IIIM + IV + V}}{\text{I + II + IIIN}}$	1.00	668.1	1.29	1.00	445.7	1.14

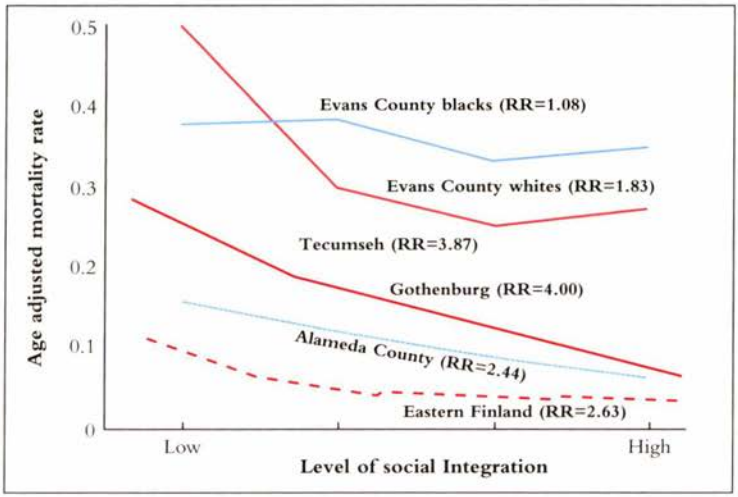
*Relative risk to total study population.

Source: Vagero and Lundberg. Lancet, 1 July 1989, p. 35-36, Vagero D, Lundberg O, Health Inequalities in Britain and Sweden. Reprinted with permission from Elsevier Science.

support with mortality was as strong as that linking tobacco and death had been in the early 1950s. Human and animal studies have shown that the presence of a 'significant other' (human or animal) powerfully reduces the physiological responses to stressful situations.

And finally, personality matters. Sapolsky (1990, 1999) lists a number of features of the personality that strongly modify the way in which an individual interprets and reacts to stressful situations. Long term observation of individual animals identified those with 'up-tight' and with 'laid-back' personality styles, and correspondingly more or less healthy physiological and behavioural responses to stress. Having female 'friends' other than for mating, and playing with the young, were for males associated with a healthier physiological profile, while hostility and aggressiveness, while they might contribute to achieving dominance while the animal is in its physical prime, were associated with a shorter period at the top and a much more stressful come-down. More generally, the ability to distinguish real from imaginary threats, to ignore the former and deal quickly and decisively with the latter, to recognize the outcome of a conflict – and to displace anger

Figure 13 **Level of social integration and age-adjusted mortality for males in five prospective studies**



RR: The relative risk ratio of mortality at the lowest versus highest level of social integration.

Source: Reprinted with permission from House JS, Landis KR, Umberson D, Social Relationships and Health published in Science Vol 241, p. 540-55. Copyright 1988, American Association for the Advancement of Science.

if defeated! – are all associated with successful dominance and with low basal glucocorticoid levels.[32]

The individual personality is, of course, the product of both genetic and environmental factors. How individuals respond to observed income differentials, for example, is likely to be considerably influenced by the importance attached to such differentials in their surrounding culture. The economic historian Donald McCloskey (1988) has described the question: 'If you're so smart, why ain't you rich?' as the quintessentially American Question, suggesting that American society places an unusually high value on personal wealth as the primary indicator of personal merit and achievement. In such an environment, the personal interpretation of inequality is likely to be

32 The personality styles associated with prolonged and low-stress dominance among baboons seem readily recognizable to human primates as those associated with success in 'office politics'.

particularly pronounced. Self-respect, as well as the respect of others, comes with money – if you were any good you *would* be rich – consistent with the observations from American states in Figures 2 and 3.

'BIOLOGICAL EMBEDDING': NEUROLOGY AS HISTORY

The discussion thus far has been largely a-temporal, implicitly treating both health and social status or hierarchy as constant and contemporaneous features of the individual life experience. But occlusion of the coronary arteries is a cumulative process, in monkeys as in men. The blunted ability of subordinate baboons to turn off their stress responses was a consequence of repeated experiences of stress imposed by the dominant animals, and the generalized effects of the suspension of 'optimistic functions' are long term. Researchers are increasingly focusing on the life course of the individual, to explore how experience may become 'biologically embedded' and so influence the subsequent responses – both biological and behavioural – to various forms of stress (Hertzman, 1994).

Perhaps the archetypical example of embedding is the well-established observation that kittens can be rendered permanently monocular if one eye is sewn shut during the fourth to sixth weeks of life – the period during which the neural 'wiring' is being laid down, the linkages among neurons being made in the visual cortex, that will process binocular signals from the two retinas. If one retina is sending no signals, no neural processing capacity is assigned to that eye. When the perfectly healthy eye is later opened, and begins to send signals to the brain, there is no neuronal capacity to receive them – and there never will be.

More generally, the neural system in humans and other animals adapts to the information coming in from the surrounding environment, and this adaptation is most rapid in very early life when there is intense competition for survival among an initially vast oversupply of neural cells. Those that form multiple connections and become part of cooperating networks of neurons survive, others become deactivated.[33] The functioning neural system is 'sculpted' out of the mass of available neurons. Experiences at this stage clearly have the capacity to influence this sculpting process in ways that permanently affect the functioning of the organism (Cynader, 1994; Cynader and Frost, 1999; McCain and Mustard, 1999, Ch. 1).

33 Success in the competition for individual survival depends on the ability to cooperate and to form networks. There may be a more general message here.

The process does not end in childhood; the neural system is not cast in biological stone by some point in the first decade of life. A recently reported study of London taxi drivers, for example, has found that the mental effort of 'doing the knowledge' – acquiring the extraordinary knowledge of London streets and addresses to qualify for a license – and of subsequently applying it on the job, is associated with enlargement of the hippocampus, a region of the brain particularly associated with learning and memory (Maguire *et al.*, 2000). It would appear not only that 'the knowledge' is biologically embedded in the brain – where else could it be? – but that the process of acquiring it generates expanded information storage capacity, more or more active neurons. This seems a very optimistic finding!

Nonetheless the neuronal plasticity generally declines with age – one can learn a language in adulthood, but it is not as easy, and rarely as well done, as in childhood. Moreover '...emerging evidence suggests that neurons in the brain that are most ready to learn (i.e. those that are within their critical periods) are the most vulnerable to the cell death and degeneration that are associated with chronic stress.' (Cynader and Frost, 1999, p. 182-183). The capacity to develop more effective coping styles – learning ability in the most general sense – may thus itself be weakened, at the neuronal level, by prolonged and unresolved stress. On the other hand there is also evidence, at least for rats, that brief but regular 'handling' of newborn pups, at a critical developmental stage (but not later), produces a resilience to stress that protects against this form of damage to cognitive function in later life (Meaney *et al.*, 1988).

PREDISPOSITIONS, OR POTENTIALS? GENE-ENVIRONMENT INTERACTION

These observations form the background to considerations of longitudinal processes, whereby health at a later stage of the life course may be affected by 'learned' coping styles or responses to various forms of challenge.[34] Again there are particularly suggestive findings

34 'Learned' here is much broader than the acquisition of conscious knowledge or explicit skills. It includes, for example, conditioned responses, or the learning that takes place when the immune system encounters a pathogen. Indeed the immune system can be 'trained' to respond to a completely innocuous stimulus. Cyclophosphamide temporarily depresses the functioning of the immune system. The immune systems of mice injected with this substance while being fed saccharine-flavoured water, will later collapse merely at the taste of saccharine (Ader and Cohen, 1975).

from a long term program of primate research (Suomi, 1996, 1999; Coe, 1999).

Rhesus macaques live in troops with a very well defined social structure, both matriarchal and matrilineal. Females remain in their natal troops for life, and derive their social status within the troop from birth order and matriline. The rankings of the different matrilines rarely change.

Males, by contrast, are expelled from their natal troops at puberty. After a period of life either alone or as part of a juvenile gang they are accepted into another troop and slowly work their way up in social status. This period of troop transfer is one of high stress and high mortality for young males. The troop as a whole is a highly effective organization for foraging and defense in the wild; isolated individuals and small groups are at much higher risk. Additional stresses are involved in the process of entering a new troop.

Coping with this high-stress time – as with the earlier period when they are temporarily abandoned by their mothers in the process of initiating a new breeding cycle, is simply part of the 'stress of life' for most rhesus monkeys. There is, however, a sub-group of 'high-reactive' animals, about 15%-20% of the population, who show extreme physiological and behavioural reactions to stress. As infants they react to novelty in their environments by showing fear and anxiety, and if possible avoidance, rather than the more normal curiosity and exploration. Their physiological reaction to such stimuli is '...significant (and often prolonged) activation of the...HPA axis.' (Suomi, 1999, p. 190) rather than moderate arousal. At maternal separation they show symptoms of depression and lethargy, and again physiological evidence of exceptionally high stress.[35] High-reactive males are late to leave their natal troops, and use 'very conservative', low-risk strategies for entering new ones. High-reactive females, while not exposed to the transfer risk, tend to be more likely to provide inadequate care for their first-born offspring. In general high-reactive animals 'follow aberrant developmental trajectories that can potentially result in increased long-term risk for behavioural pathology and even mortality.' *(ibid.)*.

35 High-reactors show the same physiological responses to both maternal separation and troop expulsion, though the behavioural responses of infants and adolescents differ.

Study of these high-reactive animals in captive populations has yielded very significant information as to the relationships between genetic endowment, early life experience, and subsequent health and fitness in adult life. High-reactivity is genetically based; the investigators have successfully bred strains of reactive individuals. Yet elementary evolutionary theory would predict that a gene consistently conferring 'increased lifetime risk for a variety of biobehavioural problems' (*ibid.*, p.191) would not itself survive long in a population. Are there not circumstances under which high-reactivity is an advantage?

The answer is yes. High-reactive infants cross-fostered to particularly competent and nurturing mothers (as shown by their care of previous infants) lose their disadvantage, and in fact convert it to an advantage. High-reactivity, if strongly buffered by sufficient security so as not to generate extreme anxiety, depression, and damaging physiological or behavioural responses to new environments, becomes a distinct advantage in social interactions and recruitment of allies, and can lead to high status within the troop. On the other hand, genetically normal animals that are separated from their mothers at birth, hand-reared, and housed with same-aged, like-reared peers, exhibit patterns of physiological and behavioural response to stress that are similar to those of genetically high-reactive animals.

These findings emphasize the significance of early life experiences in 'programming' the individual to give competent or self-damaging responses to later stresses. They also demonstrate that genetic endowment and social environment are not independent determinants, or competing explanations, of health. Both the expression of a gene, and its consequences, will depend upon the nature of immediate and past experiences and the possibilities offered by the wider environment. (A predisposition to violence may depend upon ones' genetic make-up; it will certainly depend upon the experiences of ones' upbringing, and its consequences will depend on the availability of firearms.)

The picture emerging from these and many other animal studies is thus one of a biological pathway from social environment to health status, operating through the stress response system. While that system is highly adaptive in itself, prolonged and unresolved stresses can lead to physiological changes that may be harmful in two ways. The stress response itself can produce damaging physiological consequences, by inhibiting the immune system for example, or promoting the formation of arterial plaque through associated changes

in the level and composition of blood lipids and effects on the dynamics of blood flow (Sapolsky, 1993; Marmot and Mustard, 1994). But a prolonged or frequently repeated stress response can also lead to changes in the response mechanisms themselves, such that the response is more difficult to turn off even in the absence of an external stress. More generally, past experiences – particularly those early in life – can lead to 'biologically embedded' changes in the stress response system that make the individual more or less vulnerable to later challenges. More or less effective 'styles of coping behaviour' can be learned – not necessarily consciously – by the individual, and are difficult to unlearn. But the neural structures and physiological processes within the organism are also 'learning' from environment and experience, and will in consequence also respond more or less appropriately to later challenges.

The genetic endowment is certainly part of this process, as it embodies predispositions toward particular forms of behaviour at both the individual and the physiological level. But these predispositions are not in general determinative; whether and to what extent they are expressed will depend upon the pattern of individual experience over time. There is, after all, no evidence that the social gradient in health corresponds to genetic sorting into classes of differential 'fitness'.[36] Rather the effects of hierarchy, or the social gradient, emerge from both the different social and environmental experiences of people at different levels in the social structure, and the cumulative effect of past experiences, particularly in early life, in embedding healthy or harmful responses to further experiences.

GETTING FROM THERE TO HERE: ALTERNATIVE ROUTES AND PROCESSES

The processes that define both social status and health thus begin at or prior to conception, and form a cascade of interacting environmental influences and individual responses, unfolding though time. Researchers have identified three categories of processes through which early life environments may affect adult health; the

36 There is evidence of differences by social class in such measures as IQ, or height, but these are also significantly influenced by early life experiences. The fact that these and other characteristics related to 'fitness' may be heritable, is not evidence that social class differences are of genetic origin.

categorization has implications for the choice of when and how to intervene to reduce health inequalities (Hertzman *et al.*, 2001).

Latent Effects of early life environments affect adult health independently of subsequent experience. A specific biological disadvantage at a sensitive period in early life, such as low birth weight, developmental delay, or impairment of vision or hearing, may have an impact on health and well-being that emerges in later life, regardless of intervening circumstances. An example would be the apparent link between low birth weight and the development of cardiovascular disease fifty or more years later (Barker, 1999). Similarly, early childhood stimulation programs for disadvantaged children appear to have yielded significant improvements in adult 'coping' abilities and socioeconomic circumstances, even though the children showed no benefits – and received no additional help – during the intervening years (Schweinhart *et al.*, 1993).

Cumulative Effects, on the other hand, are those in which the health effects of advantage or disadvantage accumulate over time, at a rate depending upon the duration and intensity of 'exposure'. The fact that the (negative) association commonly found between mortality and earnings is stronger when earnings are measured over several years, for example, is suggestive of a cumulative effect. Similarly, a prospective long term study of Scottish men, identifying their social classes of origin, at labour market entry, and in later adulthood, found that the association between social class and mortality was graded by cumulative social class (Blane *et al.*, 1996). The more consistent the status over the life course, the more powerful its influence for good or ill.

Pathway Effects operate through a more complex interaction between individual and environment. Early life environments influence the different trajectories of life experiences and opportunities on which individuals find themselves. But their reactions to those experiences and opportunities – also to some extent conditioned by early life experience – have the potential to modify their subsequent trajectories, at the same time as their own response patterns – again physiological as well as behavioural – are being influenced by their experiences and expectations. This interactive process can form a 'positive feed-back loop' – a vicious or virtuous circle. Highly nurturant, stimulating early life environments encourage and reward the development of positive coping strategies that lead onto more favourable life trajectories and further positive feed-back.

Conversely developmental disadvantage, an unstimulating, frustrating or threatening early environment, or a lack of social support, can lead to chronic stress and a sense of powerlessness and alienation. These early influences can result in direct health consequences, but also in reduced 'readiness to learn' at school entry and subsequent poor performance – with further negative psychosocial effects. Medium term consequences are a higher probability of low educational attainment, criminality, drug use, and teen pregnancy. These in turn increase the probability of a low status, poor quality working life, greater isolation and reduced social support, chronic disease in mid life, and accelerated aging in late life.

Pathway effects are not deterministic; the metaphor is a braided stream rather than a set of tramlines. People may move across trajectories, both up and down, as they pass through the life course, and as Figure 11 above emphasizes, social circumstances are not rigidly linked to health outcomes. But pathway effects do represent probabilities, that emerge from longitudinal studies such as the British Birth Cohort of 1958 (Power and Hertzman, 1999). On average, success builds on success, and failure on failure – advantage (disadvantage) at one point in the life course increases the probability of advantage (disadvantage) at the next.[37]

THE GREAT EXPERIMENT: HEALTH TRENDS EAST AND WEST OF THE WALL

The complexity and the long term nature of the linkages between status, stress and health place severe limits on the feasibility of experimentation with humans, quite apart from the obvious ethical limits on the range of permissible interventions. During the past generation, however, we have witnessed a vast 'natural experiment' carried out (inadvertently) in eastern Europe and the former Soviet Union. Investigators are now able to study and compare the long term health experiences of these entire populations with those of populations in the very different societies of 'the West' over the same

37 Studies of the 1958 cohort have shown both pathway and cumulative effects. Self-rated health at age 33 was linked both to level of education – a marker for different pathways – and also (independently) to a cumulative measure of socio-economic conditions from birth to age 33 (Hertzman *et al.*, 2001).

period. Measures of health status have evolved very differently between 'East' and 'West' (and also among the eastern countries themselves), in ways that are remarkably consistent with the general story sketched out so far.

This 'natural experiment' has had two distinct phases. The most dramatic has been during the period from the collapse of the Soviet-style regimes to the present. The resulting social disruption has been associated with increases in mortality that ranged from severe but short-term in those countries that most successfully managed the transition, to devastating and continuing in the former Soviet Union.

BEFORE THE FALL: THE GROWING HEALTH DISPARITY

But the twenty-five years prior to the collapse were perhaps even more instructive with respect to the determinants of health. When Bobak and Marmot (1996) wrote: 'There is a sharp divide in health status between the former socialist countries of central and eastern Europe (CEE) on the one hand and western Europe on the other.' (p. 17), their data were from 'before the fall'.

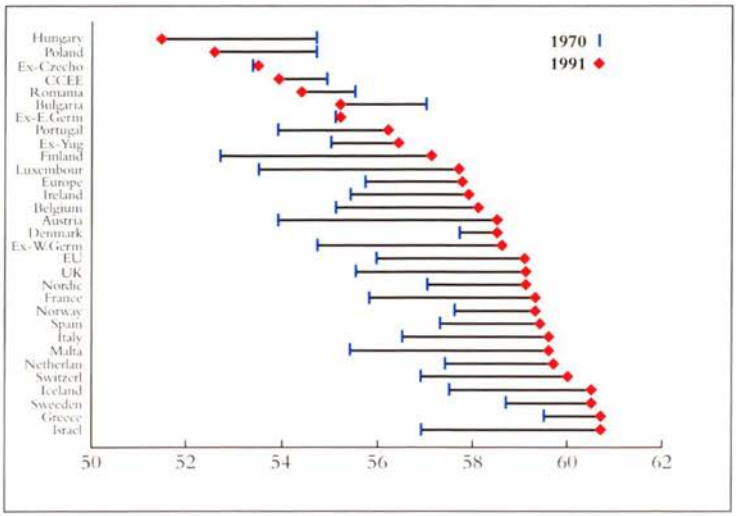
In 1991, life expectancy at birth for both males and females was lower in *every* CEE country than in *any* western European country.[38] But Figure 14 (*ibid.*, p. 21) tells an even more remarkable story. Between 1971 and 1991 male life expectancy at age 15 rose in every country 'west of the Wall' – a form of progress that we have always taken pretty much for granted. But it actually *fell* in the countries to the east.

Female life expectancies, both from birth and from age 15, rose in all countries, but rose less in the east, widening the pre-existing gap, while male life expectancy at birth rose in some CEE countries and fell in others. The conventional wisdom labels females, the elderly, and children as the 'vulnerable' populations; they are more at risk from threats such as famine or epidemic. But it is the health of working-age males that seems most sensitive to adverse social conditions.

Taking the longer view, life expectancies were much lower in the east after the Second World War, but were catching up until (depending upon the country) some point in the 1960s. From then on, however, eastern life expectancies stagnated or fell while those in the west accelerated their improvement (Hertzman, 1995, p. 70).

38 Almost. Female life expectancy in Slovenia was equal to that in Portugal.

Figure 14 **Changes in life expectancy at age 15 in men in Europe, 1971 and 1991**



Source: Figure 5 from Bobak M and Marmot M, in Hertzman C et al (Eds) East-West Life Expectancy Gap in Europe, 1996, Kluwer Academic Publishers. With kind permission from Kluwer Academic Publishers.

Conditions in the CEE countries had become seriously hazardous to health, long before their regimes collapsed.

A number of explanations have been offered. Industrial pollution was and is notorious in many parts of eastern Europe and the former Soviet Union. But while this has definitely caused considerable illness, and deaths, it is too highly localized and not on sufficient scale to account for the nation-wide deterioration in health. The inadequacies of communist-style medical care are indicted, but the mortality increases are as great, or greater, from causes of death that are not amenable to medical intervention. Large increases in mortality from AIDS and other infectious diseases may be on their way after the breakdown of the relatively competent Soviet public health systems, but these are not yet reflected in current data, and tell us nothing about the period from 1971 to 1991. High-fat diets, and smoking behaviour, have also been considered, but again these are not consistently associated with the changing mortality patterns (Hertzman, 1995; Hertzman and

Marmot, 1996). An explanation is needed that would apply across the entire populations of all of the CEE countries.

The obvious candidate is the nature of the communist regimes themselves, and the ways in which these impinged upon the life experiences of individuals. Referring back to Sapolsky's chain of effects, the Soviet world was relatively egalitarian in terms of income but had a highly structured political hierarchy in which power relationships were made very clear to all concerned – as they are in the Serengeti. The privileges of the *nomenklatura* and the influence of the various organs of state security assured that the experience of rank was quite intense, even though not expressed in relative income. 'Unequal access to perceived benefits of organized society may not have been adequately described by income distribution data...despite low income differentials there were considerable socioeconomic differences in health in the former socialist countries.' (Marmot and Bobak, 2000, p. 131).

Social supports and informal networks can buffer the stresses of such a situation. But the state apparatus had either absorbed or suppressed other and potentially competing forms of social relationships, leaving the society organizationally impoverished in the levels between the family and the state. The state then tried to occupy this gap with institutions of its own.

These institutions could be threatening rather than supportive to the individual. Wilkinson (1996b, p.203) notes 'the increase in, or return to, the use of informers and secret police on a large scale that took place throughout Eastern Europe.' following the crushing of the 'Prague Spring' and the enunciation of the Brezhnev Doctrine in 1968.[39]

Prior to that time rapid economic growth – faster than in the West – was permitting improved material conditions, while 'de-Stalinization' held out hope of a continuing relaxation of political controls and a wider scope for individual expression. Going back to the Whitehall studies, one of the great benefits of superior rank is a greater sense of control over one's own actions and life. In the West that generally comes with money and social position; in the East it required political liberalization. When both material gains and political liberalization stopped, so did the improvements in health. The coincidence in time, toward the end of the 1960s, is quite remarkable.

39 The Soviet Union would not remain inactive in the face of 'anti-socialist degeneration' in the Soviet Bloc – i.e. would intervene to suppress any liberalizing tendencies.

The political and economic stagnation of the Brezhnev era superimposed a loss of confidence in the Soviet-type regimes as a whole on top of the renewed threats to individual security and suppression of autonomy. Wilkinson (*ibid.*, p. 202) refers to the pervasive lethargy, cynicism, and social corrosion described in Gorbachev's historic address to the Central Committee of the Soviet Communist Party (January 28, 1987). Loss of confidence in the future, and mistrust of both public institutions and other individuals (social isolation), could be potent psychosocial contributors to declining population health. Again consistent with this interpretation, health conditions in the East actually stabilized or improved in the late 1980s, after the announcement of the Gorbachev reforms (Cornia and Paniccia, 2000, p. 19).

AFTER THE FALL: ECONOMIC AND HEALTH COLLAPSE

Then, of course, the regimes collapsed, taking their economies down with them, and life expectancies dropped throughout central and eastern Europe. Again the 'usual suspects' – deteriorating lifestyles (smoking, heavy alcohol use, poor diet), environmental pollution, collapsing medical systems, and general material deprivation – have been suggested as explanations. These are analysed by Cornia and Paniccia (2000: p. 27-34) under the heading 'Misplaced or partial causal explanations' before they conclude that the key factor is acute psychological stress. The health effects of disruption of hierarchy among the Wake Forest macaques suggests an obvious parallel.

As in the pre-1990 period, the worst mortality experience was observed, not among the groups typically labeled as 'vulnerable' – the elderly, children, females – but among working-age males (McKee and Shkolnikov, 2001). Losing the status and sense of self-worth associated with position in the previous hierarchy, they appeared less able to fall back on family and informal social networks both to cope with material shortages and to provide some alternative justification for their existence. Other groups with weaker social networks – the unmarried, migrants, the less educated, urban rather than rural dwellers – have also experienced greater increases in mortality.

The subsequent mortality experience of the various formerly communist countries has, however, been sharply divergent. Some – the Czech republic, Slovenia, Slovakia, Poland – have managed the massive transition to a market economy and political democracy relatively successfully. Correspondingly they experienced '... a

modest and temporary rise in mortality followed by a sharp decline' (Cornia and Paniccia 2000, p. 5). Notably, in these countries male life expectancies rose somewhat more than female between 1991 and 1996. Farther east, in Russia and republics of the former Soviet Union, life expectancies continue to decline – with males still falling faster than females. Russia in particular has also experienced a massive increase in income inequality along with a devastating decline in average incomes. There is plenty of room for psychosocial, neo-materialist, and paleo-materialist explanations to operate simultaneously. Between these groups – geographically, economically, and in terms of mortality trends, are the countries of eastern Europe. The connection between mortality, and degree and duration of social and economic disruption is quite straightforward.

But the obvious geographic pattern to these experiences is also intriguing. The farther east, the more prolonged and severe are both the social and economic disruption, and the increases in mortality. This suggests differences in the resilience and adaptability of social institutions and/or cultural attitudes, independently of the common experience of Soviet-style political and economic regimes. The countries geographically farther to the west were also more 'westernized,' with a richer and more modern array of social institutions, prior to their incorporation into the Soviet Bloc. These differences have deep historical roots; as Hertzman (personal communication) has noted the differing national experiences in the post-Soviet era correlate quite well with whether a country was previously incorporated in the Russian, the Austro-Hungarian, or the German Empires.[40] Institutional and cultural differences go back centuries, and have not been erased by fifty years of Sovietization.

The message for those with a serious intention to reduce social inequalities in health is that these may be 'socially embedded' in regional and national patterns of institutions and culture that have persisted over very long periods of time, and may not be quickly changed. As is now quite widely recognized, preaching improvements in individual behaviour is likely to achieve little. Effective interventions will have to change, permanently, the characteristics of the communities in which people live and work.

40 The inter-war period of independence for these countries was both brief and subject to the acute political tensions of location between Nazi Germany and Soviet Russia, a short-term hiatus between longer-term regimes.

That said, however, it remains true that the endpoints of the pathways linking social context to individual health, illness, and mortality have to be inside the individual body. The pattern of mortality trends, between 'east' and 'west' and among the different countries of 'the east' is also consistent with the evidence emerging from animal studies. Prolonged exposure to a social environment of uncertainty and unresolved psychosocial stresses results in biological embedding of an unhealthy stress response, in the form of elevated basal cortisol levels and reduced ability to turn off the response to particular stresses. Damaging in itself, this state of continuing low-level 'arousal' makes one even more vulnerable to the impact of a particularly severe increase in the stress level – as in the social disruption following the collapse of the Soviet regimes.

'BIOLOGICAL EMBEDDING' EAST AND WEST

This interpretation implies that there should be systematic differences in the physiological responses to stress observed in 'eastern' and 'western' populations. And, in fact, evidence of such a difference has emerged.

The LiVicordia Study of ischemic heart disease (IHD) (Kristenson *et al.*, 1996; Kristenson, 1998; Kristenson *et al.*, 1999) was motivated by the observation that in 1993 the reported mortality rate from IHD among 50-year old men was four times higher in Lithuania than in Sweden.[41] Yet twenty years earlier the rates had been similar. The LiVicordia Study collected data between October 1993 and May 1994 on two randomly chosen samples of 50-year old men (without serious acute or chronic disease) in each of Vilnius and Linkoping. The study examined the prevalence of both 'traditional' and psychosocial risk factors for heart disease.

The traditional factors – smoking, blood lipids, hypertension – were quite similar in the two populations, confirming findings from previous 'east-west' studies. (Recall also the minimal role of these 'Framingham factors' in accounting for the Whitehall gradient.) But there were very significant differences in psychological factors. The Vilnius men showed 'a picture of low self-esteem, low coping, low sense of coherence, and low social support both at leisure and at work.' (Kristenson *et al.*, 1996, p. 192). The Linkoping men, on the other hand, scored higher on

41 Official rates are subject to over-reporting; the actual difference may be closer to three times.

measures of hostility and 'immersion' (high work demand, ineffective coping, and a strong need for control) – characteristics that have been found to be associated with heart disease in western countries.

In addition to the questionnaire survey, both groups were also subjected to a set of laboratory stress tests designed to measure the extent and time pattern of their cardiovascular and hormonal responses (Kristenson *et al.*, 1999). In both groups, the stress tests produced a temporary rise in heart rate and blood pressure, but there was no significant difference in pattern. (The Vilnius men did show a somewhat higher initial blood pressure.) The interesting results were in the hormonal responses.

The Vilnius men had on average a higher concentration of cortisol in both bloodstream and saliva at the beginning of the test (baseline).[42] But they showed a markedly smaller and slower increase in concentration in response to stress. Just like Sapolsky's (1990) subordinate baboons, their responses to stress along the hypothalamic-pituitary-adrenal (HPA) axis had been blunted. These observations are consistent with 'biological embedding' of a physiologically maladaptive stress response through frequent and unresolved stressful experiences and/or a less supportive and buffering social environment. '...[C]hronic exposure to increased endocrine response, may act as a predisposing factor for harmful effects of acute stressful life events (*ibid.*, p. 122). The Swedish men, in contrast, reacted more like dominant baboons.

The authors note the consistency of their findings with those of other studies of human and animal populations that have shown an attenuated stress response among highly stressed individuals. As they point out, the form of stress matters. 'Cortisol secretion, originally characterised as a non-specific response to any stressor, is now seen as specific to situations described as unpleasant, threatening, or involving elements of helplessness or loss of control.' (*ibid.*, p. 111). The extreme stress experienced by the orchestra conductor, the alpinist, or the fighter pilot – groups not noted for lack of self-esteem or feelings of helplessness – does not lead to psychosocial strain and physiological damage, to being 'bent out of shape.'

42 The difference was significant for subjects tested at 7.30 am, but not among those tested at 9.30 am. There is a normal diurnal pattern to cortisol concentration – subjects from both cities showed higher concentrations at 7.30 – indicating the care necessary to ensure truly comparable tests.

But being lower in the social order, does. '...[W]ithin the cities, men in the low socioeconomic groups showed, as compared to men in the high socioeconomic groups, the same differences as Vilnius men relative to Linkoping men, i.e. higher psychosocial strain, higher baseline (in Vilnius only) and lower cortisol response.' (*ibid,*. p.121). The findings of the Livicordia Study thus put in place a link between the physiological findings from the animal studies described above, and the studies showing a powerful connection between mortality and social context in human populations. It confirms a biological pathway linking inequalities in health to social disadvantage, in material circumstances well above dire poverty and direct threats to survival.

The connection between the differing health experiences of east and west, and the differences across the social spectrum in western societies is also made by Cornia and Paniccia (*op. cit.*, p.12) '...the pattern of the mortality rises recorded recently in the European economies in transition resembles strikingly the 1980 pattern of excess mortality of the population of Harlem, a low-income New York neighbourhood (McCord and Freeman, 1990)...between 1960 and 1980 the death rate of the 25-64 year-olds rose steadily...The excess mortality was concentrated in the male population of 35-64 years old...' They suggest that the similar mortality patterns reflect similar social environments – 'a process of social disintegration characterized by a soft state and weak civil society.' Sen (*op. cit.*) likewise refers to 'social malaise and disruption' as explaining the higher mortality among urban African Americans more generally, relative not only to their white counterparts, but – despite their much higher average incomes – to residents of much less economically developed states and nations.

Our rapidly advancing understanding of the determinants of health is thus weaving together a diversity of biological, epidemiological, psychosocial and political/economic observations into a relatively coherent story. But it is not a simple one. As Sapolsky's 'flow chart' (Figure 11) makes clear, there is no hope of a monocausal explanation for health inequalities, 'this and not that.' A more accurate characterization would be 'it's everything, all the time'.

Advancing knowledge has thus served only to magnify the challenges faced by those assigned the task of recommending or implementing policies to reduce health inequalities. The evidence continues to strengthen, that patterns of health in a particular society are deeply rooted in the social and economic structure of that society. That same evidence suggests that there is no limited set of well-

defined policies to change these deep-rooted patterns. Furthermore, to the extent that the behavioural and biological responses to stress are biologically embedded in early life, the time horizon for significant impact may be measured not in years but in decades, or even generations. One longs for the simplicity implicit in Wilkinson's original 'Big Idea' – 'It's the income distribution, stupid!'

THE ACHESON REPORT, AND THE U.K. GOVERNMENT'S RESPONSE

These realities are reflected in the frustrating generality of so many of the recommendations in the Acheson Report (Independent Inquiry, 1998). 'We *recommend* the development of policies to...' What policies? Developed by whom? Precisely who is to do what, and with which, and to whom? Nor do the authors of the Report set priorities among their recommendations, or deal with issues of affordability and cost-effectiveness.

Yet one is hardly justified in criticizing the authors on these grounds. They were fully aware of the state of knowledge on the determinants of health, having themselves produced some of the most powerful evidence. Research is generating increasing confidence as to where we need to go, but is much less helpful as to how to get there. As Birch (1999, p.304) points out in a (rather critical) commentary:[43] 'In practice, we know very little about how to remove or reduce inequalities in health.'[44]

Accordingly the Terms of Reference for the Independent Inquiry instruct them to identify '... priority areas for future policy development ... likely to offer opportunities for Government to develop ... interventions...' It was recognized from the outset that

43 Birch emphasizes the role of the 'social context' in modifying the relationship between health determinants and health outcomes, so that no 'clear and stable relationship' exists 'out there' waiting to be discovered. The point is a good one; illustrated by the finding of the Whitehall studies that the relation between smoking and health is weaker at higher socio-economic levels. The contrasting Canada and U.S. data in Figures 2 and 3 make the same point very clearly. Yet the authors of the Acheson Report knew this – one of them (Michael Marmot) is the leader of the Whitehall studies. A recommendation to 'change the social context' is, however, of limited usefulness.

44 The scientific advisory group commissioned by Acheson quickly discovered this rather awkward handicap (Macintyre *et al.*, 2001).

effective interventions will have to be worked out on the ground, consistent with the broad targets indicated by the Report.

A firm government commitment to reducing health inequalities is, after all, a very new situation. While the Report's recommendations may have been 'remarkably similar in scope and content to the recommendations of the Black Report' (Birch, *ibid.*, p. 301), the reception by government has been rather different. Prime Minister Thatcher's efforts to suppress the Black Report were spectacularly unsuccessful, but her government's hostility at least ensured that nothing would be learned about how to make concrete and implement as programs the ideas behind its recommendations. That learning process is now beginning, nearly twenty years later, and the guiding principle now will have to be strategic consistency, but tactical flexibility.

Under these circumstances, it is hard to see how anyone could set priorities and estimated costs on the basis of '... scientific and expert evidence...'. But the evidence suggests a deeper problem with 'prioritization'. The 'social environment' is a label for a large, complex, and highly interactive set of processes, acting over the whole life course, to influence the various forms of stress to which people are subject, the resources they have available for coping with these stresses, and their degree of resilience or vulnerability. Stresses appear to act cumulatively and often multiplicatively, while different forms of coping mechanisms can substitute for one another, both contemporaneously and over time. If the sources of health inequality are 'everything, all the time', attempting to identify the most important factor or factors is probably a meaningless exercise.

The necessity of addressing health inequalities across a broad front is reflected in the subsequent actions and proposals of the government of the U.K, summarized in *From Vision to Reality* (Department of Health, 2001). In no other country has the government identified health inequalities as a major focus of policy concern, and none has as rich and extended a research tradition on which to draw. What has happened so far, as research, filtered through recommendations, meets (political) reality?

THE DOG IN THE NIGHT-TIME

Perhaps the most striking thing about both Acheson and *Vision* is what is *not* there. One of the most important findings of research on

inequalities in health is that of the social gradient, the observation that differences in health extend across the whole socioeconomic spectrum. Top people are, on average, healthier than those just below the top, who are in turn healthier than those just below them, and so on. Health inequalities are not just a problem of the poor, of 'those people down there (of whom, thank goodness, I am not one)'. *De te fabula narratur.*

This point is re-emphasized by Marmot (1999), commenting on the report he helped to prepare. Its recognition has been of fundamental importance in shaping the present understanding of the determinants of health, and escaping from an exclusive focus on poverty and material deprivation. Moreover, although people at the bottom of the socioeconomic spectrum suffer most from health inequalities, the aggregate burden of ill-health associated with social inequalities is likely to be much greater among the broad mid-range of the population – simply because this group is much larger. '...[M]easures at the tail end of the distribution that yield possibly large benefits to sick or at risk individuals produce little change in the overall health of the population...' (McKinley and Marceau, 1999).[45] There is also the practical political consideration, that a government commitment to reducing social inequalities might be easier to maintain, if they were understood to threaten a large proportion of the population.

Yet the socioeconomic gradient makes no appearance in the recommendations of the Acheson Report. Recommendation 3 speaks of 'policies which will further reduce income inequalities', but the specifics all concern increases in social benefits to raise the incomes of the poorest (Independent Inquiry, 1998, p. 120). Other recommendations are concerned with unemployment, and education and skills training, but the focus throughout is on alleviating poverty and disadvantage, not on flattening the income distribution *per se*. Whatever the understanding of its authors, the Report's recommendations seem rooted in the view, still quite widespread despite the counter-evidence, that there is some sort of poverty threshold above which the relation between income inequality and

45 Conversely Rose's prevention paradox (Rose, 1992, p. 3) states that '...a preventive measure that brings large benefits to the community affords little to each participating individual.'

health inequality either does not exist, or does not matter. This is a long way from the Whitehall studies, as well as from Wilkinson.[46]

The government's responses correspondingly include increasing various forms of public transfers for the poor and disadvantaged and raising the minimum wage. There is no suggestion of a more general use of tax and transfer policy to reduce income inequalities across the entire spectrum. Yet other U.K. governments have not been so timid about manipulating the income distribution.

As Atkinson (1999) points out, in the late 1980s Prime Minister Thatcher's government deliberately engineered a significant increase in income inequality in the U.K. through changes in tax and transfer policy. Prior to that time, students of trends in income distribution had found the subject about as exciting as watching paint dry, or grass grow; and there was some suspicion that broad distributional patterns were both extremely stable over time, and rather insensitive to public policy. Mrs. Thatcher (as usual) shattered the conventional assumptions. A determined government can, if they choose, change quite significantly the degree of income inequality. The Blair government, however, has obviously chosen to leave this issue alone. That dog has not barked.

On the other hand, *From Vision to Reality* places a great deal of emphasis on the government's program for reorganization and financial revascularization of the National Health Service, with a particular emphasis on programs of 'Action against the Big Killers' – heart disease, cancer, and mental illness. More people, more money, more equipment, better training, and better organization are promised to improve access to high quality care, reduce waiting times, and 'save lives'. Yet more and better health care, whatever health benefits it yields for particular individuals, does not necessarily reduce health inequalities. That is a crucial message from the historical data in Figure 4, and from the Black Report.

The Whitehall studies (Figure 10 *supra.*), for example, and the LiVicordia study (Kristenson, *ibid.*) both showed that the very large differences in heart disease rates in their study populations were *not*

46 It also risks giving aid and comfort to those who, in the teeth of the evidence, cling to the notion that even in wealthy countries health inequalities arise from absolute rather than relative deprivation. One fork in this road can lead to 'economic growth *über alles*', and let the rising tide lift all the boats, though in fact nothing could be farther from the spirit, or the evidence, of the Acheson Report.

accounted for by differences in rates of hypertension, or average levels of serum lipids. Yet it is these factors that receive enormous attention from clinical medicine, and particularly from the producers and prescribers of drugs. If differences in hypertension and blood lipids are not major contributors to the socioeconomic gradient in health, then one can hardly expect expanded medical therapy to flatten this gradient. As for promoting general improvements in population health, the relative effectiveness of modern health care has undoubtedly improved since the era analysed by McKeown. But it is generally recognized that the burden of excess morbidity and mortality implicit in social inequalities in health is even greater than that associated with the 'Big Killer' diseases.[47]

The Acheson Report, unlike *Vision*, has relatively little to say about health care, and what it does say focuses on ensuring 'equitable access to effective care in relation to need' (Recommendation 37, p. 129). Impossible to contest in principle – though frequently sabotaged in practice – this objective implicitly links health inequalities to inequitable access and leaves open the question of overall adequacy of resources.

Such a linkage seems highly plausible in the U.S., where access to care is so closely linked to economic position.[48] The two-tier structure of British health care might also create such a link – Recommendation 38.4 calls for '...a review of the relation of private

47 The comparison is not, of course, on all fours. When people at lower socioeconomic status die earlier, they typically die of one of the 'killer diseases'. The question is, should one tackle the disease itself, or the underlying, 'upstream' social contexts associated with greater vulnerability? Attacking the disease itself seems intuitively to be the most immediate priority; the historical persistence of gradients despite the dramatic transformation in causes of death suggests that this intuitive response may be misguided.

48 Baker *et al.* (2001), for example, find in their recent study of middle-aged American adults that those uninsured or intermittently insured are at significantly higher risk of a major deterioration in health. Ross and Mirowsky (2000), however, found that (private or public) health insurance coverage was associated with increased numbers of physician visits, and particularly pharmaceutical prescriptions, but not with improved health. They argue that any health benefits of insurance may come not from increased access to and use of care but from the relief of economic hardship associated with costly medical care. All such studies, however, face the problem of adjusting for background factors that may be associated with both absence of insurance cover and ill health, such as unemployment or indeed ill health itself.

practice to the NHS with particular reference to access to effective treatment.' (*ibid.*). But the data from Canada, where no private tier exists, demonstrate that while 'universal access on equal terms and conditions' is associated with significantly greater rates of care use at the lower end of the income distribution (e.g. Katz *et al.*, 1997, Mustard, Shanahan *et al.*, 1998; Mustard, Barer *et al.*, 1998; Finkelstein, 2001), it has not removed the gradient in health status (e.g. Roos and Mustard, 1997; Wilkins *et al.*, 2001). Health inequalities have deeper roots.

SOMETHING OLD, SOMETHING NEW... NOTHING TOO COURAGEOUS

To get at those roots, both Acheson and *Vision* offer both the extension of classic public health policies, and the introduction of a much less specific set of initiatives that might be described as efforts to change the social context and influence the process of biological embedding.

The former are mostly easy to define, and their benefits largely beyond (serious) question. The Acheson Report states bluntly that drinking water supplies should be fluoridated (Recommendation 22.2, p. 124) – and so they should. The government apparently agree – but not just yet. They would like to receive still more evidence (!!!), after which they '...will be encouraging local authorities with particular dental health problems to consider fluoridating...' Faced with the opportunity to make a 'courageous' decision, they appear to have chosen instead to delegate and disappear.[49] Let the local authorities take the flak from the lunatic fringe.

On the other hand *Vision* does contain a number of specific measures aimed at improving diets, and reducing environmental pollutants. Unlike fluoridation, such measures do not have (overt) political opponents. They obviously will, however, have economic implications for particular industries, and it will be interesting to see how they play out in practice.

It would be churlish not to applaud such efforts. There does seem to be a good deal of evidence that many, perhaps most, people would benefit from eating more fruits and vegetables, and less fat and salt.

49 In the finest tradition of 'Yes, Minister.'

Under these circumstances the annual destruction of millions of kilotons of fruits and vegetables, at enormous public expense, stands out as stark lunacy. The identification in the Acheson Report of the Common Agricultural Policy as a major barrier to healthier diets would seem worthy of more serious attention. It receives none in *Vision*; lunatics have their reasons.[50]

With respect to health inequalities, however, it may be significant that, as noted above, the predominant view among analysts is that neither poor diet nor severe air and water pollution can account for the emergence of the east-west mortality divide. Each has been put forward as an explanatory factor, but neither is supported by the evidence. While undoubtedly good diets and clean air and water contribute to health, it has been the deterioration of the broader social environments in which people live and work that lies behind the remarkably poor health experience of 'the east'. If the same mechanisms are at work in generating health inequalities within 'western' countries, improvements in diet and physical environment may again not touch their roots.

One aspect of the broader social environment that receives a good deal of attention from Acheson is transport policy – five out of 39 recommendations. These include the development of high quality public transportation systems, measures to encourage cycling and walking, and efforts to reduce the usage of motor cars and reduce traffic speeds, along with concessionary fares for the elderly and disadvantaged. The objectives here include but run deeper than concerns with road safety and various forms of pollution.

The Acheson recommendations draw on a belief – perhaps difficult or impossible to document with hard evidence – that patterns and processes of mobility are a major component of the social environment, and that an automobile-based society is fundamentally different from one with extensive, high-quality and well-used public transport. The latter is a society of public spaces, shared on equal terms by all citizens. The automobile, on the other hand, is intensely private, isolating people from each other, and intruding relentlessly on public spaces, particularly in urban areas (and egregiously in London!). Where it is dominant, what

50 Some of the initiatives in *Vision*, such as the National School Fruit Scheme could, of course, absorb some of the surplus European production that might otherwise be destroyed. But it is the overall Policy that is Acheson's target.

public transport there is is used primarily by those who have to.[51] Both mobility, and the level of stress associated with it, are socially graded.

Individuals cannot by their own choices influence this aspect of the social environment. That requires collective action, which is what governments are for. Yet this central concern in the Acheson Report finds no echo in *Vision*. Transportation has an exiguous presence in references to the benefits of exercise in the discussion of heart disease, and later '...the NHS and partner agencies are asked to develop policies on smoking, healthy eating, physical activity, obesity, workplaces and green transport [and] to report on progress at board level.' But there is no indication of national-level policies to promote public transport, or even recognition of the social consequences of the private automobile. One may or may not agree with the members of the Independent Inquiry as to these consequences (I do), but in the government's response the issue is simply ignored.

Vision takes quite a forward position, however, on the expansion of clinical preventive services – immunization and screening. These are not explicitly included in the Acheson recommendations, though they would presumably be subsumed under equity of access to effective care. Such activities are undoubtedly laudable, though it is not clear that one needs any extended exploration of evidence on the social determinants of health to justify promoting them.[52]

Unlike water fluoridation, however, the impact of clinical prevention on health inequalities depends on patterns of individual uptake. If uptake is correlated with socioeconomic status, then this form of expanded prevention may serve to increase health inequalities while improving overall population health. This is a criticism often brought against health education and promotion programs as well, that their messages are more likely to be heard, and acted upon, toward the upper end of the socioeconomic spectrum where health is

51 London is, of course, *sui generis*. But the negative effect of the automobile on the efficiency of public surface transport is impossible to avoid.

52 A very positive feature of *Vision* is that, though there are a number of references to screening programs underway or proposed, there is none to genetic screening. Genetic screening has enormous potential for the conversion of people into patients, with great commercial but little health benefit. The possibility exists for its explosive expansion, driven by profit-motivated marketing, and the U.K. government are to be congratulated if they can keep that genie in the bottle. (The medical private sector, however, provides grounds for concern.)

already better.[53] There are echoes in *Vision* of the profoundly contradictory slogan of the health promotion movement: 'empowering people to make healthy choices'. What if, once empowered, they continue to make unhealthy choices?

A response such as 'Well, it's a free country' does not meet the evidence. Systematic inequalities in health reflect systematic patterns of 'choice', not just the vagaries of individual preferences. '...[W]e are talking about systematic differences between groups, not simply differences among individuals.' (Marmot, 1999, p. 42): The LiVicordia study, as noted above, found in the Vilnius men a psycho-social pattern of low self-esteem, low coping, low sense of coherence, and low social support. Their social context had embedded in them a psychological (and a physiological) pattern consistent with what could be summarized as a low sense of self-efficacy. If what you do doesn't matter, then why bother? Implicitly, if you continue to make unhealthy, self-damaging choices, you probably are not really 'empowered'.

Which brings us to smoking.

Smoking provides a classic example of (relatively) successful health promotion that has probably, in the process, contributed to widening health inequalities. Population-wide, multi-pronged anti-smoking campaigns may well have been among the factors that have convinced most of those in the middle or upper end of the socioeconomic spectrum to quit, or never to start. In becoming much less widespread, smoking has become primarily a low-SES activity. Overall population health has improved as a result, but inequalities are made wider. Effective public programs targeted directly at those remaining smokers, however, are likely to be more difficult to design and carry out, and may lack a broad public constituency.[54]

53 Some representative examples are provided in Macintyre *et al.* (2001).

54 Some commentators on the Acheson report have criticized its failure to recognize explicitly a possible trade-off between reducing health inequalities, and raising overall health status. Spending money to help one group means not spending it to help another, and if the cost-effectiveness of programs targeted to the least healthy is in fact lower, then logically there must be such a trade-off. But the state of our knowledge on the effectiveness of different programs for different groups is far too weak to give empirical teeth to such a trade-off, and contemplating it in the abstract seems more distracting than enlightening. *Vision* is probably politically wise, if imperfectly logical, in ignoring the trade-off issue and simply declaring a dual objective of reducing inequalities and raising overall health status. Launching a successful smoking cessation program, for example, that nonetheless fails to reach the most deprived in the population, should not be a hanging offense.

The recommendations of the Acheson Report, reasonably enough, are intended to reduce smoking, ideally to zero, by all means possible. Raise the taxes on tobacco (with compensating income supplements for those with lower incomes), eliminate tobacco advertising, restrict smoking in public places, and provide nicotine replacement therapy for would-be quitters. The strategy described in *Vision* only partially adopts this agenda. The government are expanding the standard health promotion approach – anti-smoking publicity combined with greater restrictions on the industry's efforts to recruit new smokers. At the same time they are expanding the scope of clinical prevention by improving access to nicotine replacement products and shifting the focus of smoking cessation into primary care. The National Health Service will be offering more assistance to those who want to quit. To the extent that successful quitting is also socially graded – as it appears to be – this extra assistance may improve quit rates toward the lower end of the socioeconomic spectrum. (This presumes, however, that *wanting* to quit is not socially graded, only the success rate.)

The Government's claim to be providing 'world-leading smoking cessation services' is certainly grounds for some congratulation, particularly if they are effective. But it is the *next* generation of smokers who are key. The tobacco industry well understand that their survival depends upon inducing children to become addicted to their toxic products – few people take up smoking in adult life. What are the features of the social environment of children in the lower SES ranges that make them so much more vulnerable to this commercial predation? If one could identify and act to modify *those*, the dilemma of differential uptake would be at least mitigated, and might largely disappear. Help from the NHS for would-be quitters is wholly to be applauded, but why did they start in the first place? You and I did not. (If you did, have you quit? If not, see your doctor.)

STARTING EARLY: IT'S NEVER TOO SOON

If biological embedding of successful or unsuccessful coping strategies, and development of a corresponding sense of self-efficacy (or lack of it) takes place early in life, then programs that require some form of positive individual response are more likely to be taken up by the already relatively healthy. On the other hand programs targeted at the least healthy may turn out to be the least cost-effective. The cluster of present and past influences that is associated with ill-health also

inhibits the development of more positive responses to information or opportunities. At the same time their funding support must come primarily from those who are not the targeted beneficiaries. They will require broad political support both to maintain this funding, and to overcome the entrenched political opposition of those whose 'pecuniary interest or ... personal convenience' will be threatened.

The recognition of biological embedding of individual responses motivates the consistent focus, in the Acheson Report, on interventions aimed at children and women of child-bearing age. The traditional justification for such a focus has been that these groups are more vulnerable than, say, adult males; their health is more at risk from external threats. Yet much of the accumulating evidence suggests exactly the opposite. It is the adult males who are most at risk from social disadvantage, disruption and stress.

The reason for focusing on children and child-bearing women is that the patterns of biological and behavioural response to stresses in the social environment, and the degree of resilience or vulnerability these confer, are largely set very early in life, and to some extent even before birth. Intervening in the social environments of children is intended to improve the health, not just of children, but of the adults they will later become (and the children they will rear). The child is father to the man.

It is hard to tell from the text of *From Vision to Reality*, how far the evidence on biological embedding and human development has penetrated the governmental policy-making process. The Ontario Early Years Study (McCain and Mustard, 1999), by contrast, traces out explicitly the biological pathways through which early social environment influences the development of the neural system. This early neural development has implications not only for vulnerability to illness (and accident), but also for 'readiness to learn', school performance – and status – and the whole subsequent life trajectory of work and home environment and social position, and through them, health.[55]

While not spelling out the biological evidence, *Vision* does devote a good deal of attention to children. Its specific measures focus on child poverty, traditional clinical prevention (screening and

55 No one imagines that the process is deterministic, but the probabilities faced by the individual are shifted for good or ill.

vaccination), and diet. But the Sure Start program proposes to addresses the whole range of physical, intellectual, and emotional development, prior to school age, and the resources to be committed are substantial.

The description is thin – the program is to be led by local partnerships – but this initiative seems quite consistent with the emerging evidence on child development and its relation to health inequalities. Catch them as early as possible, provide enriched and nurturing environments, and promote their sense of security and confidence in their own capacities. Prevention of illness and accident, and early identification and remediation of specific developmental problems, are necessary but not sufficient for a sure start.

A focus on early intervention to assure children a secure start in life should not, however, obscure the importance of the socioeconomic environment throughout the life course. Analysing the British Birth Cohort of 1958, Power and Hertzman *(op. cit.)* note the cumulative effects on health of community characteristics, labour market conditions, and peer relationships and conclude '...ameliorating social class gradients in health and well-being requires broad social and economic change and not just targeted interventions designed to improve the individual life course.' (p.54).

FROM RESEARCH TO RHETORIC:...TO REALITY? NO CLEAR PICTURE YET

A consistent theme running through *Vision*, much more prominent than in the Acheson Report, is the development of local leadership and local action. A cynic might recall the rhetoric of U.S. president George Bush (the Elder), that 'a thousand points of light shall lead the way.' Local and voluntary initiatives would make the United States a better place; behind the rhetoric the central government would delegate and disappear. The difference, however, is that the U.K. government have explicitly taken responsibility, or at least set targets, for particular outcomes, and propose to provide both significant financing and an organizational framework in which local or regional partnerships and initiatives can develop – and be evaluated for their results.[56]

56 If a thousand points of light are to lead the way, someone has to supply the oil – and make sure that the lights do not drift off as 'feux follets'.

The government's chosen instrument is, as recommended by the Acheson Report, the National Health Service. 'For too long the NHS has been seen as a sickness service not a health service. We believe that the role of the NHS, in partnership across the community, should be to prevent sickness and ill health, as well as treating problems once they arise.' *From Vision to Reality* makes clear that this includes, but goes far beyond, expanding traditional clinical prevention and health promotion. The NHS has been assigned broad responsibilities for coordinating cross-governmental activities at regional level, and for stimulating and leading local and regional partnerships. The new Primary Care Trusts are to have a central role in this process.[57]

So the answer to the major question left dangling in so many of the recommendations of the Acheson Report – 'What policies, developed by whom?' – is 'Whatever policies emerge from variously structured regional and local partnerships (led or coordinated by the NHS, and funded by the central government) and are shown to be successful.' It is quite a task; can the NHS do it?

A cynical North American would fear that the clinical services will simply take the money and run.[58] Action to reduce inequalities and improve the social determinants of health may receive priority, but not resources.[59] On the other hand, the NHS is a very different

57 Interestingly Kindig (1998), after a compact review of the evidence on the determinants of health, proposes a program for extending the scope of the American managed care systems from purchasing sickness care to purchasing health. His central idea is that third party payers – employers and government agencies – would contract for, and pay according to, health outcomes for defined populations, leaving provider organizations to decide how best to achieve those outcomes. It is hard to see, however, how this could work in an institutional environment that permits providers, and often payers as well, to select the populations for whom they will accept responsibility (Ostry and Evans, 1999).

58 Being a 'sickness care system' is, after all, nothing to be ashamed of! Quite the contrary. And the demands on a sickness care system, unlike those that would face a true health care system, are much more immediate and personal. It is easy to understand the typical clinician's response – I need the resources *now*! – but like the biological stress response of the organism, the indefinite prolongation of this response may reduce the long-run effectiveness of the whole enterprise.

59 'We have two classes of boys at this school, Class A and Class B. At mealtimes, the Class B boys get priority.' 'And the Class A boys?' 'Oh, they get food.' – *Ancient Goon Show script.*

institution from anything west of the Atlantic, and the political and managerial structure may be able to hold in check Wildavsky's (1977, p. 109) 'Law of Medical Money'.[60] And *Vision* recognizes clearly the need to strengthen public health, upgrading the numbers, skills, and resources of those who have the deepest understanding of, and concern for, the role of the social environment in health. Finally, is there another sector of government that would be better suited to the task?

The NHS may have the scale, resources, and staying power to take on or lead the job of translating the emerging evidence on the sources of health inequalities into concrete and effective policies, a job that is clearly yet to be done. The Acheson recommendations do not achieve this – they merely call for such policies to be developed. And the government's specific policy responses so far, as reflected in *Vision*, have been rather loosely linked to the emerging evidence.

The most specific and concrete initiatives have been very traditional – 'more for the poor', and a lot more spending on clinical medicine, including clinical prevention. The central role of the broad social gradient, *not* just 'poverty', that emerges so clearly from research on the determinants of health, has vanished from the policy response. That research also points to the very limited role of clinical medicine, both in advancing overall population health, and particularly in reducing inequalities. For a government that has put a good deal of emphasis on 'evidence-based' policies in health, this is a surprising disconnect.

What happened?

Two major considerations constrain efforts to turn the findings of research on health inequalities into policy. Crudely put, we do not know how, and we are not sure we really want to.

While researchers have an increasingly good appreciation, in general terms, for where the origins of health inequalities lie, we still do not know, as Birch *(op. cit.)* points out, what to do about them. And the public commitment to doing anything about them is in reality not nearly as strong as rhetoric might suggest. There are deeply-entrenched and powerful social interests, both narrow and broad, that find the present 'social environment' quite satisfactory, thank you, and that might be threatened by effective action.

60 'Costs will increase to the level of available funds . . . that level must be limited to keep costs down.'

These constraints interact. A unified and determined population could probably figure out fairly quickly what works and what does not – learning by doing, abandoning failure and expediting success. Alternatively, if the evidence were overwhelming – not just as to the importance of socioeconomic factors for health, which *is* overwhelming, but as to the effectiveness of specific and feasible interventions – then the opposing interests could probably be overcome. But lack of knowledge of specific policy effects provides excuses for those who do not really want to address the issue of health inequalities in any fundamental way, who deplore their existence and are willing to render all possible assistance short of actual help.

The association between income inequality and health inequality, for example, is now pretty well established, at least in comparisons across countries, but the causality is less clear and the 'Big Idea,' that greater income inequality *per se* is associated with lower *overall* health status, is running into increasing problems with the evidence. Meanwhile, in many industrialized nations the incomes that people earn in the marketplace have become increasingly unequal over the last two decades (Atkinson *et al.*, 1995; Smeeding, 2000).[61] In the U.K., the Thatcher government changed tax and transfer policies to accentuate the impact of this trend on family disposable incomes – and was re-elected. Canadian fiscal policy, in contrast, leaned against the market wind until the mid-1990s, moderating the after-tax impact of increasing inequality. The United States government took a neutral stance. But in the last five years, North American governments have brought in major tax cuts designed to increase significantly the share of disposable income received by the better-off and especially by the very wealthy.

It is an interesting speculation as to why populations in the English-speaking world have become so much more generous toward their richest members, and so much less so toward the poorest. The role of the media, and of the wealthy ideologues who own so many of them, is of particular interest.[62] How many people actually know

61 This trend is not, however, found in all developed countries, leaving open a question as to whether it is due to fundamental changes in patterns of economic activity, or to changes in the public regulatory framework.

62 Also of interest is the rightward shift in ideology among professional economists in government and out, a shift based neither in economic theory nor in empirical understanding, but possibly related to income opportunities.

what is going on? – the realities are buried in bafflegab, if not outright lies. But however one interprets this phenomenon, it is hard to argue that there is widespread, or at least politically effective, public support for flattening the income distribution. *Vision* studiously avoids the whole issue.

On the other hand, despite considerable scepticism among students of health and health care as to the benefits of a general expansion in the clinical services, the level of public support is universally high. Reversing Rose's prevention paradox (note 44 *supra.*), clinical measures that have little impact on population health may afford large benefits to particular individuals. These potential individual benefits appear to have far more impact on public opinion and public policy than more abstract considerations of population health. We know people; who has ever met a population?

Such attitudes are shared and stimulated by those who provide clinical services. They care for individuals, not populations; moreover they receive health spending as personal income. Expanding the clinical services thus has the very active encouragement of strategically placed and influential interest groups, as well as a broad base of public support. Income equalization, by contrast, has the very active opposition of strategically placed and influential minorities, and (whether from indifference or ignorance) no obvious base of public support.[63]

To some extent the broad public commitment to reducing health inequalities is weakened or diverted by 'them', narrow but powerful interest groups that have interests and agendas independent of or directly in conflict with that objective. The tobacco industry, for example, very narrowly-based but well-resourced and strategically-placed, is a 'politically potent pathogen' that has been remarkably successful world-wide in resisting and frustrating a broad consensus for decisive action against smoking. The industry has few friends, but

63 In the American presidential campaign of 2000, Vice-president Gore raised the issue of increasing income inequality. Governor Bush successfully dismissed this as an attempt to stir up 'class war'. In reality he was preventing the stirring up of awareness, on the losing side, of a class war already well underway. As president, Bush has introduced massive tax cuts whose main beneficiaries are the wealthy – in the most inegalitarian country in the developed world, and after many years of remarkable accumulation among the top one percent of the U.S. population. But there is no sign of a backlash.

those include the advertising industry and the media that they feed – and who knows who else? (And in any case the children they prey on are now typically from well down the social ladder.)

But in other cases 'they' look rather more like 'us', or at least quite a number of us. The Acheson Report, as noted, makes quite a strong case for more and better public transport, and indeed more walking and cycling. The deleterious effects of the automobile, in aggregate, are as hard to question as to avoid. But who will be first to '...yield something of ...personal convenience...' and give up the car? The transportation problem is akin to the famous Prisoner's Dilemma' – unless all act together, those who do 'the right thing' are at a disadvantage relative to those who do not. Why don't you all take public transport, leaving the roads (and the air) clear for me and my car? (Only don't ask me to pay more taxes to improve your public transportation system – I'm not using it. Maybe it should be privatized.) The strong emphasis placed on transportation in the Acheson Report has disappeared from *Vision*.

Narrow interests and broadly based patterns of behaviour may interact. Producers of convenience foods overload them with salt, sugar, and fat – deliberately promoting unhealthy diets – not only because these ingredients are cheap, but also because they sell. If most people rejected these foods in favour of organically grown vegetables, suppliers would respond.[64] Attempts to regulate food content can be expected to meet resistance from the food industry if they threaten sales, and would probably receive at best half-hearted support from their intended beneficiaries. It is one thing to put iodine in the salt and vitamin D in the milk – (almost) no one notices. But pulling out the salt or the sugar is another matter. And if dietary quality is correlated with socioeconomic status – with education and income – then that feeds (so to speak) into health inequalities.

All in all, the U.K. government's approach thus far to the issue of health inequalities seems to be rhetorically powerful, but politically very cautious. It has ducked not only big issues like the overall distribution of income and the national transportation structure, but even water fluoridation – a compact, well-defined intervention where

64 This is, of course, too simplistic and 'econo-mystical' – advertising budgets for unhealthy foods are gigantic because they are corporate products. Broccoli does not really compete on a level playing field with McDonald's hamburgers. But what about the local chip shop?

the evidence of benefit is overwhelming. It has thrown considerable new resources into rather traditional areas – clinical medicine and clinical prevention, anti-smoking campaigns – that will draw much political support and little or no opposition. Better nutrition for disadvantaged school and pre-school children would also be a hard issue around which to mobilize political opposition. More generally, efforts to alleviate poverty, particularly among children and the old, combined with greater educational and employment opportunities for those in the working years, tap abroad base of public support.

Little of this, however, can be said to draw on the evolving research on the determinants of health, on the role of the social environment in embedding differential patterns of stress response that underlie the social gradient. The translation of these new understandings into concrete programs and policies is work yet to be done.

The U.K. government have attacked this much larger task primarily with initiatives intended to stimulate and lead (and finance) processes, rather than by launching programs. The major emphasis is on putting together local partnerships, through quite a wide variety of different schemes, to generate specific interventions in specific regions. (The Sure Start program is perhaps an exception, since its focus and objectives (and perhaps its methods?) seem already set.)

Such an approach has several advantages. Since not much is known about how to 'change the social environment' so as to reduce health inequalities, drawing in a lot of people to try different things in a number of different areas seems like a sensible way to find out.[65] Secondly the assembly of local partnerships to formulate local initiatives simultaneously creates a constituency of support, to stand beside or in front of the central government in responding to opposition and criticism. And the process of partnership building will also be an educational one, an opportunity to spread much more widely the broader understandings of the sources of health inequalities.

But will it work? Local partnerships are at risk of diversion or loss of focus, or simple loss of cohesion and energy. The NHS leadership role is critical to ensure that too much tactical flexibility does not lead to loss of strategic consistency – to make sure that everyone's eyes are

65 An alternative, the large-scale and carefully designed and evaluated social experiment, is very expensive and time-consuming and may be difficult or impossible to generalize.

kept on the ball. The central government will also have to take very seriously the process of measuring and evaluating performance – delegate, but don't disappear. This includes the political courage to abandon failure, as well as to expedite success.

In tackling inequalities in health, the government have embarked on a long-term process. Setting its specific targets a decade away, in 2010, is not at all unreasonable. One might hope to see earlier effects, particularly among children, but in fact one of the most widely quoted studies of early childhood intervention showed its most dramatic (and positive) results in early adulthood – twenty and more years out (Schweinhart *et al.*, *op cit.*).[66] The 'east-west' comparative studies have shown that dramatic changes in the social environment can have large and quite rapid effects on health, but we have no similar examples showing *improvement*. (Mobilization for the Second World War may be one such, but not one of any help to the present government. The reduction of health inequalities is not a matter of national survival.)

So again, is the vision really in process of being translated into reality? *From Vision to Reality* presents a broad array of initiatives, but with a strong flavour of political caution and a considerable weight of traditional approaches. The really difficult work remains for the multitude of local and regional partnerships that they are only in process of establishing. Marmot *(ibid.*, p.44) wrote two years ago: 'That the government cared about this issue sufficiently to set up an Independent Inquiry is an important first step. It will be vital to monitor what happens next.' That remains true.

66 Even this is a rapid effect relative to studies showing an impact of nutrition in utero on heart disease rates fifty years later.

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