

# IMPROVING POPULATION HEALTH IN INDUSTRIALISED NATIONS

Based on papers delivered at the OHE  
Conference, London, 6 December 1999

Edited by Jon Sussex

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Based on papers delivered at the OHE Conference,  
London, 6 December 1999: 'The Causes of  
Improved Population Health in Industrialised  
Nations: How will the 21st century differ from  
the 20th?'

Edited by Jon Sussex



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- commission and undertake research on the economics of health and health care;
- collect and analyse health and health care data from the UK and other countries;
- disseminate the results of this work and stimulate discussion of them and their policy implications.

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# Chapter 1

## McKeown updated

JON SUSSEX AND PETER YUEN

### Introduction

Many factors influence the health of a population but their relative importance is a matter of controversy. Further, the most important drivers of reduced population mortality and morbidity over the next 100 years in economically developed countries may differ from those that dominated in the twentieth century. So where is the best place to invest our effort over the next 100 years if we want to improve population health the most; and what can the past 100 years' experience tell us? The purpose of this and the following chapters is to provide information, analyses and insights to help answer this question. The book is based on papers presented and discussed at the Office of Health Economics Conference held at the Royal Horticultural Halls Conference Centre in London on 6th December 1999.

### Thomas McKeown

The current debate about the causes of long-term population health improvement has been heavily influenced by the works of Thomas McKeown, the eminent social physician and epidemiologist who died in 1988. His arguments are summarised in two books that have become classics in their field: *The Modern Rise of Population* (1976) and *The Role of Medicine* (1979). The later book was based on McKeown's 1976 lecture of the same title for the Nuffield Provincial Hospitals Trust, and used the same data as *The Modern Rise of Population*. You do not have to agree with everything McKeown wrote to be impressed by the sweep and clarity of his analysis. By adding to his data the information that has become available over the quarter of a century since the early 1970s, we can assess to what extent McKeown might have modified his arguments had he been writing today.

Driven largely by the limitations of available data and by his even longer timescale than the past century considered in this book,

McKeown focused on death rates in England and Wales. The measured, large, decline in mortality from the mid-nineteenth century to 1971 was, he said, ‘the main evidence of improvement in health’ and ‘was due essentially to a reduction of deaths from infectious diseases’ (McKeown, 1979, pp.8-9). He put the defeat of infections largely down to improvements in nutrition, in the environment in which we live – by which he meant particularly the safety of water and sanitation, and food supplies – and to changes in personal behaviour concerning smoking, diet, exercise, etc. The contribution of personal medical measures over the long period from the mid-nineteenth century to 1971 was in his view relatively minor in comparison. McKeown knew the huge problems with the inaccuracy of the data he was using; not least whether recorded causes of death bore any resemblance to the true causes. Nevertheless, he drew strong and clear conclusions.

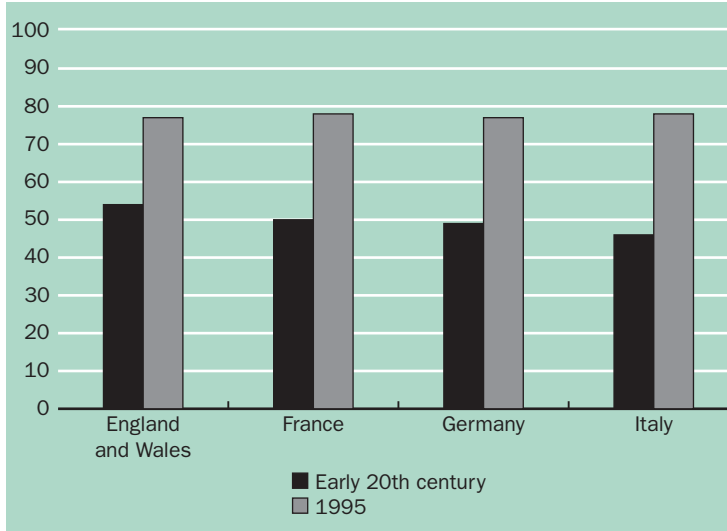
Although McKeown explicitly recognised that the world of health in developed countries was very different in the 1970s from the nineteenth century and thought that nutrition had become less of an issue, he considered that:

‘in advanced countries health is still determined mainly by personal behaviour and the environment. . . . . the influences which result from the individual’s behaviour (smoking, diet, exercise, etc.) are now relatively more important than those which depend on action by society. The contribution of personal medical measures remains tertiary in relation to the predominant behavioural and environmental influences’ (McKeown, 1979, p.9).

## Falling death rates

During the twentieth century, industrialised countries all experienced great improvements in life expectancy. Shortly before the First World War a life expectancy at birth of around 50 years was the norm in major European countries (Figure 1.1). Today it is in the high 70s. The experience illustrated in Figure 1.1 is remarkably uniform across countries and shows a dramatic increase over the century in the average lengths of people’s lives.

Figure 1.1 **Improved life expectancy at birth**



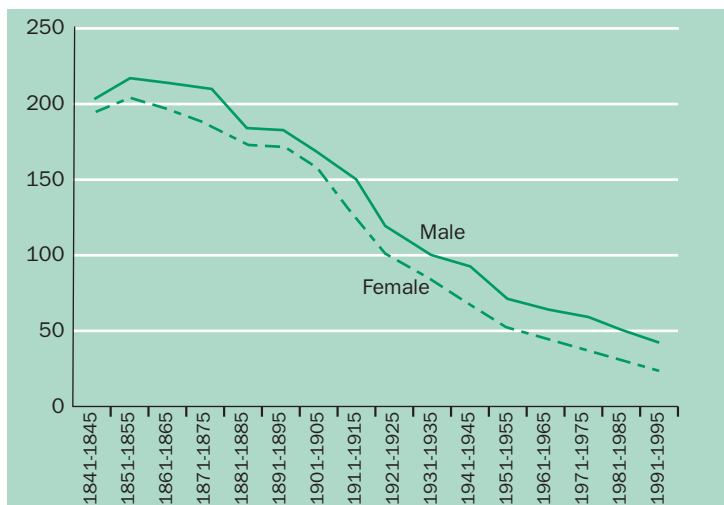
Notes: England and Wales data for the early 20th century are for 1910-11; France 1908-13; German Empire 1910-11; Italy 1910-12. All these data are taken from Vallin, 1991. The borders of Germany and Italy in 1995 differ from those in the early 20th century.

Sources: Vallin, 1991; OECD, 1999.

McKeown drew a graph to illustrate the great fall in male and female mortality rates in England and Wales from the mid-nineteenth century until 1971. To distinguish the effect of the declining risk of death at any age from the partially offsetting effect on crude mortality rates of the consequent ageing of the population, he showed how the overall population mortality rate would look if the population age structure were held constant at that of 1901. Figure 1.2 shows that, on the same 1901 population structure basis, the falling trend in mortality rates has continued in England and Wales since 1971.

Most of this improvement is due to there now being many fewer deaths in childhood than was the case in the nineteenth century.

Figure 1.2 Trends in death rates per 1,000 (1901 population basis), in England and Wales, 1841-1995

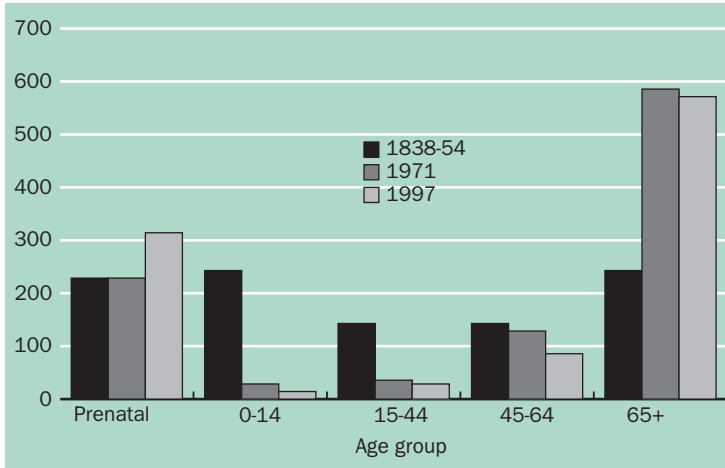


Note: Age adjustment to the death rates is by the direct standardisation method: the 1901 age structure of the population of England and Wales has been combined with the age-specific death rates in each period. This separates the decline in mortality from the changes in population structure from the mid-nineteenth century to the end of the twentieth.

Sources: Office for National Statistics, 1997; 1998b.

McKeown presented his data on this in the form of deaths per 1,000 conceptions. The first two stacks in each of the five age groups in Figure 1.3 reproduce McKeown's chart. The third stack in each case shows our calculations for 1997 based on mortality and population data from the Office for National Statistics (1997; 1998b). 'Prenatal' deaths combine estimates of miscarriages with terminations of pregnancy, both legal and illegal. The recent rise in the rate of prenatal deaths reflects increasing numbers of legal abortions. Figure 1.3 makes very clear the fall in childhood death rates, which has continued since 1971. Over the last century and a half there has also been a large fall in mortality rates for people of working age, particularly

**Figure 1.3 Deaths per 1,000 conceptions, by age group, England and Wales, 1838-1997**



Notes and sources: Data for 1838-54 and 1971 are taken from McKeown, 1979. Figures for 1997 are the authors' estimates based on data in Office for National Statistics, 1997.

for those aged under 45. As death is only deferred, never avoided, this inevitably means that mortality rates in the oldest of McKeown's age groups – 65 and over – have risen substantially.

The result is a very different population age structure at the end of the twentieth century compared to that at the beginning. In 1901, 32 per cent of the England and Wales population were aged under 15, compared with 19 per cent today. Conversely, fewer than 5 per cent of the population were aged 65 or over in 1901, compared with 16 per cent today. The proportion of the population in the middle age group is about the same now as it was 100 years ago.

With mortality among children thankfully now so low, any major improvements in life expectancy at the population level will in future have to come from reduced mortality among older people.

## Disease-specific mortality

We turn now to updating McKeown's data for the contributions of different groups of diseases to the long-term fall in mortality. In the limited space available, we have not attempted to review the weaknesses and heroic assumptions underlying McKeown's figures. Instead we take these data simply as broad indicators of trends and, in that spirit, extend them to the late 1990s, warts and all. The raw data for our calculations of disease-specific mortality rates in England and Wales are taken from the Office for National Statistics (1998c).

As can be seen from Table 1.1, over the period from 1901 to 1971 McKeown's figures show infectious diseases accounting for two thirds of the fall in the age standardised death rate in England and Wales. All other causes accounted for only one third of the fall. Among infectious diseases, the airborne infections are where the biggest reductions have been achieved. If McKeown had been writing today, with the benefit of data to 1997, his findings would still have been striking, although slightly less dramatic: 62 per cent of the fall in mortality from 1901 to 1997 was due to fewer deaths from infections.

Over the later years, 1971-1997, the story looks very different. The final column of Table 1.1 is only indicative as it is based on the 1901 population age structure and so overstates the impact of diseases that

**Table 1.1 Share of total fall in standardised mortality rate\* in each period attributable to each disease group, England and Wales**

|  | <b>1901-1971</b> | <b>1901-1997</b> | <i>1971-1997</i> |
|--|------------------|------------------|------------------|
| Attributable to micro-organisms:           |                  |                  |                  |
| 1. Airborne diseases                       | 39%              | 37%              | 20%              |
| 2. Water- and food-borne diseases          | 16%              | 15%              | 2%               |
| 3. Other infections                        | 12%              | 11%              | -1%              |
| <b>Total infections</b>                    | <b>67%</b>       | <b>62%</b>       | <b>21%</b>       |
| <b>Not attributable to micro-organisms</b> | <b>33%</b>       | <b>38%</b>       | <b>79%</b>       |

Note: \*Age-standardised mortality rate based on 1901 population structure.

**Table 1.2 Airborne infections: share of total fall in standardised\* mortality rates from all causes, England and Wales**

|                                    | <b>1901-1971</b> | <b>1901-1997</b> |
|------------------------------------|------------------|------------------|
| Tuberculosis (respiratory)         | 11%              | 10%              |
| Bronchitis, pneumonia, influenza   | 19%              | 18%              |
| Whooping cough                     | 3%               | 2%               |
| Measles                            | 2%               | 2%               |
| Scarlet fever and diphtheria       | 4%               | 3%               |
| Smallpox                           | 0%               | 0%               |
| Infections of ear, pharynx, larynx | 1%               | 1%               |
| <b>Total</b>                       | <b>39%</b>       | <b>37%</b>       |

Note: \*Age-standardised mortality rate based on 1901 population structure.

kill children and understates the impact of diseases of old age. Nevertheless it is clear that in the last quarter of the twentieth century the story has changed and most (at least 79 per cent) of the fall in overall mortality in that period has been due to lower death rates from non-infectious causes.

Turning now to McKeown's more detailed analysis, we have replicated as nearly as possible his calculations of mortality rates for a more disaggregated set of disease groupings for 1901 and 1971 and have added our own estimates for 1997 on the same basis.

Airborne infections accounted for the majority of the fall in mortality from infectious diseases between 1901 and 1971 where his data stop. Table 1.2 identifies the contributions of different types of airborne diseases. Over the 1901-1971 period, falling numbers of deaths from bronchitis, pneumonia and influenza represented half of the improvement due to airborne infections. Respiratory tuberculosis accounted for much of the remaining fall. Extending the analysis from 1971 to 1997 shows no change in the relative contributions of the different groups of airborne infections. Death rates from all types of airborne infections have continued to fall. One cautionary note is that although mortality from respiratory tuberculosis has now



**Table 1.3 Water- and food-borne infections: share of total fall in standardised\* mortality rates from all causes, England and Wales**

|                                | <b>1901-1971</b> | <b>1901-1997</b> |
|--------------------------------|------------------|------------------|
| Cholera, diarrhoea, dysentery  | 10%              | 10%              |
| Tuberculosis (non-respiratory) | 5%               | 4%               |
| Typhoid, typhus                | 1%               | 1%               |
| <b>Total</b>                   | <b>16%</b>       | <b>15%</b>       |

Note: \*Age-standardised mortality rate based on 1901 population structure.

declined to a very low level in England and Wales, there is some sign of a small upturn there. The same upturn is being seen elsewhere, especially in US cities. Deaths from airborne infections other than respiratory tuberculosis, bronchitis, pneumonia and influenza were already relatively few by 1971 and have remained so.

Tables 1.3 and 1.4 demonstrate that declines in mortality from water- and food-borne and other infections have contributed nearly the same share of the total fall in mortality rates over the period 1901-1997 as they did for the somewhat shorter, 1901-1971, period recorded by McKeown.

**Table 1.4 Other infections: share of total fall in standardised\* mortality rates from all causes, England and Wales**

|                           | <b>1901-1971</b> | <b>1901-1997</b> |
|---------------------------|------------------|------------------|
| Convulsions, teething     | 6%               | 5%               |
| Syphilis                  | 1%               | 1%               |
| Appendicitis, peritonitis | 1%               | 1%               |
| Puerperal fever           | 1%               | 0%               |
| Other infections          | 4%               | 3%               |
| <b>Total</b>              | <b>12%</b>       | <b>11%</b>       |

Note: \*Age-standardised mortality rate based on 1901 population structure.

Overall, his assessment of the role of the infectious diseases would almost certainly be the same today as it was when he wrote in the 1970s: that largely overcoming them has been the proximate cause of the major reduction of mortality achieved in the twentieth century.

Table 1.5 shows the impact of non-infectious causes of death on the total decline in mortality that has been achieved this century. It covers a wide range of afflictions. For most of the different sub-headings of non-infections, definitional problems make it hard to place much confidence in small apparent changes to their contributions to the overall fall in mortality in the two periods (1901-1971 and 1901-1997 respectively). However, there is one very striking feature of these data: namely the significant fall in mortality from cardiovascular disease since McKeown was

**Table 1.5 Non-infections: share of total fall in standardised\* mortality rates from all causes, England and Wales**

|  | <b>1901-1971</b> | <b>1901-1997</b> | <b>1971-1997</b> |
|--|------------------|------------------|------------------|
| Congenital defects                                 | 0%               | 1%               | 7%               |
| Prematurity, immaturity, other diseases of infancy | 9%               | 9%               | 7%               |
| Cerebrovascular disease                            | 2%               | 3%               | 20%              |
| <b>Cardiovascular disease</b>                      | <b>-1%</b>       | <b>4%</b>        | <b>50%</b>       |
| Cancer   | -3%              | -1%              | 6%               |
| Other diseases of digestive system                 | 4%               | 3%               | -2%              |
| Other diseases of nervous system                   | 2%               | 2%               | -1%              |
| Nephritis and other diseases of urinary system     | 3%               | 3%               | 2%               |
| Pregnancy and childbirth (excluding sepsis)        | 1%               | 1%               | 0%               |
| Violence   | 3%               | 3%               | 9%               |
| Other  | 13%              | 10%              | -19%             |
| <b>Total</b>                                       | <b>33%</b>       | <b>38%</b>       | <b>79%</b>       |

Note: \*Age-standardised mortality rate based on 1901 population structure.

writing. In 1971 the age standardised mortality rate for cardiovascular diseases was little different from that in 1901; indeed it was even a little higher. But over the last quarter century, death rates from this cause have fallen considerably, as the final column of Table 1.5 highlights.

The big killers today are cardiovascular and cerebrovascular diseases and cancers, throughout the industrialised world, although the measured rates can vary quite widely between individual countries, especially for circulatory diseases. Table 1.6 illustrates this point. The numbers here are for whole ICD chapters. We recognise, however, even at this highly aggregated level, the minefield that exists in attempting international comparisons of this kind. For example, France has an apparently exceptionally low (relatively speaking) death rate from circulatory diseases, but other WHO statistics make clear that France has, conversely, an exceptionally high measured death rate in the catch-all 'other' category. This may have something to do with different practices in data recording. So we will just leave our observations at the level of the unexceptionable: circulatory diseases and cancers are today major killers in all developed countries (as well as elsewhere).

**Table 1.6 Standardised\* death rates per 100,000 in major European countries, 1995**

|                   | <b>Circulatory diseases<br/>(ICD Chapter 7)</b> | <b>Neoplasms<br/>(ICD Chapter 2)</b> |
|-------------------|---|--------------------------------------|
| England and Wales | 310   | 207                                  |
| France            | 183   | 198                                  |
| Germany           | 344   | 202                                  |
| Italy**           | 285   | 205                                  |

Notes: \*Age standardised using the European Standard Population (taken from WHO, 1991).

\*\*Italian data are for 1993.

Sources: WHO, 1991; 1999.

## Morbidity

So far, the discussion has been entirely about death rates. McKeown's reason for concentrating on mortality was the lack of long time series of any other kind of data about population health. But a great deal of ill health and suffering is caused by non-fatal conditions, as McKeown recognised. Looking at the prevalence of illness, as well as the incidence of death, may imply a rather different emphasis when considering how best to improve population health in future.

For example, attacking non-fatal illness implies focusing a lot more attention on mental health than would a narrow concentration on mortality alone. World Health Organization (WHO) estimates of disability adjusted life years (DALYs) lost in developed countries in 1990 as a result of various categories of disease, attribute one quarter of the total disease burden to mental (neuropsychiatric) illnesses (Murray and Lopez, 1996). See Table 1.7. The burdens of cancers and circulatory diseases are also large when measured in DALY terms but are a lot smaller than for mental illness. This is in sharp contrast to mortality alone, where cancers and circulatory diseases dominate.

As mortality rates fall and life expectancy increases, the importance of improving the quality of people's lives, and not just their lengths, grows. But it is not just the decline of mortality which is making

**Table 1.7 Disease burden in established market economies, 1990: per cent of total DALYs\* lost**

|   |             |
|---|-------------|
| Communicable, material perinatal, nutritional | 7.1%        |
| Neoplasms                                     | 15.0%       |
| Neuropsychiatric                              | 25.0%       |
| Cardiovascular, including cerebrovascular     | 18.6%       |
| Other non-communicable diseases               | 22.4%       |
| Injuries                                      | 11.9%       |
| <b>Total</b>                                  | <b>100%</b> |

Note: \*Disability adjusted life years.

Source: Murray and Lopez, 1996.

Table 1.8 Per cent of GB population reporting chronic ill health

|      | Age group |       |     |
|------|-----------|-------|-----|
|      | <16       | 16-64 | 65+ |
| 1972 | 6%        | 20%   | 53% |
| 1980 | 12%       | 30%   | 60% |
| 1990 | 17%       | 33%   | 63% |
| 1996 | 16%       | 34%   | 62% |

Source: Office for National Statistics, 1998a.

morbidity relatively more important as a focus of health policy. The prevalence of illness also seems to be growing. Or at least people's perception that they are ill has been growing. Figures from the Great Britain General Household Survey, shown in Table 1.8, display two main features. First, reported chronic ill health increased considerably in all age groups between the 1970s, when McKeown was writing, and 1990, but during the 1990s it appears to have plateaued. Second, the number of children reporting chronic ill health has increased particularly starkly, from one in 16 to one in every six.

A similarly depressing picture was reported by Bebbington and Darton (1996). They found, from an analysis of mortality data and General Household Survey data, that although life expectancy in England and Wales increased strikingly for both men and women between 1976 and 1994, nearly all of these extra years were, on average, marked by the presence of limiting long-term illness. Total male life expectancy at birth increased in this period by 4.2 years to 74.2 and for women it rose 3.5 years to 79.6. However, the increase in healthy life expectancy was small, and in the case of women not statistically significant. Expectancy of years free from limiting long-term illness increased by just 0.8 years for men and 0.1 years for women. However, further analysis from the same source shows that there was no increase in average years spent suffering from major disabilities, as measured by inability to perform activities of daily living. The implication therefore appears to be that the additional reported chronic illness is at least not greatly disabling.

## Government health policy

So, finally, how are the issues raised by updating McKeown's analysis being dealt with in current British government health policy? We have seen a minor epidemic of public health policy statements in the form of government health reports and White Papers between late 1998 and mid-1999:

- Report of the Independent Inquiry into Inequalities in Health, generally referred to as 'The Acheson Report', November 1998;
- Smoking Kills – A White Paper on Tobacco, December 1998;
- Saving Lives: Our Healthier Nation, July 1999.

Despite this recent flurry of activity, there remain many similarities between the Labour government's public health policy at the end of 1999 and that of its Conservative predecessor as stated in 1992 in *The Health of the Nation*. The focus of all UK government public health policy in the 1990s has been on cancers, coronary heart disease and stroke. Infections get little mention. The figures we presented earlier show that this is justified, despite the historical importance of overcoming infections. A rider to this, however, is that *The Health of the Nation* had a chapter dedicated to HIV and AIDS, and referred to them as 'perhaps the greatest new threat to public health this century'. But this chapter has been dropped from 1999's *Saving Lives*. HIV/AIDS and other communicable diseases are swept up with a range of other health issues in a catch-all, miscellaneous chapter. A sign perhaps that in official circles HIV and AIDS are no longer perceived as a major threat to public health in Britain?

Strong official emphasis is being given to tackling morbidity as well as mortality. Taking the government's approach to mental health as an indicator of the relative importance accorded to morbidity rather than mortality:

- mental health warrants a chapter to itself in *Saving Lives*, just as it did in *The Health of the Nation*;
- mental health is the subject of the first NHS National Service Framework; published by the Department of Health in September

1999. This defines national standards and service models for the provision of mental health care throughout the NHS;

- however, the dramatic titles of the current government's two public health White Papers – *Smoking Kills and Saving Lives* – seem to imply that mortality is still the main focus: quantity of life before quality of life;
- but against that, at a practical level, many of the numerous activities and initiatives listed in the White Papers are aimed at reducing morbidity, not just mortality.

Returning to the overall question that prompted the OHE conference and this book, namely where to invest to get the biggest health gain in return, what does the government's answer appear to be?

Both *Saving Lives* and the earlier *The Health of the Nation* have reflected McKeown's great emphasis on individuals' behaviour as the most important driver of population health in the UK today. McKeown listed smoking, diet and exercise as the most important behavioural influences on health. Recent government policy statements do so too but focus particularly strongly, and repeatedly, on smoking far more than on diet and exercise.

In *Saving Lives*, government responsibility for improving the population's health is described largely as twofold. First, the government should provide means by which individuals and communities can make themselves healthier. The means specified are information, education and encouragement, rather than more doctors or nurses, let alone cash. The government's second responsibility is to improve the socio-economic environment. This is to be achieved through social, economic and environmental policies rather than health care provision directly. Social security, education, transport, employment and so on are all now explicitly involved, not just the government ministers with 'health' in their titles.

The new 'big idea' for policies to improve population health is to tackle it – along with many other social problems – by trying to reduce social inequalities, or 'social exclusion' in the current phrase. This theme recurs throughout *Saving Lives* (1999) but you had to wait

to page 121 out of 126 to see explicit mention of it in *The Health of the Nation* (1992).

It is notable how little emphasis is given to the role of health care interventions in either the Conservatives' or the new Labour White Papers. No mention is made of the desirability of encouraging development and use of new medicines and other health care technologies as a route to long-term health improvement. Nor is there any discussion of increasing the quantity made available of the existing types of health care provision. The White Papers say nothing about training more doctors or any other health care professionals, for example.

The White Papers are also silent about priorities: about which policies should be enacted soonest and accorded the greatest share of resources. This lack explains the purpose of the OHE conference and this book of the proceedings. The aim of both has been to bring together in one place discussion of a wide range of major factors that have determined population health in the past century, so as to provide a clear, if high-level, basis for considering health policy priorities for the 21st century.

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## Chapter 2

# The impact of nutrition on health

MICHAEL RAYNER

### Introduction

McKeown argued that ‘an improvement in nutrition was the critical advance which led to the modern reduction of mortality and growth of population’ (McKeown, 1976). He had four reasons for this. First, he argued that ‘something remarkable has happened since the seventeenth century’ to explain the improvement in health and identified this as the agricultural and industrial developments of the past three centuries. Secondly, McKeown showed the weaknesses of alternative explanations – of improvements in medical practice in particular. Thirdly, he put forward evidence to show that there was a large increase in food supplies from about the end of the seventeenth century. Fourthly, he argued that ‘in the conditions that existed before the eighteenth century an improvement in nutrition was a necessary condition for a substantial and sustained decline of mortality and growth of population’. In this final argument McKeown was taking a Malthusian/Darwinian view that population numbers are limited by a high level of mortality attributable directly or indirectly to lack of food.

As many others have pointed out, the weight of presumption in favour of nutrition as the primary causal factor in the decline in mortality emerged in McKeown’s thesis primarily by default. That is as a result of the sceptical devaluation of other factors – and in particular medical interventions – rather than from any convincing positive evidence in its support (Szreter, 1988). Szreter and other ‘revisionists’ have pointed out that McKeown probably underestimated the role of improvements in the supply of clean water brought about by the early Victorian public health movement and in particular by local government departments charged with various duties aimed at preventing known pathogens entering the water supply. Mackenbach has pointed out that McKeown may have underestimated the role of antibiotics and other medical interventions in the decline in mortality (Mackenbach, 1996, and see Chapter 4 below).

Nevertheless the revisionists do not completely dismiss the importance of increasing food quality. Szreter notes, for example, the possible role of the increasingly close regulation of the quality of the urban food supply ‘which resulted directly from the attention which Medical Officers in the 1860s had begun to pay to adulterated and defective foodstuffs – particularly meat and milk – as a source of disease’ (Szreter, 1998). He points out that the Adulteration of Food Acts in the 1870s led to the appointment of professional inspectors and public analysts by most local authorities in the 1880s. So the question is not whether improvements in food and nutrition were important, but how important they were.

When McKeown and the revisionists have talked about the impact of nutrition on health in the late nineteenth and early twentieth centuries, they have assumed that good nutrition means basically a sufficient supply of calories and possibly other essential nutrients such as protein, essential fats, vitamins and minerals but they have not been particularly specific. McKeown argued that in the 1970s nutrition was internationally ‘probably still the most important determinant of health’ (McKeown, 1976) but he accepted that ‘The significance of food in developed countries is of course somewhat different [from that in developing countries]. There it can be assumed that most people have enough to eat, and the more usual problem is consumption of excessive or ill-balanced diets’ (McKeown, 1976).

The rest of this chapter explores the effects of excessive or ill-balanced diets on health in developed countries in the late twentieth century and their possible effects in the early part of the twenty-first century. It looks at different models of the food system and speculates on the likely direction of food policy in developed countries and its impact on diets and health. It shows that the debate about the importance of nutrition to health in the late twentieth century resembles that about the late nineteenth early twentieth centuries. Nutrition is viewed as possibly important but opinions differ about how important it is.

## The burden of ill health in the late twentieth and early twenty-first centuries

As Sussex has pointed out in the preceding chapter, it is no longer enough to measure the burden of disease solely in terms of mortality. A consensus is emerging that health should be measured in terms of morbidity as well. The WHO's Global Burden of Disease Study measures the disease burden in terms of years of life lost, years of life lost in disability, and in terms of a measure which combines both of these elements, namely disability adjusted life years (DALYs) (Murray and Lopez, 1996).

For the economically developed nations, which it terms 'established market economies (EMEs)', the Global Burden of Disease Study has shown that nearly 60 per cent of years of life lost are due to cardiovascular disease (CVD), cancer and diabetes (see Table 2.1). These are

Table 2.1 Years of life lost in early death and in disability, and DALYs lost, by cause, established market economies, 1990

|                            | % of total<br>years of<br>life lost | % of total<br>years of<br>life lost<br>in disability | % of total<br>DALYs<br>lost |
|----------------------------|-------------------------------------|--|-----------------------------|
| Infectious diseases        | 8.8                                 | 5.5  | 7.1                         |
| Cardiovascular disease     | 30.8                                | 6.2  | 18.6                        |
| Coronary heart disease     | 16.3                                | 1.6  | 9.0                         |
| Stroke                     | 6.9                                 | 3.2  | 5.0                         |
| Diabetes                   | 1.6                                 | 3.2  | 2.4                         |
| Cancer                     | 26.1                                | 3.8  | 15.0                        |
| Neuropsychiatric disorders | 3.1                                 | 47.2   | 25.0                        |
| Injuries                   | 15.9                                | 7.8  | 11.9                        |
| Other                      | 13.7                                | 26.3   | 20.0                        |
| <b>Total</b>               | <b>100.0</b>                        | <b>100.0</b>   | <b>100.0</b>                |

Source: Murray and Lopez, 1996.

all diseases that are associated with excessive or ill-balanced diets. However, looking at morbidity rather than mortality, Table 2.1 shows that only 13 per cent of years of life lost in disability are due to CVD, cancer and diabetes. When you combine morbidity and mortality in a single DALY measure, however, you find that CVD, cancer and diabetes in total cause about 36 per cent of DALYs lost (Table 2.1).

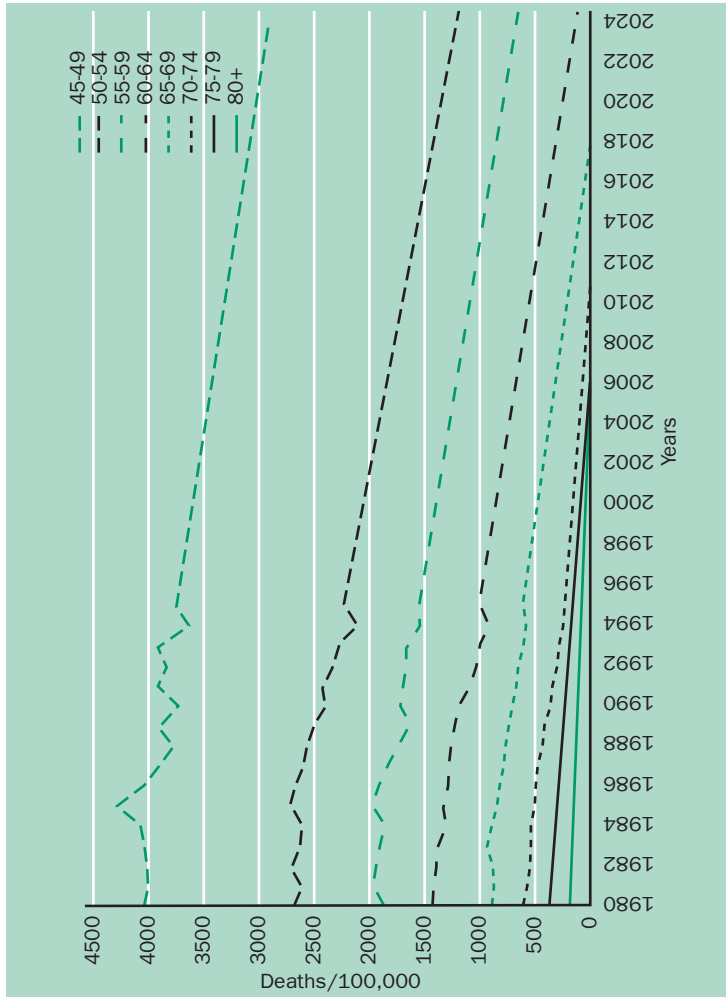
Focusing on CVD, mortality rates are coming down in most developed European countries but going up in many of the formerly centrally planned economies of Europe. Generally, the pattern of disease in different industrialised countries is converging: so that coronary heart disease mortality rates are coming down faster, from a higher starting point, in northern and western Europe than in southern Europe. The same is true also of stroke.

Although life expectancy has increased in many developed countries, there is a degree of controversy about whether the extra years of life gained have been 'healthy' or not. For example, between 1970 and 1990 life expectancy at birth rose from 69 to 73 for men and from 75 to 79 for women in England and Wales, but Dunnell argues that 'there has been no comparable increase in number of years of healthy life' (Dunnell, 1997).

One way of looking at trends in the burden of disease is to look at incidence rates rather than death rates. The MONICA (monitoring trends and determinants in CVD) Project has examined the incidence of CVD in 37 different populations in 21 countries. It shows that the incidence of coronary events is falling rapidly in most of the MONICA Project populations in which mortality from coronary heart disease is falling and rising where mortality is rising (Tunstall-Pedoe et al., 1999).

Of course, if the disease is becoming less common in younger age groups it still might be becoming more common in older age groups. We do not, I think, have good data on trends in age-specific incidence rates of coronary heart disease (CHD) but Figures 2.1 and 2.2 show that mortality rates are declining across all age groups, even the oldest, and are predicted to carry on doing so. That is true for both men and women.

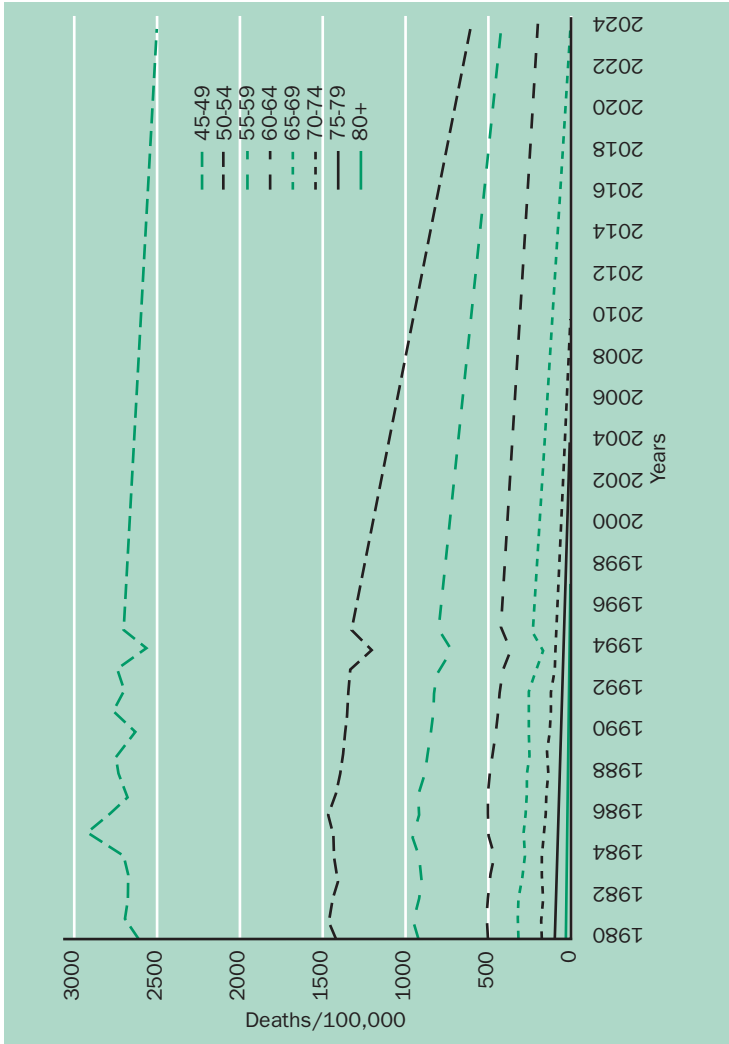
Figure 2.1 UK age-specific death rates from CHD in men (1980-1994) and predicted rates based on current trends



Note: CHD=coronary heart disease.

Source: WHO, 1999.

Figure 2.2 UK age-specific death rates from CHD in women (1980-1994) and predicted rates based on current trends



Source: WHO, 1999.

**Table 2.2 DALYs lost by cause, in established market economies, 1990 and 2020**

|                            | <b>1990</b><br><b>(millions)</b> | <b>2020</b><br><b>(millions)</b> |
|----------------------------|----------------------------------|----------------------------------|
| Infectious diseases        | 7.0                              | 5.0                              |
| Cardiovascular disease     | 18.3                             | 18.8                             |
| Coronary heart disease     | 8.9                              | 9.5                              |
| Stroke                     | 5.0                              | 5.0                              |
| Diabetes                   | 2.4                              | 2.0                              |
| Cancer                     | 14.8                             | 16.8                             |
| Neuropsychiatric disorders | 24.7                             | 24.6                             |
| Injuries                   | 11.8                             | 9.8                              |
| Other                      | 19.8                             | 20.0                             |
| <b>Total</b>               | <b>98.8</b>                      | <b>97.0</b>                      |

Source: Murray and Lopez, 1996.

However, mortality from a disease might be completely wiped out whilst morbidity from that disease continued to increase. The Global Burden of Disease Study predicts that DALYs lost from CVD will rise by 2020, but only slightly and against a backdrop of a slight decline in total DALYs lost from all causes in EMEs (see Table 2.2).

### **What proportion of this burden is due to poor nutrition?**

The Global Burden of Disease Study shows that in developed countries 36 per cent of DALYs lost are due to nutrition-related disease, i.e. CVD, cancer and diabetes. Taking poor nutrition to mean excessive or ill-balanced diets, there are now clearly demonstrable relationships between poor nutrition and chronic disease. For example, there is now well established evidence that high saturated fat consumption increases risk of coronary heart disease; that high fruit and vegetable consumption reduces risk of certain forms of cancer – particularly colorectal, gastric and oesophageal cancer; and that overweight and obesity increases risk of diabetes. For other diet-health relationships there is substantial evidence but still controversy: for example



whether a high total fat consumption, irrespective of the type of fat, increases risk of coronary heart disease; and whether high total fat or saturated fat consumption increases risk of cancer.

Excessive or ill-balanced nutrition is more difficult to define than, say, cigarette consumption or raised blood pressure. It is therefore difficult to calculate the amount of ill-health attributable to poor nutrition. Epidemiologists have generally shied away from doing so, but the World Cancer Research Fund (1997) estimates that 30-40 per cent of cancer deaths are due to poor diets, and the European Heart Network (1998) estimates that around 30 per cent of deaths from CVD are attributable to the same cause. These seem overestimates when compared with Peto et al.'s estimates of the number of deaths in the EU that are attributable to smoking, namely 24 per cent of all cancer deaths and 10 per cent of deaths from CVD (Peto et al., 1994, and British Heart Foundation, 2000).

Perhaps the best evidence of the burden of disease due to poor diets is that of the National Institute of Public Health in Stockholm (1997). They suggest that 5 per cent of DALYs lost in the EU are due to diets high in saturated fat and low in fruit and vegetables, and that a further 4 per cent are due to overweight, giving a total of 9 per cent of DALYs lost due to poor diets. They estimate that smoking accounts for the same proportion, 9 per cent, of DALYs lost in the EU.

In most medical circles, smoking has been given much more weight than nutrition as a cause of illness. Nutrition is more complex and includes many different factors, but probably the burden of disease attributable to it is equivalent to that attributable to smoking.

### **Trends in nutrition and food consumption patterns**

Assuming that we have an excessive and ill-balanced diet in developed countries, or that at least some of us do, are things getting better or worse, and how do these trends impact upon health?

In the UK we have relatively good data on diets, since 1942, from the National Food Survey. This survey does have its drawbacks, not least that it measures consumption by households rather than by individ-

uals. However it shows, for example, that total fat and saturated fat consumption are now falling: total fat by about 5 per cent in the last 20 years and saturated fat by about 22 per cent (British Heart Foundation, 1999).

A recent meta-analysis by Stephen and Sieber (1994) of studies of fat intake from 1900 to 1985 indicated that among British adults total fat as percentage of dietary energy increased from 25 per cent during the early part of the century to 33 per cent by the late 1930s. It continued to increase after the war to peak at 40 per cent of dietary energy by the late 1970s before starting to fall.

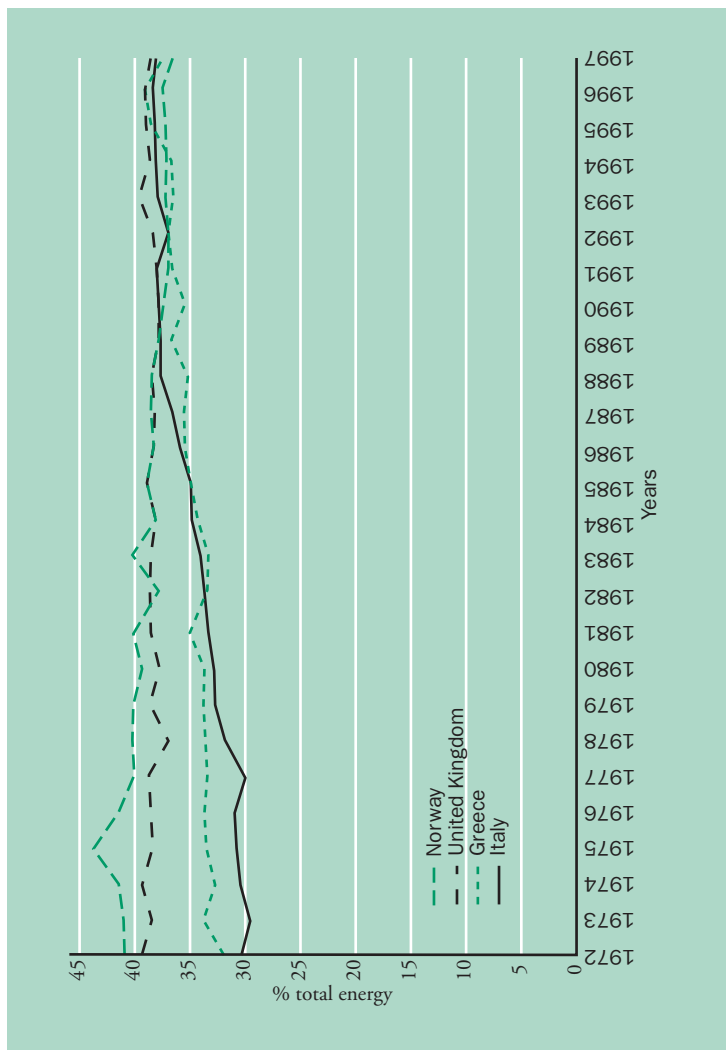
The National Food Survey also suggests that in the UK: fruit and vegetable intake is increasing; consumption of whole milk and butter is decreasing ; and consumption of low-fat milks and soft margarine is increasing. In other words, the balance of the average UK diet is improving.

On the other hand, levels of overweight and obesity in the UK are increasing. The percentage of men who are obese doubled in the last 10 years and the percentage of women who are obese increased by about 42 per cent. Similar, if less rapid, upward trends in obesity are evident throughout Europe.

Similar trends are observed in other developed countries but not all. Figure 2.3 presents trends in fat consumption in some European countries, taken from Food and Agriculture Organization (FAO) data. This figure shows that the percentage of total energy that is obtained from fat has fallen slightly in northern and western European countries but risen slightly in southern Europe. Note that this parallels the geographical pattern of trends in CVD mortality.

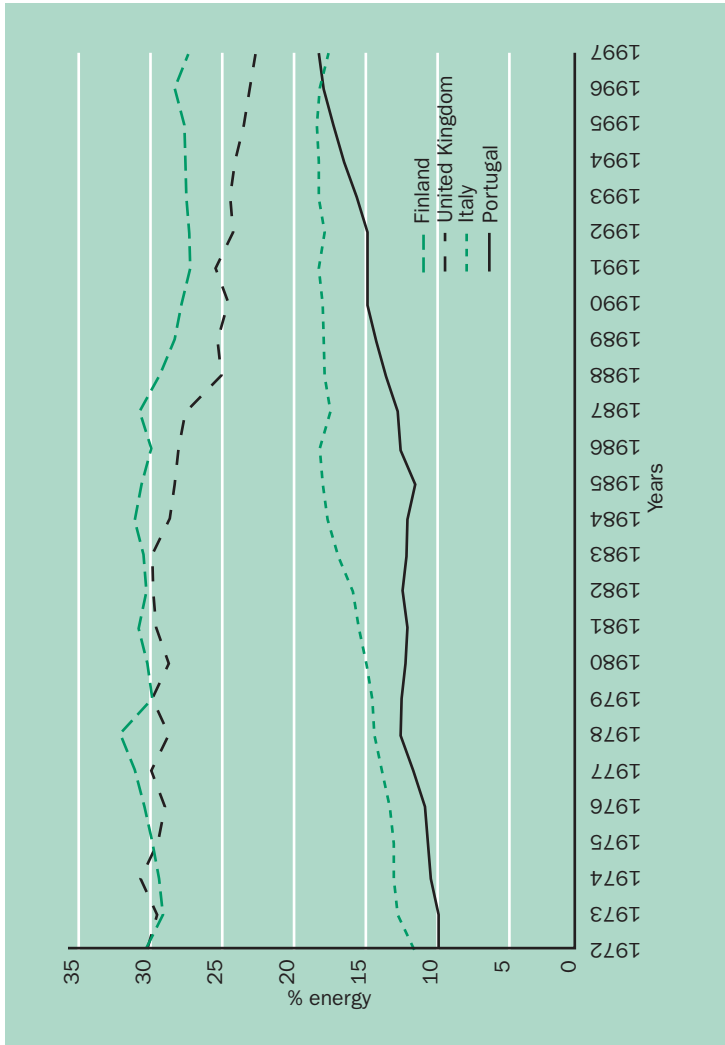
Figure 2.4 shows an even more dramatic picture in the case of total energy from fat from animal products – the best measure of saturated fat intake that is available from the FAO database. This shows that in northern and western European countries like Finland and the UK the total energy intake from animal fat is falling, whereas in southern European countries like Italy and Portugal it is rising. In each case the trends are steeper for animal fat than for total fat.

Figure 2.3 Percentage of total energy from fat, 1972-1997, selected countries



Source: British Heart Foundation, 2000.

Figure 2.4 Percentage of total energy from fat from animal products, 1972-1997, selected countries



Source: FAO, 1999.

Figures 2.3 and 2.4 show the tendency for the dietary patterns of developed (and indeed of all) countries to converge. This convergence is evident for other foods as well. Figure 2.5 shows the percentage of total energy from cereals from 1972 to 1997 in selected countries. In northern European countries cereal consumption is increasing slightly – in Ireland and Norway, for example – but it is declining in southern Europe.

The convergence is not so obvious with some other foods, such as fruit and vegetables. Fruit and vegetable consumption is high in southern European countries, such as Italy and Greece, and low in northern European countries, such as the UK and Norway. Although fruit and vegetable consumption is increasing slightly in northern Europe, the dietary patterns in this case are not coming together to the same extent as with other foods.

### **Are trends in chronic disease attributable to the trends in food consumption patterns?**

As we have seen, mortality from cardiovascular disease is generally declining. This may be associated with the improvements in diets. The tendency for international patterns in CVD to converge may also be associated with the tendency for diets to converge. The incidence of diabetes is increasing in most developed countries and this could be associated with the increase in obesity.

Cancer mortality and incidence remain approximately stable, although certain forms of diet-related cancers are declining, possibly as a result of changes in diets. There has, for example, been a fall in stomach cancer in many developed countries, possibly associated with increasing consumption of fruit and vegetables. On the other hand, there has been an increase in colorectal cancer, possibly associated with decreasing consumption of dietary fibre and other complex carbohydrates (World Cancer Research Fund, 1997).

The question remains: what proportion of the trends in mortality and morbidity from chronic disease are attributable to the trends in food consumption patterns? The debate relates to the question of whether the trends in mortality are attributable to changes in disease incidence

Figure 2.5 Percentage of total energy from cereals, 1972-1997, selected countries



Source: British Heart Foundation, 2000.

or to some other factor such as changes in case fatality and, if the latter, whether this is due to improvements in treatment or in the severity of the disease. The MONICA project has concluded that the 'contribution to changing coronary heart disease mortality varied, but in populations in which mortality decreased, coronary event rates contributed two thirds and case fatality one third' (Tunstall-Pedoe et al., 1999).

Whatever the extent of morbidity and mortality that is currently the result of nutritional factors, the key question for policy is how effectively can population health be improved by deliberate measures to influence nutrition? Unfortunately, research into the effectiveness of population-based interventions to improve nutrition and hence health is particularly weak. This is partly because research has been dominated by a medical model which implies that randomised control trials are required to reveal the evidence of effectiveness reliably. The lack of evidence is also a question of funding. There has been very little funding of research into the evidence of the effectiveness of population-based interventions to affect nutrition.

### **'Globalisation' of food consumption patterns**

What lies behind the international convergence in food consumption patterns? In my view, the major discernible trend affecting world food consumption patterns is the growth of trans-national corporations and the decreasing number of companies involved in the food trade. Trans-national corporations now account for 70 per cent of total world trade. One cereal company alone, Cargill, controls 60 per cent of the world cereal trade; three companies control 80 per cent of the banana market; three control 83 per cent of the cocoa market; and so on (Lang and Hines, 1993).

Another sign of the globalisation of the world food trade is the move from national to international control of food legislation. Food legislation in Europe used to be set nationally but since 1992 it has been set at the EU level for member states. The Uruguay Round of the General Agreement on Tariffs and Trade (GATT) meant that the Codex Alimentarius Commission – a joint WHO/FAO international food

standards-setting body – has assumed much greater importance in shaping food legislation.

## Two models of the future

Trends in consumption, trends in disease and trends in the way the food system is organised are all tending to globalise (Lang and Heaseman, 2000). There are two, competing, models of the food system with different goals: the neo-liberal model and the ecological model. Adherents of the neo-liberal model consider that price and availability should mediate food supply and demand. Adherents of the ecological model consider that food production should be regulated to ensure optimum human and environmental health.

Neo-liberals and ecologists also propose different solutions to the problem of nutrition-related diseases in developed countries. Neo-liberals tend to propose a ‘therapeutic approach’ with increasing dependence on, firstly, functional foods. These are the new types of foods that are on the borderline between drugs and foods. It is speculated that there will be a huge growth in the market of these foods over the next few years. Functional foods have added nutrients and pharmacological-like effects to correct supposed nutrition deficiencies in the regular food supply. Secondly, the ‘therapeutic approach’ concentrates on diets tailored to individuals’ genetic profiles. One future scenario is therefore that diets will become more and more tailored to people’s individual genes. You go to your doctor, get your genetic profile and he or she will tell you what functional foods you should eat.

Ecologists, on the other hand, favour a ‘public health approach’, with more sustainable food production methods and food consumed in closer proximity to its production.

The neo-liberal model has recently been dominant in food policy-making circles. It is likely to continue to predominate until the damaging effects of many food production methods on both human and environmental health are more fully realised.



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## Chapter 3

# Changing individual behaviour: smoking and substance abuse

CHRISTINE GODFREY

### Introduction

I want to talk about four areas. First some data about smoking, drink and drugs, and how they affect life and death in the twentieth and twenty-first centuries. Secondly, what role should governments take in tackling the health problems that arise from them. Thirdly, I will consider what evidence exists for cost-effective interventions that can change some of these individual behaviours. Finally, I will look at the role that the health sector could play.

### Consumption

Drugs of all sorts are popular and make up quite a large part of people's consumption expenditure. There was £29.7 billion spent on alcohol in the UK in 1998, which is 5.7 per cent of all consumers' expenditure (Penny, 1999), and with the revision of laws governing licensing and shop trading hours, we can buy alcohol almost every minute of the day and night.

There remains a high level of spending on cigarettes, even though only 26 per cent of the Great Britain population now smoke (Bridgwood, 2000). £11 billion was spent on cigarettes in 1998 (Penny, 1999).

Obviously our estimates of the illicit drug market are much more speculative, but the Office of National Statistics has produced estimates that in 1997 between £3.9 and £8.5 billion was spent on illicit drugs in the UK (referred to in DrugScope, 2000).

### Smoking and disease

In the late twentieth century smoking is the largest preventable cause of premature deaths in all the industrialised countries. We know that

about one in two smokers die prematurely and half of those smokers die in middle age. That is where the health loss is coming from. Peto et al. (1992) have suggested that about 20 per cent of all deaths in developed countries are attributable to tobacco. The proportion is slightly higher in countries such as the UK, where we have been smoking longer. The deaths do not occur until quite a long time after smoking takes off in a country. For policy-makers this long time lag is quite a difficult concept to get across. The epidemic started in the UK and the US and so we have seen high death rates here. In many other countries, particularly in Europe and particularly among women, the lung cancer rates are still rising.

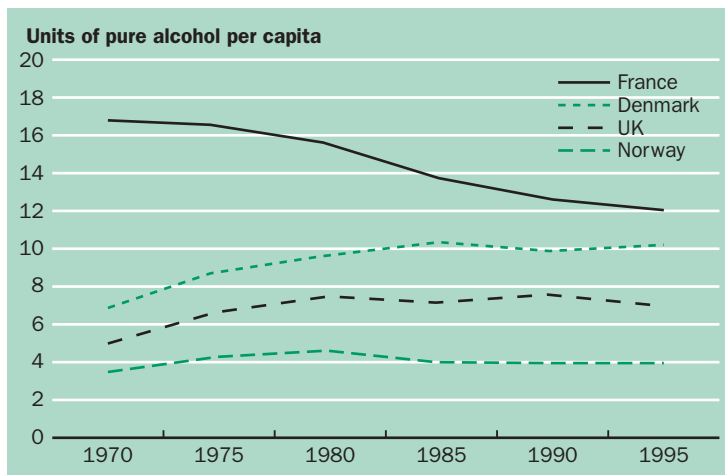
My colleague Paul Kind's data on quality of life in the UK show that the quality of life of smokers is diminished relative to that of non-smokers in all age groups (Kind, 1999, personal communication). We know that smokers are also heavier consumers of health care resources at all ages. The other thing that is often forgotten, particularly by smokers, is that stopping smoking has huge health gains and these continue into old age as well as middle age.

### Alcohol and disease

Alcohol is one of the oldest and favourite drugs in the industrialised world, and because it has been around for so long there is quite a lot of evidence on its health impact and the impacts of policies to tackle it (Raistrick et al., 1999). We have quite a lot of experience of epidemics of drinking in other centuries. At the end of the twentieth century there is an epidemic of alcohol consumption in Russia and some other eastern European countries, and we see the major impact that it can have on population health. We have a rather strange circumstance in the UK: alcohol consumption in 1900, in 1950, and what it looks like being in 1999/2000, are all at the same level, although there has been a huge variation in consumption levels between those dates.

Figure 3.1 shows the convergence in alcohol consumption per head that has been occurring across Europe since the 1970. Per capita consumption in France has been falling from its very high level and consumption in northern European countries has either risen or

Figure 3.1 Per capita alcohol consumption



Source: BLRA, 1999.

remained stable. Despite the convergence, alcohol consumption remains much higher in France than in northern Europe.

In terms of the global burden of disease, alcohol is a major problem world-wide. Alcohol diseases affect 5-10 per cent of the world's population each year and according to the World Bank accounted for 3 per cent of the global burden of disease in 1990 (World Bank, 1993). There is a range of problems with alcohol, varying from the acute to the chronic. For example, in the UK we have a large number of deaths amongst young people resulting from drunkenness and from alcohol poisoning. There is also chronic alcohol dependence. There is a smaller number of deaths than tobacco currently, but the accidents and poisonings resulting from alcohol occur at the younger age. Looking at life years lost, tobacco is still a slightly larger cause but alcohol is starting to catch up.

We know that quite a large proportion of people drink heavily, i.e. 27 per cent of men drink more than 21 units of alcohol per week and 14 per cent of women drink more than 14 units. The annual prevalence

rate of alcohol dependence detected by the UK mental health survey in 1994 is 75 per 1,000 for men in ages 16 to 24, and 21 per 1,000 for women (Meltzer et al., 1995). The same male-female difference is also present for dependence on illicit drugs.

There has been a rapid rise in the number of reported deaths in the UK directly attributed to alcohol-related causes, even though alcohol consumption has been relatively flat since 1980. We have also seen, in disability benefit surveys, the number of long-term alcohol-related sicknesses and days absent from work rise enormously in the 1980s and 1990s. See Goodwin, 1992, and Godfrey and Hardman, 1994. From a survey of doctors' certificates these authors calculated that the number of days lost for men had risen from 931,200 in 1983/84 to 2,581,900 in 1990/91. Estimating total alcohol related sicknesses from the same source yielded over 13 million days of absence in 1990/91.

We have to remember, however, that not all alcohol consumption is necessarily bad. This is an aspect that makes research into alcohol more complex than for smoking.

### **Illicit drugs and disease**

In policy terms, illicit drugs have until recently received far more attention than smoking, and certainly more than alcohol, in the UK and in Europe more generally. Only in the twentieth century have we had this very different approach towards illicit drugs to make them illegal. In the nineteenth century opium was quite popular. There was even a nice cough syrup for children, called 'Godfrey's', which contained opium.

There are very different trends in epidemics in relation to illicit drugs, because across a population there may be a rise in popularity of a particular drug, which then drops off again. This is in contrast to alcohol and tobacco where rises and falls in use occur only over much longer time scales.

By far the most popular drug in industrialised countries is cannabis. Some of the drugs that receive the most press attention, such as hero-

in and cocaine, are very much a minority habit in terms of regular use.

There are far fewer deaths resulting from consumption of illicit drugs than from either tobacco or alcohol, although drug deaths are more highly concentrated in the younger age groups. There are, however, significant physical and mental health problems amongst dependent drug users. More recently there has been the link between injecting drug use and a range of infectious diseases. First of all the concern was with HIV. In southern Europe a large part of their increased HIV rates was in the injecting drug-using population, mainly because treatment facilities were not available for them. We are also seeing very high levels of hepatitis B and, more worryingly, hepatitis C. These will bring significant future problems. We are therefore already beginning to build up health problems for the twenty-first century.

### Future prospects

What could happen in the twenty-first century? We are currently seeing great attention paid to tobacco, not only in the UK but also through the WHO. Globally, the tobacco-related disease burden is set to rise. It is estimated in a World Bank study (Prabhat and Chaloupka, 1999) that by 2030 10 million deaths a year will be from smoking-related causes. It is by no means a conquered problem, therefore. As a cigarette manufacturing country, even if we succeed in reducing our smoking rates, we are to some extent exporting our health problems to poorer countries.

We have been fairly quiet on the alcohol front, but it is an old and favourite drug and could well re-emerge as a major health policy issue.

Today's young people have a much more liberal attitude to drugs of all sorts and abuse many substances in many different ways. They have a very different drug-taking history from earlier generations, and it is not clear what will happen to the poly-drug users as they age or what are the health consequences of the mixture of drugs. Many of the compounds now being taken are quite new. We have seen in the twentieth century a switch from use of naturally occurring substances

to more consumption of manufactured substances. It is unclear what the health consequences of that switch will be. In the twenty-first century there will be new drugs and new problems. We are, however, beginning to understand better – both from a psychological and a brain chemistry perspective – the nature of dependence: why people go on taking things that are harmful for them, even when they are not necessarily obtaining the benefit which they got at the start.

### The government role

When we look back over the twentieth century, we have not been very successful in devoting attention to the legal drugs, tobacco and alcohol. There are many reasons given for this lack of policy action. The one we have seen most, certainly in the media in this country, is the idea that government should not control individual behaviour, should not be a ‘nanny state’. There may also be some political anxiety that if drugs are popular, then attempts to reduce their use will drive away voters. The other major reason given for inertia, and one that is also much misunderstood, is that because alcohol and tobacco are so important economically, their health effects should be ignored. That says something about the agenda and the political values that have been in place in the twentieth century.

There are many reasons why government should intervene in this area. First, there are still many deficiencies in knowledge; for instance that giving up smoking will bring benefit. People cannot make rational decisions in the neo-liberal sense if they do not have information. Secondly, there is a whole range of third-party effects. Smokers, drinkers and drug-takers do not only damage themselves; they also affect third parties. There may be direct harm such as drink driving – a major impetus behind policy in the US. There may also be other, indirect, reasons why particular individual behaviour impacts on others – through our institutional structures, because, for instance, we have a free health service in the UK. Finally, there is the problem of dependency, which can be seen as undermining the liberal desire to rely on people to behave rationally rather than risk a ‘nanny state’.



## Are there cost-effective interventions?

In looking at policies to improve health by changing individuals' consumption of tobacco, alcohol and illicit drugs, there are many types of cost and benefits to be taken into account. Costs and cost savings may arise in several areas:

- direct costs of administering and implementing the policy;
- indirect costs, borne by other agencies than health care providers;
- costs to individuals and families, both the direct costs of the policy and indirect costs such as loss of benefits from consumption. It may be tempting to overlook this category of costs but we have to recognise that for many people their drug-taking habits bring them benefits. So if we impose policies aimed at controlling their habits, there may be some loss of benefit to those people.

Benefits can also arise under a variety of headings:

- contributions to quality and quantity of life for the individual and their family;
- benefits to third parties. Governments are pressing health authorities to put more money into drug treatment, but what they are actually aiming for in this is to reduce crime, not necessarily to improve the health of the population.

There are various types of policies and these have different patterns of costs and benefits. We can have population-based policies. Some of these, like taxation for smoking and alcohol, are very indiscriminate, blunt instruments. We can have information provision policies, which libertarians would see as being preferable because they are getting people to change their behaviour themselves, without loss of consumption benefit – because they are making the choice freely. The difficulty is that, when we look at the evidence, the more draconian policies tend to work better than the others do.

Then there are problem-based policies. In the drugs world this includes the harm-reduction strategies rather than tackling drugs and drugs consumption *per se*.

**Table 3.1 Potential contributions of policies to reducing smoking prevalence**

|   |     |
|---|-----|
| Advertising ban                               | 5%  |
| Sustained health campaigns                    | 7%  |
| GP advice                                     | 5%  |
| Smoking bans in public places and work places | 5%  |
| Tax policy (after adjustment for income)      | 20% |

Source: Townsend, 1993.

Finally, you can have face-to-face policies, such as health service interventions.

There is quite a lot of evidence on cost and cost-effectiveness, much of it from the US, particularly for policies against smoking but also against alcohol abuse. In general, tax has been shown to be a more cost-effective strategy than the problem-based approach. For instance, in the US they raised the legal age for drinking, which did have some effect; but higher taxes on alcohol were both more effective and more cost-effective.

There is a lack of evidence on the cost-effectiveness of simple information giving, particularly for school-based programmes, changing individual behaviour. Despite this, governments like spending money on school-based programmes.

If we look at policy options, there are a lot of different trade-offs between what costs and what benefits there will be. What is crucial in evaluating them is what values we put on these different items. It is a very moral-based, value-driven area and I do not think we can get away from that. If we look at most of the evaluations of treatments for people with dependencies, many of those evaluations have put no value on the individual's gains at all, which, for a health economist, seems strange. Instead the evaluations have stressed that if we treat people with alcohol dependency or drug dependency we will bring benefits to other people; implicitly saying: 'as a society, we don't value people with alcohol and drug problems'.

Table 3.1 shows some figures estimated by Townsend (1993) for what, if we had a comprehensive programme of anti-smoking policies, the contributions of different types of policies would be to reducing the prevalence of smoking. The point is that if we want in the twenty-first century to change individual behaviour, then effective policies exist but you would probably need to apply quite a lot of them to have a major impact. There is very unlikely ever to be a magic bullet.

Townsend estimated that if there were more intervention by the health service, this would make a major contribution towards reducing the prevalence of smoking. There have been a number of studies on smoking cessation interventions, using a number of different techniques. These are among the most cost-effective things the health service could do to improve quality and quantity of life. Parrott et al. (1998) estimated a cost per life year saved in the range £174-873. All the surveys suggest, however, that smoking cessation interventions are not being applied in a widespread way. One of the actions being taken as a result of the UK government's 1998 White Paper on tobacco, *Smoking Kills*, is to put more money directly into health service care, and this may be one area where we see a change.

There is very similar evidence in relation to alcohol. Brief and opportunistic interventions by primary care physicians have been shown world-wide to reduce drinking levels quite significantly and at low cost. There is less epidemiological evidence about what those drinking levels mean in terms of improved health and reduced mortality rates but, from all the other evidence we have, one would expect those benefits to follow.

The studies that have been carried out on drug and alcohol treatments seem to suggest that not only are these cost-effective, they might also yield net financial savings.

There is also a huge range of potential for new treatments in this area. With more understanding of the pathways by which dependency is formed for the more serious cases, we are beginning to develop new therapeutic drugs. Through better research, we are also beginning to know more about social and psychological treatments. The problem remains, however, that these are not being delivered.

## The future contribution of health services

Summing up, will the health service contribute more to improving quality and quantity of life in this area in the twenty-first century? A big issue here is how much weight we put on health in the future. This is particularly important for areas such as smoking where the health impact develops over a long time. In economists' terms, this is all to do with discounting. It is also to do with political discounting as well as health discounting, however. In what sense do governments, which have only a limited life, commit themselves to long-term population health improvement and the policies necessary for it? This becomes even more important if we are looking at children's health.

To achieve progress the health service must feel that it owns the problems of illness related to smoking, alcohol and illicit drugs. If we have a genuine move towards evidence based medicine this should reinforce and increase activity in this area, because the evidence of cost-effectiveness exists and is very favourable for many of the interventions.

Countering that is may be attitudes in the twenty-first century towards lifestyles. Arguably, during a lot of the twentieth century we have tended towards victim blaming. 'It's people's individual responsibility. It's their fault if they get into bad lifestyles.' What we are not clear on is how these attitudes might change in the twenty-first century, and how our health and care agencies will react towards it. Increasingly, certainly with smoking, we see it as a poverty issue. These wider aspects may start to weaken and undermine some of the victim-blaming attitudes.

We also need more joined-up policies. I am rather sceptical as to whether these will work, because our whole system of government is set up with different people having different vested interests in different departments, to keep their areas nice and secure and to maximise their budgets. If we are to tackle many of these problems, they are not just health problems; they are social problems and we need more joined-up thinking.

Finally, the extent of future intervention will in part depend on the moral argument that underlies treatment of smokers, excessive drinkers and other substance abusers. The prevailing morals are very time-specific. If we look back at substance misuse, which is age-old, we see different social attitudes and hence different policy approaches prevailing at different times. It is not clear whether the twenty-first century will see a change to greater intervention than in the twentieth in order to resolve the health problems caused by these individual behaviours.

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## Chapter 4

# How important have medical advances been?

JOHAN MACKENBACH

### Introduction

In St James's Church, Sussex Gardens, London there is a stained glass window, which, in addition to many saints, depicts a small image of Sir Alexander Fleming discovering penicillin. He had his laboratory in the vicinity of the church and ended up on one of its windows. This church window demonstrates the very deep respect shown for advances in medicine amongst popular opinion. But this popular opinion was undermined, quite suddenly, by the work of Thomas McKeown (McKeown, 1976).

### McKeown and his critics

McKeown's analysis of the contribution of medical care to improvements in life expectancy and declines in mortality has the logical structure of a syllogism: it consists of two statements which, if both are true, inevitably lead to the third statement. He first of all showed that three-quarters of total mortality decline in England and Wales was due to infectious disease mortality decline. He then showed that three-quarters of infectious disease mortality decline occurred before antibiotics and vaccinations became available, and then reached the conclusion that the mortality decline was not due to medical care.

He went on from that and looked at other possible influences on mortality decline, reasoning by exclusion. After he had excluded medical care as the major influence, he also excluded several other explanations. He turned to favourable changes in the environment as the most likely explanation of declines in mortality and looked at a number of specific environmental changes. He excluded hygienic measures, as he had excluded medical care, and ended up with his last factor, nutrition, as the main explanation for mortality decline during the twentieth century.

His final conclusion, based on this analysis, was that investments in medical care are misdirected and that changes in the environment will

still be the most likely and most powerful way to solve the health problems of the late twentieth century, as they had been in solving the health problems of the late nineteenth century.

Although McKeown's work has been largely accepted, even inside the medical profession, there have been a number of critics of it. First of all, McKeown has been criticised for the fact that his analysis of the direct effects of medical care was informal only. He did not come up with a quantitative estimate of the effects of medical care. According to his critics, he also neglected the indirect effects of medical care; for example, the role that doctors played in advocating sanitary reform. Then he was criticised for reasoning by exclusion, and for not presenting any direct evidence on the role of nutrition.

Finally, he was criticised for the fact that his comparison of environmental change and medical care seemed a little unfair, because 'The environmental measures were tested when mortality was high; antibiotics, when the mortality was low; environmental measures were tested alone, antibiotics against the background of environmental change' (Lever, 1977). It was suggested that if the order of events had been the reverse, perhaps a stronger impact of medical care would have been found. There is some evidence from developing countries that, when antibiotics and vaccinations were introduced there, the relative effect of these measures was larger than has been observed in western Europe. The same critic pointed to the fact that although English archers won a battle at Agincourt in the Hundred Years War and English riflemen failed to do so on the Somme in the First World War, it does not follow that archers have greater military potential (Lever, 1977).

### **Revisiting McKeown: Mackenbach (1988)**

Since McKeown wrote, a number of authors have published quantitative estimates of the role of medical care in mortality decline, including John Bunker and myself. In a 1988 paper, some colleagues and I tried to come up with a quantitative estimate of the contribution of medical care to improvements in life expectancy – life years gained – in the Netherlands in the period between 1950-54 and 1980-84 (Mackenbach et al., 1988). We identified a number of causes of death for which it would be



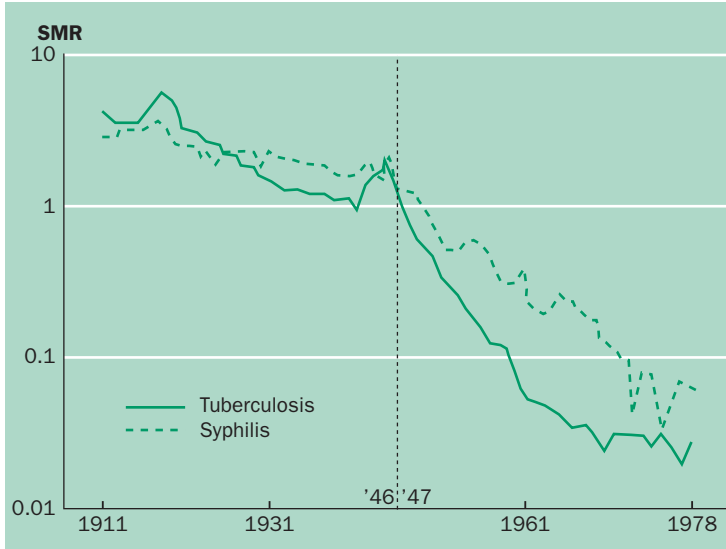
reasonable to assume that improvements in medical care had contributed to declines in mortality. We then reviewed evidence on efficacious treatments for each of these causes of death. Frequently that evidence was not based on randomised clinical trials because of the early dates at which the treatments had been introduced. However, many observational clinical studies were available, which provide some, and sometimes very convincing, evidence on the efficacy of treatments.

We then checked whether mortality for these conditions did indeed decline following the introduction of the new treatments. If both conditions were fulfilled – the treatment was efficacious and mortality from the relevant cause declined following the introduction of the treatment – we included such a cause of death in our selection. We then quantified mortality declines for all these causes of death and calculated their combined effect on the life expectancy of the Dutch population. The result was that, between the periods 1950-54 and 1980-84, declines in mortality from these causes of death added three years to the life expectancy of men and four years to the life expectancy of women in the Netherlands.

Figure 4.1 shows the decline in mortality in the Netherlands from tuberculosis and syphilis; two infectious diseases for which the introduction of antibiotics in the late 1940s was an important medical intervention. The graph presents the age-standardised mortality rates in the Netherlands between 1911 and 1978, on a logarithmic scale. The decline of tuberculosis, which was already present before the introduction of antibiotics, speeded up after the introduction of antibiotics. In the case of syphilis, mortality was more or less stable before antibiotics were introduced, but after 1946/47, when penicillin, the first effective drug for syphilis, was introduced, mortality declined very steeply indeed. Similar evidence was found for a number of other causes of death. For some other infectious diseases I found not only evidence for a steeper decline of mortality, but sometimes also for a very strong stepwise decrease of mortality during the introduction of antibiotics.

Infectious diseases are not the only conditions for which efficacious treatments have been introduced. Many others can also be included.

Figure 4.1 **Falling mortality from tuberculosis and syphilis, the Netherlands, 1911-1978**

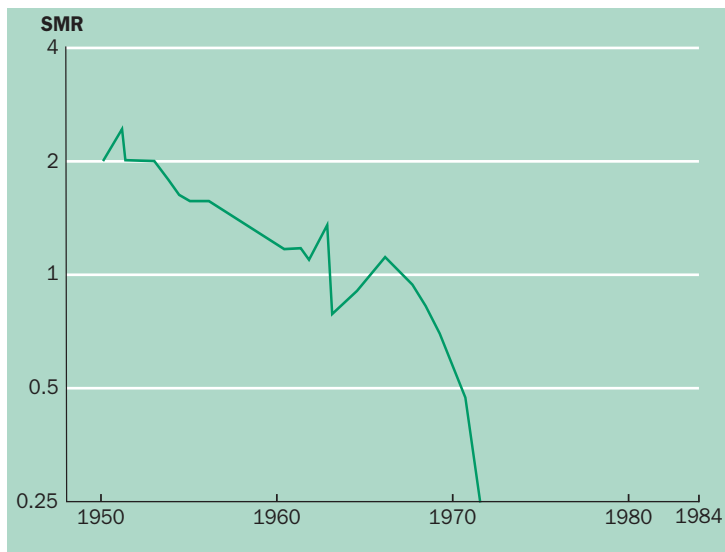


Note: SMR=Standardised mortality ratio, shown on a logarithmic scale and with the average mortality rate for 1911 to 1978 set equal to 1.

Source: Mackenbach, 1988.

For example, haemolytic disease of the new-born, where an abrupt decline of mortality is seen after the introduction of effective treatments (see Figure 4.2). Another example is congenital cardiovascular anomalies, where important innovations in surgical treatment were introduced during the 1950s and 1960s. There is a decline in mortality starting around 1960, approximately coinciding with the introduction of these new surgical treatments (Figure 4.3). As a final example, Figure 4.4 shows the case for Hodgkin's disease, where the introduction of combination chemotherapy around 1970 led to a strong decline of mortality in the Netherlands as well as in many other developed countries.

Figure 4.2 **Falling mortality from haemolytic disease of the newborn, the Netherlands, 1950-1984**

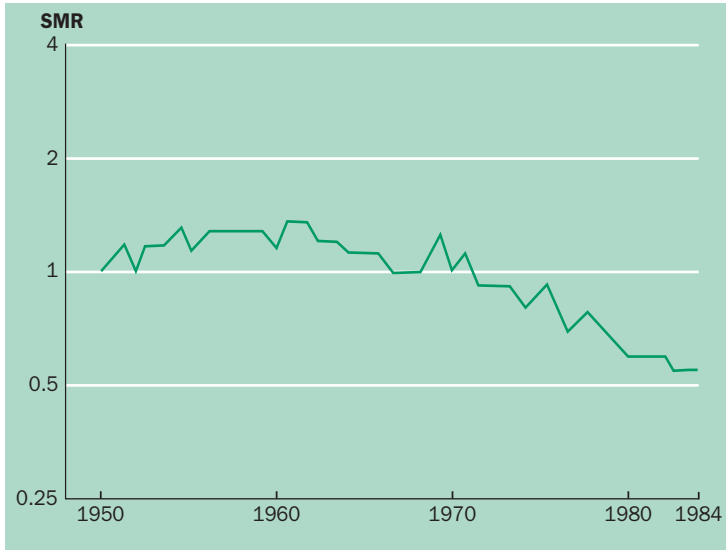


Note: SMR=Standardised mortality ratio, shown on a logarithmic scale and with the average mortality rate for 1950 to 1984 set equal to 1.

Source: Mackenbach, 1988.

Combining all of the conditions where medical intervention accelerated the decline in mortality, implied that three years had been added to male life expectancy in the Netherlands and four years to female life expectancy (see Table 4.1). These are the gains in life expectancy expected on the basis of the reduction of mortality from these conditions. Actual gains in life expectancy in the Dutch population over this period, 1950-54 to 1980-84, were only two years for men but six years for women. Thus, without these declines in mortality from conditions amenable to medical intervention, life expectancy in the Netherlands for men would actually have declined in this period, and for women would only have increased by two years instead of six.

Figure 4.3 **Falling mortality from congenital cardiovascular anomalies, the Netherlands, 1950-1984**



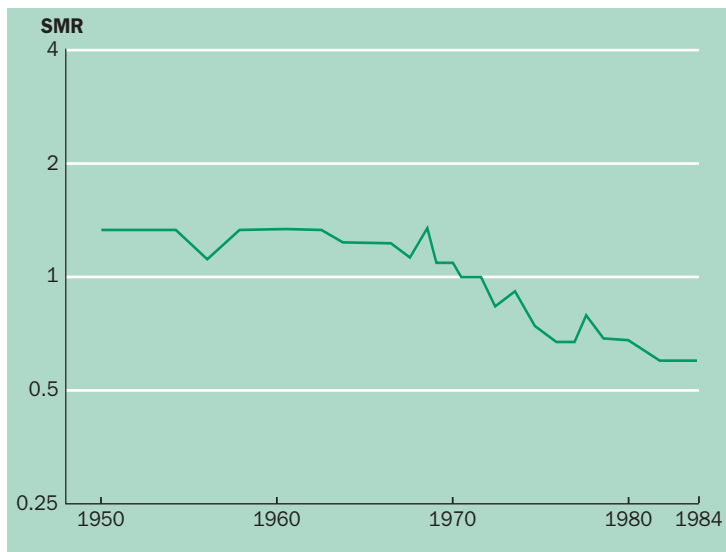
Note: SMR=Standardised mortality ratio, shown on a logarithmic scale and with the average mortality rate for 1950 to 1984 set equal to 1.

Source: Mackenbach, 1988.

I have now updated this analysis to cover the period until 1991-95. There have of course been further innovations in medical care, which may have contributed to improved life expectancy, but I started by recalculating the gains in life expectancy due to the same conditions covered in the 1988 paper. They are all very small further increases in life expectancy, due to the further declines in infectious disease mortality or perinatal mortality.

There have been other causes of death where there have important advances in medical care, however. Ischaemic heart disease is an example. The introduction of thrombolytic therapy is an undisputed innovation that has led to improved survival of patients. It has diffused very

Figure 4.4 **Falling mortality from Hodgkin's disease, the Netherlands, 1950-1984**



Note: SMR=Standardised mortality ratio, shown on a logarithmic scale and with the average mortality rate for 1950 to 1984 set equal to 1.

Source: Mackenbach, 1988.

quickly into the population and has contributed to a change in the trend for ischaemic heart disease mortality: a change that cannot readily be explained by changes in risk factors. I think that there is therefore good evidence that the decline of ischaemic heart disease mortality in this period is at least partly due to improvements in medical care.

Similarly, one of the major cancers has seen an improvement in medical care. The introduction of more radical surgery combined with pre-operative radiotherapy has led to a decline of rectal cancer mortality, instead of the stable figures prevailing before its introduction.

Also for hip fracture, the introduction of more aggressive surgical treatment in elderly patients, with earlier mobilisation of patients, has

**Table 4.1 Life expectancy gains (in years) due to declines in mortality from amenable conditions, the Netherlands, 1950/54-1980/84**

|                                  | <b>Men</b>  | <b>Women</b> |
|----------------------------------|-------------|--------------|
| 'Surgical conditions'            | 0.36        | 0.25         |
| Perinatal/maternal               | 0.72        | 0.63         |
| Infectious diseases              | 0.94        | 1.13         |
| Hypertension/stroke              | 0.43        | 1.32         |
| Other                            | 0.58        | 0.80         |
| <b>All amenable conditions</b>   | <b>2.96</b> | <b>3.95</b>  |
| Observed gain in life expectancy | 1.91        | 5.86         |

Source: Mackenbach et al., 1988.

probably led to a decline of case fatality and a decline of mortality in the population as a whole.

The figures for the contributions of these new conditions to improvements in life expectancy are more substantial. Even if only a part of that were due to improvements in medical care, it would still account for quite a substantial part of the observed gains in life expectancy in the period 1980-84 to 1991-95, which is one and a half years for men and three-quarters of a year for women. Table 4.2 sets out my estimates of the medical contribution.

But there are weaknesses in this analysis and particularly the assumption that all of the declines in mortality after the introduction of effective medical treatments were due to these medical innovations. For example, I counted all declines in mortality from infectious diseases after the introduction of antibiotics and then assumed that all of that decline was due to antibiotics. Of course that is unlikely to be true. Infectious diseases may decline for other reasons too, such as improved living conditions.

**Table 4.2 Life expectancy gains (in years) due to declines in mortality from amenable conditions, the Netherlands, 1980/84–1991/95**

|   | <b>Men</b>  | <b>Women</b> |
|---|-------------|--------------|
| 'Surgical conditions'                     | 0.05        | 0.03         |
| Perinatal/maternal                        | 0.04        | 0.04         |
| Infectious diseases                       | –0.04       | –0.02        |
| Hypertension/stroke                       | 0.14        | 0.18         |
| Other                                     | 0.04        | 0.04         |
| Ischaemic, rectal cancer and hip fracture | 0.84        | 0.55         |
| <b>Total</b>                              | <b>1.07</b> | <b>0.82</b>  |
| Observed gain in life expectancy          | 1.54        | 0.76         |

Source: Mackenbach, 1999, unpublished data.

### Revisiting McKeown: Mackenbach (1996)

I therefore made a fresh attempt, in which I tried to estimate more directly the contribution of medical care to declines in mortality after the introduction of effective medical treatment. I started with the same selection of causes of death as in my previous attempt, but now tried to estimate the proportion of mortality decline that may have been due to medical care after the introduction of efficacious treatment. This enabled me to calculate the proportion of total mortality decline that was due to medical care. This second attempt also included medical innovations occurring before 1950, so that I had an estimate for the century since 1875 (Mackenbach, 1996). The end result was an estimate that between 5 per cent and 18 per cent of total mortality decline in the Netherlands in the period 1875–79 to 1970 can be attributed to improvements in medical care. Although the width of this range of values indicates the substantial uncertainty that exists, it nevertheless indicates the order of magnitude of the impact of medical interventions.

The way in which this range was calculated is illustrated in Tables 4.3 and 4.4. As an example these just focus on the part of the calculation that is concerned with the impact of antibiotics on mortality from infectious

**Table 4.3 Trends in age- and sex-adjusted mortality from infectious diseases before the introduction of antibiotics (1921-1939) and after the introduction of antibiotics (1947-1968)**

| Disease                      | Annual percentage change in mortality* |           |
|------------------------------|--|-----------|
|                              | 1921-1939                              | 1947-1968 |
| Bacillary dysentery          | +5                                     | -19       |
| Typhoid fever                | -10                                    | -2        |
| Scarlet fever                | -3                                     | -13       |
| Measles                      | -8                                     | -9        |
| Tuberculosis                 | -6                                     | -16       |
| Pneumonia                    | -4                                     | -5        |
| Acute bronchitis             | -7                                     | -9        |
| Influenza                    | -1                                     | -5        |
| Syphilis                     | -1                                     | -9        |
| Erysipelas                   | -2                                     | -11       |
| Septicaemia                  | -6                                     | -4        |
| Meningococcal meningitis     | -1                                     | -6        |
| Non-meningococcal meningitis | -8                                     | -4        |
| Otitis media                 | +5                                     | -8        |
| Upper respiratory infections | +3                                     | -5        |
| Cystitis                     | -4                                     | -6        |
| Skin infections              | -3                                     | -7        |
| Osteomyelitis                | -1                                     | -5        |
| Rheumatic fever              | 0                                      | -16       |
| Puerperal fever              | -1                                     | -11       |
| All infectious diseases      | -4                                     | -10       |
| All other diseases           | -1                                     | 0         |
| <b>Total mortality</b>       | <b>-2</b>                              | <b>-1</b> |

Note: \*Estimated on the basis of a Poisson regression analysis, in which the mortality rate was modelled as a function of calendar year and the number of person-years at risk by age and sex. The period 1940-1946 was left out of the analysis because of the effects of World War II.

Source: Mackenbach, 1996.



diseases. Table 4.3 shows annual percentage changes in mortality from a number of infectious diseases. The two columns show respectively the average annual percentage change in mortality before and after the introduction of antibiotics. There was an average 4 per cent per annum decline in mortality from all infectious diseases taken together before the introduction of antibiotics, and an average 10 per cent per annum fall afterwards. We can then take the difference between these two as an estimate of the contribution of antibiotics to mortality decline for infectious diseases. I therefore took 60 per cent of the decline after the introduction of antibiotics to be a reasonable estimate of the impact of medical care on the decline in infectious disease mortality after that period. To allow for the uncertainty that surrounds this estimate, I assumed that the lower limit in this case was 25 per cent and the upper limit was 75 per cent. Table 4.4 shows how this range was then used to calculate the contribution of antibiotics to the decline in mortality in the Netherlands over the whole period between 1875-79 and 1970. Antibiotics account for 1.6 per cent to 4.8 per cent of the total decline in Dutch mortality over that period (Mackenbach, 1996).

**Table 4.4 An estimate of the direct contribution of medical care to the decline in mortality between 1875/1879 and 1970 in the Netherlands**

| Steps in calculation   | Intermediate results (%) | Contribution of medical care to decline in mortality |                 |
|--|--------------------------|--|-----------------|
|  |                          | Lower limit (%)                                      | Upper limit (%) |
| a. Contribution of specific infectious diseases to decline in total mortality 1875/1879-1970 | 46                       |  |                 |
| b. Of which after 1946   | 14                       |  |                 |
| c. Of which due to medical care  | 25-75                    |  |                 |
| d. $a \times b \times c$   |                          | 1.6  | 4.8             |

Source: Mackenbach, 1996.

I made similar calculations for other groups of causes of death. By summing across all causes of death, the result was the total estimate of between 5 per cent and 18 per cent – let us say 10-12 per cent to take the middle of the range – of total mortality decline since 1875 in the Netherlands which may be due to improvements in medical care (Mackenbach, 1996).

### Revisiting McKeown: Bunker (1994)

A completely different approach was taken by John Bunker and colleagues, who produced an elegant attempt to estimate the contribution of medical care to improvements in life expectancy (Bunker et al., 1994). While my approach was from observed mortality declines, Bunker et al.'s approach has a different starting point. It does not look backward from mortality declines to the influence of medical care, but looks forward, starting from what we know about the effects of medical care on patients and extrapolating the effect for the total population.

Bunker and his colleagues started, as did my colleagues and I, with a selection of causes of death for which there is evidence of efficacious treatment. This evidence was largely based on clinical studies, sometimes carried out some years ago, for treatments in disease areas where there is evidence of mortality decline following the introduction of effective medical treatments. Although his selection of causes of death is not identical to mine, it shows a substantial degree of overlap. Bunker et al.'s approach was then to estimate the size of the target group in the total US population for each of these conditions. That is, they first estimated the number of patients suffering from the relevant diseases. Then they estimated the proportion of those patients reached by medical care, which is not necessarily 100 per cent – for example, we know that perhaps only half of all patients with hypertension in the population are detected. Then, for the proportion reached, Bunker et al. estimated the number of life years gained per individual. This estimate was based mainly on the results of randomised controlled trials or other clinical studies, quantifying the effect of medical care on mortality risks for patients who suffered from these conditions and were treated for them. The outcome of all

this was that they estimated that the US population had on average gained five life years as a result of medical interventions (Bunker et al., 1994).

As an example of how Bunker and colleagues reached this estimate, let us look at one medical intervention: screening for hypertension. They estimated that 58 million Americans may potentially benefit from preventive services in this area but that, in practice, only 50 per cent of them would be reached by such screening. On the basis of trial results, they estimated that those who are reached by the screening and, when required, the subsequent preventive regimen gain three months' life expectancy on average. On this basis Bunker et al. therefore estimated that the total gain in life expectancy when averaged out across the total US population was between one and a half and two months.

They repeated that analysis for many different causes of death, and so came to the final estimate of five years of life expectancy gained by the US population, with a potential for adding a further one and a half or two years if the existing services could be made to reach 100 per cent of the relevant target populations (Bunker et al., 1994).

It is perhaps useful to compare these different attempts. It is interesting to see that there are similar but not identical selections of causes of death. I included perinatal conditions, rheumatic disease and certain cancers in children, which are lacking in Bunker et al.'s analysis, and they included some conditions that I did not. This demonstrates some uncertainty on the part of those working in this area about the conditions for which there is convincing evidence of effective medical interventions. As I have already noted, our methods are different. Mine is essentially a backward reasoning method: starting with the observed decline in mortality and reasoning back to what the possible contribution of medical care could have been. Bunker and colleagues used a forward reasoning method: taking what we know from clinical trials about the effects of medical care and then extrapolating those results to the population as a whole.

Despite these differences, the results of the two analyses are broadly similar. Bunker et al.'s effect size is five years out of a total increase of

life expectancy of approximately 30 years (from 45 to 75) over the twentieth century. Thus they are attributing to medical interventions about one-sixth of the decline in mortality during the twentieth century. My estimate for the contribution of medical care to declines in mortality in the century from 1875 was between 5 and 18 per cent; not so far from what Bunker and his colleagues estimated.

Although the results are similar, it is important to acknowledge the limitations of these studies. They can be characterised as rather heroic attempts at a formal analysis of the direct effects of medical care. There have been a number of assumptions made by both. The methods have been rather crude. They ignore heterogeneity within the population, heterogeneity of patient groups. The evidence base is fragmentary and the evidence for effective medical intervention is not strong for all these conditions.

I think that it is likely that, for selected causes of death, the estimates may be too generous. For example, in Bunker et al.'s analysis the extrapolation of trial results may suffer from a common bias in this type of analysis. Because trials are done in selected patient groups under carefully controlled circumstances, we cannot readily extrapolate the results to the population as a whole and everyday usage. A treatment may prove less effective in widespread use than it was in the clinical trial. In my own analysis, attributing all of the difference in the rate of mortality decline before and after the introduction of a medical intervention may also be too generous. Other factors may also have changed, together with the changes in medical care.

On the other hand, although we may have been too generous for the selected causes of death, in the estimates there is a total neglect of other causes of death. If medical care has made a difference for the selected causes of death, it is likely that it will also have made at least some impact on other causes of death. For example, improved surgery will not only have affected the three or four conditions which were selected by Bunker and myself, but may also have had smaller effects on other conditions for which surgery is offered to patients.

At least Bunker's and my results can be taken as a basis for comparison with other determinants of mortality decline, if such estimates

are available – but I do not know of any similarly quantified estimates for the contribution of other factors to the fall in mortality over the twentieth century.

### **A broader perspective**

McKeown's work, Bunker's and my own are all based on historical evidence, and I think that we must acknowledge that such historical evidence is of limited value. We have a serious lack of relevant data to help us make causal connections between improvements in medical care and mortality declines. Furthermore, the evidence is at best quasi-experimental but mostly observational in nature.

It is important to be aware, however, that this limitation of historical evidence applies not only to medical care but also to the competing explanations for mortality decline. For example, the evidence for the effectiveness of piped drinking water, which is something we all believe in as a contributor to the decline of mortality since the late nineteenth century, is also very shaky.

What should we do when historical evidence does not help us any further; does not help us to decide about the most cost-effective areas for health care investments now? It is obvious that, when we consider current investments in health care, we should use contemporary evidence to support our decision, e.g. cost-effectiveness studies. This should apply both to innovations in medical care and to innovations in other areas aimed at improving population health. I am not so sure that the other options which we could consider for improving health in the population would then prove to be more cost-effective than innovations in medical care.

Current evaluation procedures for medical care compare rather favourably with those of many other policy options. As Michael Rayner has already said for nutrition, the evidence base for improvements in nutrition and for policy options in that area is not very strong. The same applies to many other policy options. Our conviction that nutrition or many other factors – socioeconomic factors, education – are important for public health is mostly based on observational evidence, not on experimental evidence showing the effectiveness of the policies which

we are recommending. As a paradoxical effect of McKeown and other work in this area, medical care has very much improved its evidence base and may now serve as a role model for many other policy areas.

It is perhaps not necessary that we should make a trade-off between investments in medical care and other policy options. Most developed countries spend 7 per cent to 10 per cent of national income on health care and surveys show that the population is generally unwilling to support any cutbacks in this funding.

What McKeown alerted us to, however, was that other health policy options, in the field of nutrition or socioeconomic factors, are potentially powerful and that the development of an evidence base to support recommendations in those areas should have high priority.

## Conclusions

My main conclusion is that McKeown was both right and wrong. He was right because medical care's contribution to improvements in life expectancy in the twentieth century was less than 20 per cent; so it is only a small fraction of the total improvement in life expectancy. In my opinion, however, even if it is less than 20 per cent, even if it is only 10 per cent, that is still a substantial contribution. Furthermore, the more recent the period you look at, the greater the relative contribution of medical care appears to be. I therefore also think that McKeown was wrong. There is no basis in his work for the conclusion that investments in medical care are misdirected.

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## Chapter 5

# The effect of urban pollution and transport on health

MARK MCCARTHY

### Introduction

At the beginning of the twentieth century, public policy in the industrialised nations was actively directed towards environmental improvement on health grounds. There was new housing, streets were paved, drains were built for surface water and sewage, and waste disposal was organised. During the century, the conversion of domestic heating from coal fires to gas, oil and electricity has cleaned up urban air. Heavy industry has been sited away from population centres. Economic activity also is changing, from production of goods towards provision of services, and the future, perhaps, is electronic rather than mechanical.

The paradigm of this environmental improvement may be found in the 'sanitary idea' of Edwin Chadwick, whose bicentennial anniversary is celebrated this year. As secretary to the Poor Law Commission, Chadwick wrote the Report on the Sanitary Condition of the Labouring Population in Great Britain, which was published in 1842, and led to the Public Health Act of 1848. The Sanitary Report drew on a survey by Chadwick of local informants, usually practising doctors, across the country. Yet Chadwick was selective in his use of this information (see Hamlin, 1998). Many doctors, and especially the two thousand who were medical officers to Poor Law Unions, saw and described disease caused by poverty – lack of food, of heating, of clothing.

But Chadwick rejected this interpretation. Doctors, he wrote, 'deceive themselves... by what they call the evidence of their own eyes'. He claimed disease was caused 'by atmospheric impurities produced by decomposing animal and vegetable substances, by damp and filth and close and overcrowded dwellings'. Chadwick firmly believed in environmental causes of disease, and, through the General Board of Health, promoted 'sanitary' improvements of clean piped water, municipal drainage, and new cemeteries. Furthermore, it has been suggested that Chadwick's reforms protected conservative power in



government from more radical criticisms centring on the economic causes of disease (Hamlin, 1998).

### Urban pollution and health

When people in London were asked at the end of 1998 what affected their health, 60 per cent cited poor air and traffic (Evening Standard, 10 December 1998). Twenty-seven per cent were concerned with dirty streets, and a similar number replied smoking. Less than 10 per cent described poor quality housing. Of course, in part this reflects the fact that everyone is affected by air, whereas only some people smoke. But public perception must be one consideration for public policy. The environmental issues of atmospheric pollution and traffic are evidently still related to health in the minds of the many citizens in London, and probably therefore in many other urban settings.

Several issues arise in discussing the effects of urban pollution on health:

1. Urban environments are not homogenous. Some people live in dense urban areas, close to roads. Others live at lower densities e.g. in the suburbs, which imitate countryside while retaining easy access to urban facilities. Cities themselves are not identical: apart from size, they differ in planning and construction as well as occupational structure. To investigate 'urban pollution' we therefore need to define which characteristics of the urban environment concern us.
2. It is commonplace now for epidemiology to recognise that a single disease may have many causes. Thus, respiratory disease in adults is caused by (any) combination of smoking, infections, development in childhood, allergens, etc.. How does 'air' contribute; and which aspects of air: gases, chemical vapours, suspended acids, or particles?
3. The basis for some environmental epidemiology is extrapolation from high exposures in controlled settings (for example, industrial pollution of workers). Less may be known about low expo-

tures of larger populations over their lifetimes. Is there a threshold effect, or a simple dose-relationship?

4. Painstaking epidemiological studies have been needed to demonstrate the effect on health of individual agents, such as smoking. Larger sample sizes, new methods of quantification and long-term follow-up are still needed to investigate the effect of population environmental exposures. Migration, increasingly common in urban settings, can seriously affect interpretation of results.
5. There has been much success in technical environmental control. A good urban environment depends on continuing success. We tend to acknowledge the impact of the environment when control is lost – in an ‘accident’ or ‘incident’. Rather than poor health being due to environmental pollution, current good health depends on keeping this control.

Beyond interpretation of the relationship between environment and health, there is the question of action. While health care and, increasingly, health promotion fall within the control of the health care system, environmental issues are typically controlled by other public agencies. So they are subject to competing values. For the health service, high value is placed on health improvement; for an economic project, an improved environment may be consonant with health benefits, but the choices may be more influenced by economic benefits than by health impacts.

## **Environment and health in London**

Some of these issues can be considered through studies we have recently undertaken for the National Health Service (NHS) Executive’s Regional Office for London (McCarthy, 1999a; McCarthy and Ferguson, 1999). A new political assembly is being created for London, with elections for the mayor in May 2000. The mayor and the Greater London Assembly will inevitably take an active interest in public health in London. The NHS Executive Regional Office is developing a London-wide Health Strategy, and has asked for analyses of the impact of environmental pollution and transport on health in London.

London has a population of seven million within its current boundaries and has an economy of similar size to some European Union states (Denmark, Ireland, Portugal). London was once a major industrial city, with factories and industry scattered between dense terraced housing. Now London's economy is linked to the south-east region of England, as many people working in the city centre commute to work (mainly via public transport), and globally through the City, which creates one third of London's total income through financial services.

Judged through mortality rates, London has a health status around the average of European capitals (Bardsley, 1999). But there are, of course, variations within London. Mortality is higher in the inner eastern and southern parts of the capital, and lower in the outer areas. This spatial distribution is associated with poorer housing, and more broadly with social deprivation. Some of this difference may be due to migration, both inwards and outwards. Thus, registrations of tuberculosis are higher in the inner London boroughs; but BCG vaccination has protected most children in London, and new cases of tuberculosis there now are mostly diagnosed in people who were first infected abroad.

Within that overall perspective, four specific aspects of the urban environment in London may be considered separately: air, water, waste and noise.

### **Air**

External air, internal air and personal air (environmentally transmitted smoke) all impact on health. The effects of external air assessed in two meta-analyses (Kumzli et al., 1999; Committee on the Medical Effects of Air Pollution, 1999) are summarised in Table 5.1. This table illustrates the effects on a population the size of London's of reductions in the quantities of air pollutants present. The WHO report drew from Austria, France and Switzerland information about particulates, and also drew on American prospective studies, to try to suggest the long-term effect of air pollution on health. The first row of Table 5.1 shows that against a current background level of particulates (PM10s) in the air of 7-8  $\mu\text{g}/\text{mm}^3$ , a 1  $\mu\text{g}/\text{mm}^3$  reduction in the level of air pollution would be expected to lead eventually to 245 fewer deaths

**Table 5.1 Air pollution: estimates of additional deaths and hospital admissions for an urban population of 7 million**

|  | <b>Deaths<br/>All causes</b> | <b>Admissions<br/>Respiratory</b> | <b>Admissions<br/>Cardiovascular</b> |
|--|------------------------------|-----------------------------------|--------------------------------------|
| WHO (long-term):<br>per 1 $\mu\text{g}/\text{mm}^3$ PM10 | 245                          | 1,120                             | 210                                  |
| UK (acute):<br>all PM10 and NO <sub>2</sub>              | 740                          | 1,300                             | not known                            |

Sources: Kumzli et al., 1999; Committee on the Medical Effects of Air Pollution, 1999.

each year in London. There should also be 1,120 fewer hospital admissions for respiratory diseases and 210 fewer admissions for cardiovascular reasons. The WHO report considers the impact of an incremental improvement in pollution levels; it does not address the question of the total impact of air particulates on urban mortality.

However, that sort of estimate of total impact has been made in the UK for particulates and nitrogen dioxide (Committee on the Medical Effects of Air Pollution, 1999). The second row of Table 5.1 shows that estimate: for a population of seven million, i.e. London, 740 deaths per year may be attributable to air pollution by particulates (PM10s) and nitrogen dioxide (NO<sub>2</sub>), and 1,300 hospital admissions. These are very rough figures and must be interpreted cautiously, but they are suggestive of the current ideas of the direct impact of air pollution on health in an urban setting such as London.

These meta-analyses of several studies suggest that short-term increases of air pollutants can increase death rates from chronic respiratory disease. However, there is less evidence of an effect on asthma: asthmatic symptom rates appear to be similar between rural and urban settings. Studies in the US suggest that air pollution also causes respiratory disease over the long term, but there is as yet no direct evidence for European populations (Kumzli et al., 1999).

Internal air relates both to quality of housing (especially insulation and damp-proofing) and also how people live in houses (ventilation

and heating). Asthma is closely associated with allergens from house dust mites; moulds and chemical products also cause respiratory symptoms. But smoking probably has the greatest individual effect on respiratory disease.

### Water

The quality of water in London has improved during the 1990s, through active measures taken by water companies to limit pollution, and by attention to monitoring reservoirs and treatment works. Nevertheless there are still occasional major, and many minor, pollution incidents recorded each year (Table 5.2). There is also some dispute whether the 200 or so *Cyptosporidium* infections reported each year are from drinking water or from other sources. As a precautionary measure, the government has required new continuous monitoring of water supplies at source rather than relying only on tap water samples.

### Waste

Waste is a growing problem. It comes from household, commercial and industrial sources. Most of London's waste is taken to landfill sites outside London (some down the River Thames in barges, but much – especially construction waste – taken by private contractor lorries to private sites). The hazards of landfill are poorly determined. Even where tips are protected from human trespassers, there remain two potential environmental health risks: methane and other gases arising by combustion, and materials leaching into water supply. The

Table 5.2 **Water pollution incidents in London, 1997**

|                | Major | All | % for UK |
|----------------|-------|-----|----------|
| Organic wastes | 0     | 1   | 0.1      |
| Fuels and oils | 0     | 251 | 4.5      |
| Sewage         | 2     | 182 | 3.0      |
| Chemicals      | 0     | 44  | 2.8      |
| Other          | 1     | 120 | 2.3      |

EU has sought to reduce the use of landfill for waste disposal, and the UK government has supported this by taxing landfill by weight. However, there is resistance to alternatives such as incineration, and environmentalists would prefer other options of waste reduction, reuse and recycling (London Waste Regulation Authority, 1995). Not only do we not have much action in controlling our waste, neither do we really have much knowledge of the current levels of health impact of the waste. There is very little monitoring of the impact of landfill or of incineration.

### **Noise**

The EU is currently developing legislation and directives on ambient noise. Noise is of concern in both occupational and leisure settings. Noise exposure at work that is likely to cause deafness is regulated in the UK by the Health and Safety Executive. Domestic noise at night causing nuisance to others is controlled by local authority environmental health departments.

Noise from transport is becoming the main area of contention. A major report in the Netherlands (Health Council of the Netherlands, 1999) reviewed the health consequences of large airports (in relation to Amsterdam Schipol Airport). It drew on the scientific literature and studies of other airports, including the inquiry into the proposed fifth terminal at London Heathrow. The report affirmed evidence that chronic noise exposure causes hypertension and ischaemic heart disease, as well as sleep disturbance and annoyance (both of which may contribute to mental illness). This report does not estimate a population disease burden as a result of noise but at least highlights the importance of the impact of noise on health. Studies of people living next to main roads confirm that noise stress increases with proximity and volume of traffic.

The biological mechanisms of the effects of noise need further research. They may be direct, by stimulating the nervous system and increasing hormonal activity; they may be indirect, through feelings of fear, depression or frustration. Familiarity with the noise, attitude towards it (e.g. control over it), or co-existence of other medical conditions may modify an individual's response.

## Transport and health

The discussion of noise indicates the complexity of revealing the effects of the environment on health. We do not always know the intensity of exposure, the numbers of people exposed or the numbers of people affected over time. But health protection for air, water and noise seeks to minimise exposure towards a goal of absolute safety. In transport, the debate is different. The objective of health itself is contested. Public and private agencies appear in their actions to condone damaging effects of transport, and they are deficient in supporting health-promoting transport. The situation is perhaps similar to that for smoking in the 1960s. In 1962, at a time when half of all doctors were smokers, the UK Royal Colleges of Physicians produced its report *Smoking and Health*. With this, British doctors led the way in recognising the harmful effects of smoking that are now widely accepted.

Ten years ago the Transport and Health Study Group, created by academics in transport studies and public health practitioners, started a series of seminars to gather evidence on the effects of transport and health, and published it in 1991 (Transport and Health Study Group, 1991). More recently, the British Medical Association (1997), the Health Education Authority (1998) and the WHO (1999) have also developed policies for transport and health issues.

Transport is a fact of human life, especially for city based communities that depend on trade. Self-sufficient rural communities have relatively low transport needs. Trade in goods requires transport, while large settlements use more transport when individuals are segmented in their choices of work and leisure activities. But providing transport creates demand and use. People live successfully in central Fez and Venice without any mechanised road transport. In Houston and Los Angeles, however, road transport is fundamental to the city. These differences are not chance; they have been designed through choices by public planning authorities.

The health effects of transport can be summarised in four areas: accidents; emissions (air and noise); exercise and community severance (McCarthy, 1999b). To summarise the debate: motor vehicles are

generally harmful to health in these four areas, whereas walking and cycling are beneficial. On the other hand, motor vehicle transport is convenient to users, and there are substantial commercial interests to maintain the status quo.

**Accidents**

Motor vehicle accidents are a major cause of death and disability. Alcohol and other drugs may be a contributory factor for road accidents, but the key point is the individual travelling independently at speeds that kill. Public transport (buses, trains, aircraft) at the same or higher speeds has much lower death and injury rates.

The health effects are significant at population and individual level. In many western countries road accidents are now the leading cause of death in young adults. Head injuries, with effects ranging from days off work to permanent vegetative state, are predominantly from road accidents (statistically this affects motor cyclists, despite wearing helmets, and car occupants more than cyclists (McCarthy, 1991)).

In London in 1996 there were 251 deaths from road accidents; 122 of the people killed were pedestrians and 20 were cyclists (Table 5.3). It is important to emphasise that almost all these deaths are preventable: they are almost all caused by the impact of motor vehicles

Table 5.3 **Fatalities in London by mode of travel, 1996**

|                        |            |
|------------------------|------------|
| Taxi                   | 0          |
| Other vehicle          | 0          |
| Public service vehicle | 2          |
| Goods vehicle          | 6          |
| Pedal cycle            | 20         |
| Powered cycle          | 35         |
| Car                    | 66         |
| Pedestrian             | 122        |
| <b>Total</b>           | <b>251</b> |

Source: Ferguson and McCarthy, 1997.



on human bodies. Why should any of the seven million residents and one million commuters and visitors to London be at risk from being hit by a motor vehicle?

### **Air and noise**

Motor vehicle traffic is a major contributor to environmental pollution and hence, as discussed above, to ill health. New cars are certainly 'cleaner' now than before. But electric vehicles, although technically feasible, are rarely used, and the sheer quantity of traffic on many roads maintains the drone of noise.

### **Exercise**

There is evidence that children are more frequently taken to school by car now than in the past. This is not surprising: public policies have encouraged competitive choice of school and thus greater distances on average for schoolchildren to travel, while more cars and drivers are available to do 'the school run'. Yet the benefits of walking and cycling are probably greater for adults than children. More children take exercise regularly: in our study of 15 year olds in inner London, three quarters exercised more than once a week outside school – football, cycling, running, aerobics and swimming were the most frequent types of exercise (Rogers et al., 1997). For adults after the age of 25, however, exercise rates fall rapidly. Walking and cycling provide natural ways of integrating exercise with other life activities. Yet nationally only 2 per cent of journeys are by bicycle: the government's National Cycling Strategy seeks to raise this level to 10 per cent by 2012 (DETR, 1999).

### **Community severance**

A study almost 30 years ago in San Francisco looked at the relationship between traffic volume and social contacts (Appleyard and Lintell, 1972). With light traffic, people had frequent contact across the street; with heavy traffic, contact was mainly on one side, and people had greater anxiety about using the street because of the traffic. 'Community severance' occurs from through traffic (e.g. in a small town on a main road) rather than when the road is used to access local facilities. A second link in the chain is the evidence that

communities with greater social cohesion have higher health status (Stansfield, 1999). This social cohesion has been measured in terms of personal contacts, membership of community groups, household composition.

**Direct impact of traffic volume on health**

There have not been studies yet to assess the direct effects on health of traffic volume – there are important confounding factors in cross-sectional studies, since traffic density has a negative effect on property prices and poorer people have poorer health for a variety of reasons. But, as the very rich prefer to live in a small village or a suburban cul-de-sac, it is clear that living near heavy traffic contributes to social deprivation and exclusion.

We have tried to put some of this together for London and suggest some quantification of the overall impact of the health effects of traffic (Söderlund et al., 1996). The numbers are no more than guesstimates, but this is at least a start. The second column in Table 5.4 looks at the effect of traffic on the diseases listed. It suggests that probably road accidents are all traffic-related, whereas for air pollution and reduced exercise traffic is only partly responsible for the overall effect.

Table 5.4 **Quantifying the total health effects of traffic in London**

|                                    | <b>% traffic related<br/>(estimates)</b> | <b>10% change –<br/>deaths</b> | <b>10% change –<br/>admissions</b> |
|------------------------------------|--|--------------------------------|------------------------------------|
| Road accidents                     | 100%                                     | 29                             | 600                                |
| Pollution –<br>respiratory and CHD | 1% of deaths<br>5% of admissions         | 24                             | 570                                |
| Exercise –<br>CHD and stroke       | Under 65: 2.5%<br>Over 65: 5%            | 34                             | 102                                |
| Mental                             | 1%                                       | ?                              | 33                                 |
| <b>Total</b>                       |  | <b>87</b>                      | <b>1,305</b>                       |

Source: Söderlund et al., 1996.

Then, taking a 10 per cent change in traffic or a modal shift towards exercise, you might be able to sum something in the region of 80 to 100 deaths and 1,300 hospital admissions that might be prevented each year, from transport policies that were promoting walking, cycling and public transport and that were working against cars.

### Wider issues

Perhaps the debate about environment and health should not be reduced too much to direct measures of health status. Most epidemiological research has used death, life expectancy or disease event rates as measures of health. Yet the 'state of complete physical, mental and social well-being' described in the 1946 WHO definition is probably closer to defining quality of life than health. The environment is both objective pollution and subjective experience, and there are many aspects of urban environment contributing to the quality of life. Table 5.5 shows the characteristics of the physical environment that people in the Netherlands desire, or do not desire, for their neighbourhood.

The industrialised nations are committed to controlling pollution to allow sustainable development – that is, using present resources in ways that do not harm the choices of future generations. The health sector has an important place in debates on policies for sustainable development. Health should be a direct goal of economic development; and there will be health consequences of failure to achieve sustainability.

Indeed, if the definition of health is the wider version encompassed by the WHO, akin to quality of life, then the product of economic development can be quality of life. McMichael and Powles (1999) describe four kinds of wealth for accumulation: normal (physical and environmental) capital; produced (economic) capital; human capital; and social capital. 'In all but the very poorest countries, human and social resources determine mortality levels more than does income.' Even where per capita incomes are not rising, improved human capital (for example falling child mortality rates) is found in countries in association with social policies of public health and essential clinical services, school education for girls and means for communicating

Table 5.5 Attributes of the physical environment

|                  | Scale  |   |  |   |
|------------------|--|---|--|---|
|                  | Dwelling   | Quarter   | Region   | State   |
| <b>Desired</b>   | <ul style="list-style-type: none"> <li>● number of rooms</li> <li>● size</li> <li>● suitable for family</li> <li>● garden</li> <li>● privacy</li> </ul>                              | <ul style="list-style-type: none"> <li>● shops</li> <li>● post office</li> <li>● bank</li> <li>● schools</li> <li>● park, trees, public garden</li> <li>● public transport stops</li> <li>● community centre</li> <li>● pub</li> <li>● library</li> <li>● family doctor</li> <li>● children's playground</li> </ul> | <ul style="list-style-type: none"> <li>● greenery</li> <li>● open landscape</li> <li>● water supply</li> <li>● hospitals</li> <li>● theatres</li> <li>● skating rinks</li> <li>● soccer stadiums</li> <li>● transport infrastructure</li> <li>● employment</li> <li>● housing</li> </ul> | <ul style="list-style-type: none"> <li>● monuments (cultural)</li> <li>● facilities for events</li> <li>● oil and gas supplies</li> <li>● connections abroad</li> <li>● spatial distribution of functions</li> <li>● environmental diversity</li> </ul> |
| <b>Undesired</b> | <ul style="list-style-type: none"> <li>● neighbour-hood noise</li> <li>● radon</li> <li>● draughts</li> <li>● damp house</li> <li>● leaden water piping</li> <li>● vermin</li> </ul> | <ul style="list-style-type: none"> <li>● dangerous traffic points</li> <li>● noise annoyance</li> <li>● dirty streets</li> <li>● dog mess</li> <li>● unsafe corners</li> <li>● city road air pollution</li> <li>● polluted soil</li> <li>● untended houses</li> </ul>   | <ul style="list-style-type: none"> <li>● flood risks</li> <li>● aircraft noise</li> <li>● visual pollution</li> <li>● air pollution</li> <li>● traffic jams</li> </ul>   | <ul style="list-style-type: none"> <li>● airborne particulates</li> <li>● ultraviolet radiation</li> <li>● climate change risks</li> </ul>  |

Source: Health Council of the Netherlands, 1999 (Annex D).

knowledge (radios and television sets). Sustainable development by this reckoning focuses on the enhancement of human life. Economic investment should be measured through its social and human product as well as economic development.

From the perspective of environmental pollution, the health risks of economic development are longer-term (McMichael and Haines, 1997). Climate change will probably impact earlier on poorer than on industrialised countries. Changes in weather patterns in Africa and Asia may lead to changing disease patterns and to population migrations. The direct effects of climate change on the industrialised countries may be less dramatic. In Britain, for example, the rise in sea level will mean that the Thames Barrier, currently needing to be closed at peak tides three to five times each year, may by the end of the twenty-first century need to be closed 200 times a year (The Environment Agency, 1999). More than half the annual budget of the Environment Agency is for flood defences. But there will also be indirect effects via global trade and population migration.

Global warming is caused by greenhouse gases from fossil fuels. This will be the greatest challenge to health from pollution in the twenty-first century (WHO, 1996). The prospect can be seen in the current rapid rise in use of cars in developing countries. Can China, with more than 20 per cent of the world's population, have in the future as many cars per capita as Europe? The West cannot say 'no' at present on grounds of equity. Instead, we need calculations on sustainable, equitable levels of car use for the whole planet. If we are already above the carrying capacity for the world, stabilisation at current levels is a less than adequate target. Transport policies that take account of the global impact of pollution as well as the local effects affirm the importance of travel by walking, cycling, buses and trains rather than by car.

## **Conclusion**

Environmental protection has reduced some of the burden of disease produced by industrialisation. Cities have cleaner air and water, but waste and noise are of increasing concern. Cities are not homoge-

nous, and there is a research agenda to explore the relative health impacts of local physical environments and the effects of personal socioeconomic characteristics. Transport will be a key area for health policy debate in the twenty-first century, as current patterns in industrialised countries are both health damaging and environmentally unsustainable.

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## Chapter 6

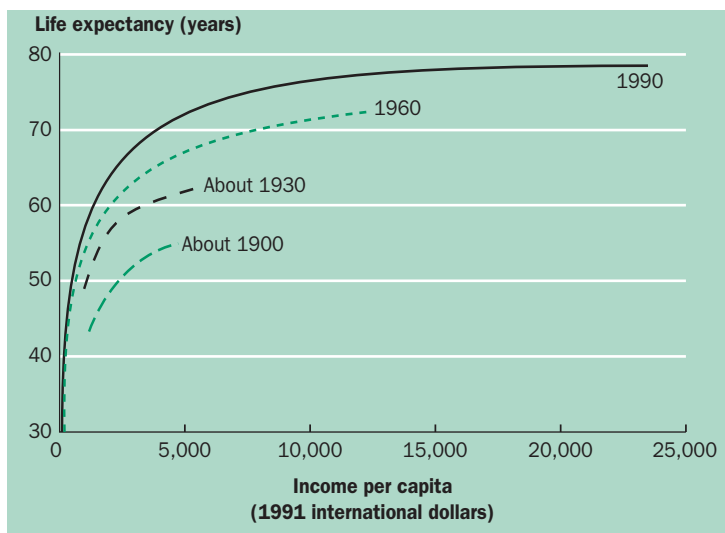
# The socioeconomic, psychosocial and developmental environment

CLYDE HERTZMAN

### Health differences among wealthy countries

What makes some wealthy countries healthier than others? Figure 6.1 plots level of income per capita against life expectancy in different countries in 1900, 1930, 1960 and 1990. By 1990 a substantial flattening of the health-wealth curve had occurred, beyond about

Figure 6.1 Life expectancy and income per capita for selected countries and periods



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.

Source: World Bank, 1993, Figure 1.9.

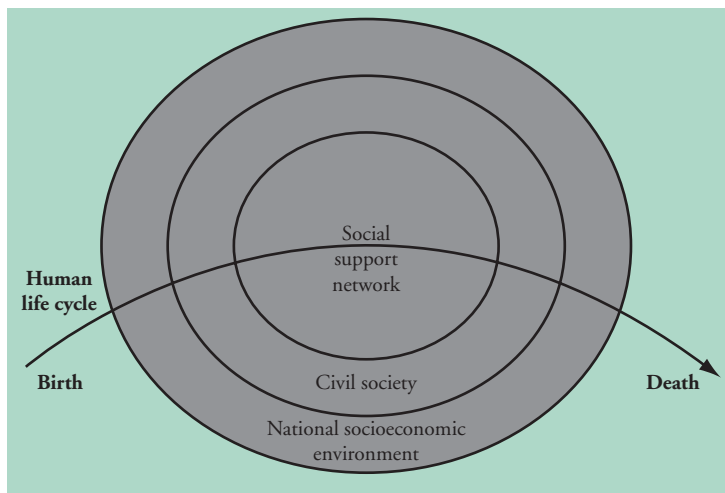
US\$ 11,000 or US\$ 12,000 per capita income. There are about 30 countries on the flat of the curve. In other words, they are in a range where differences in gross domestic product (GDP) per person between those countries no longer seem to correlate very strongly with differences in life expectancy among them. Yet there are relatively healthy and relatively unhealthy countries within that range, with a life expectancy difference of about six or seven years between them, which converts into about a twofold difference in mortality in the age range between 25 and 70. The difference in health status between the wealthy countries is therefore of some interest.

We know some things that do not seem to be associated with these differences. One is that it does not appear that the level of expenditure on health care is associated with those differences. After all, the US is one of the least healthy of the wealthy countries despite spending 15 per cent of its GDP on health care and having some of the most hi-tech health care in the world. Japan, on the other hand, is one of the healthiest of the countries – at least, as far as life expectancy goes – although it spends only about 6 per cent of its GDP on health care.

Nor is there any evidence that the quality of health care that is delivered is associated with differences among rich countries in health status. Concerning this linkage, there is an absence of evidence, rather than evidence of an absence. The onus, however, is on those who believe that health care is a significant determinant of population health to produce evidence that differences in the quality of care are important causes of differences in health status.

Understanding the differences in health status among the wealthy countries involves thinking about the determinants of health within those countries, according to a four-compartment model as illustrated in Figure 6.2. Within the overall global environment, we have at the most macro level the national socioeconomic environment. This has to do with the distribution of resources within society and the structure of opportunity. At the meso level we have the civil society, which relates to whether or not the institutions and structures of daily living that people encounter support or undermine their lives as citizens. In other words, are they stress-buffering or stress-inducing;

Figure 6.2 A model of health determinants



inclusive or exclusive; accessible or inaccessible? Within this we include everything from networks of trust, to institutional responsiveness and to psychosocial working conditions. At the most micro, intimate, level we have the social support network. This can be represented by something as simple as being married, which consistently comes out as a predictor of differential health status. Everyone's life course transects those three different levels of aggregation of society. I propose that the differences in health status that we see between the wealthy countries of the world are most likely due to the differential qualities of interactions between the individual life course and the conditions that people encounter at the national socioeconomic, civil and intimate levels.

### The 'gradient'

The agency of the translation of the socioeconomic, the psychosocial and the developmental environment into health status is referred to as the 'gradient'. There are some conditions in society which do not show a socioeconomic gradient, or indeed show an inverse gradient.

But the fact is that heart disease, stroke, most cancers, most diseases which have a behavioural component, rheumatic and arthritic diseases, all show a socioeconomic gradient, going from lowest incidence among the most privileged in society to the highest incidence amongst the least privileged. Moreover, the gradient has shown an ability to reproduce itself for new conditions as they arise over time. Thus the gradient at the turn of the century primarily related to infectious diseases. Heart disease came into society originally as an affliction of those who were privileged enough to live long enough to get heart disease but, gradually, by the 1950s the gradient established itself in the usual direction. In the last 20 years we have seen the gradient establishing itself for HIV-related conditions too.

In understanding the gradient, therefore, it does not do to break everything down disease by disease and come up with a series of disease-specific explanations. In order to understand the gradient we have to understand something that is deeper than individual conditions: something that has to do with the production or undermining of vulnerability or resilience at a broader biological level. Understanding the way in which the socioeconomic, the psychosocial and the developmental environments shape health status means looking at human biology in a new way.

A number of years ago Wilkinson got the discussion going on the factors associated with differences in health status by looking at the equity of income distribution (see Wilkinson, 1996). He suggested that those wealthy countries that had a more equitable income distribution tended on average to be healthier than those that had a less equitable income distribution. As a result of challenges to this hypothesis, based on the incomparability of data internationally, Kaplan and colleagues (1996) looked within the 50 US states, so that he could control for a number of the variables that were problematic at the international level. Kaplan et al. looked at the median share of income, in other words the proportion of each state's total income going to the poorest 50 per cent of the population. They found that as the median share of income rose across states, so mortality dropped quite considerably. See Figure 6.3. The association was quite similar to the one generated by Wilkinson at the international level (Wilkinson, 1996).

Figure 6.3 All-age mortality rates by median income share, all rates standardised to the Canadian population in 1991



Sources: Kaplan et al., 1996; Ross et al., 2000.

Ross et al. (2000) then extended Kaplan et al.'s analysis by adding the 10 Canadian provinces to the graph. They re-worked the data so that all of the niceties of how income was being calculated, how median share was being calculated and how family size was being weighted, were comparable between the Canadian and US data. Figure 6.3 shows the 10 Canadian provinces clustering at the bottom-right of the US states' analysis, at a relatively high level of equity of income distribution and relatively low mortality by the US states' standards. Canada spends about 9 per cent of its GDP on health care, and that GDP is, per capita, about 15 per cent below the US GDP per capita. Yet the Canadian provinces cluster amongst the healthiest of the US states: at a relatively high level of income equity and low level of mortality.

## Social trust

When you study the historical pattern of economic development in those US states that are clustered with the Canadian provinces, it is interesting to see that these states share with the Canadian provinces a heritage of smallholding. In other words, the economy developed on the basis not, primarily, of plantations organised on a vertical basis – as in the southern US states – but instead on the basis of smallholders who each had their own plot of land and had to band together in horizontal institutions in order to survive. There is a literature on why it was that the northern part of North America ended up doing better over the last 150 years than parts of the Americas, such as Brazil, that seemed like the most promising societies early in the nineteenth century (Engerman and Sokoloff, 1997). The best answer that the economic historians have come up with is that those parts of North America that developed on a base of smallholding consequently developed strong horizontal institutional structures and practices. That facilitated a higher level of income equity, with industrialisation, broader levels of civic participation generally speaking, and more rapid socioeconomic development. As you move farther south, where large plantations were economically viable, there were instead relatively few owners and bosses, and everybody else hired (or enslaved) to work for them. These are vertical structures, under which demands for education and autonomy were relatively weak, along with the ability to develop strong horizontal structures of trust and participation.

Looking internationally, we see that social trust as a variable is associated with a number of other factors that impact people's ability to cope on a day-to-day basis: the adequacy of the education system; the quality of physical infrastructure; the level of civic participation; the quality of bureaucratic functions and the efficiency of judiciaries. These all have an effect on health status too.

One of the most useful experiments of nature that helps us to understand these kinds of influences on health, is what happened in central and eastern Europe after the end of the Soviet era. The region experienced large economic shocks during this period; real wage declines of 20 per cent to over 50 per cent; evidence of social disruption;

abrupt declines in new marriage formations; and also evidence of the withdrawal of previously accessible forms of social services, such as universally accessible pre-primary school. What also happened was that one of the few genuine achievements of Soviet-style socialism – relative income equity – started to erode, but at different rates in different countries. Within some of the states of the former Soviet Union there was a very large increase in income inequality. In some of the other countries there were increases, although more modest in size.

With all these changes, there were abrupt rises in mortality in some countries, particularly in Russia and Ukraine. The patterns of mortality, including the fact that there have been similar changes for both men and women, suggest that smoking, diet and alcohol consumption were not the major factors accounting for the sudden rise in death rates. Based on survey data on self-rated health collected in Russia (Rose, 1996), there appear to be significant effects due to differences in education and material deprivation (a very large effect). The issue of coping with economic circumstances appears borderline. But in terms of civil society, there are large differences in health status between those people who were stuck, relying on the traditional institutions of Soviet society which were rapidly disappearing, versus those who were able to get on board the brave new capitalist institutions emerging within Russian society.

### **The longitudinal dimension**

As I have already pointed out, the agency of the gradient leads to a concern regarding general vulnerability and resistance to disease, rather than to disease-specific risk factors. In order to understand what might produce general vulnerability or resilience to disease, we have to look at early developmental factors and especially the relationship between child development and the development of defence mechanisms against disease. The best way to understand these factors epidemiologically is through birth cohort studies.

In Britain there are several cohort studies available. Table 6.1 shows some of Power's important findings from the 1958 British birth cohort (Power, 1995). The table shows the emergence of gradients

**Table 6.1 Gradients in health outcome, age 33, by social class at birth (1958 British birth cohort)**

|                                   | Men         | Women |
|-----------------------------------|-------------|-------|
| Fair/poor self-rated health       | 2.67        | 3.22  |
| Limiting illness                  | 1.58 (n.s.) | 2.14  |
| Respiratory symptoms ( $\geq 1$ ) | 1.8         | 2.56  |
| High malaise score                | 2.10        | 2.97  |
| Asthma                            | 1.15 (n.s.) | 1.42  |
| BMI – obese                       | 2.19        | 1.99  |

Notes: Numbers are odds ratios, based on the slope index.

n.s.=not significant.

Source: Power, 1995.

for six different health outcomes by age 33. The numbers are slope coefficients for the social class of these individuals at the time of birth. They can be interpreted as the range of risk from the lowest to the highest social class. Thus, for fair/poor self-rated health among men, risk increases by a factor of 2.67, in a monotonic fashion, from the lowest to the highest social class at birth.

These findings can be taken as some indication of a potentially latent effect. In other words, things that occur early on in life can have an effect on health status two, three, four, five decades later, potentially irrespective of intervening experience.

There is, however, also what we might want to call a pathway effect. If you look at this same population cohort and analyse the connection between their health and their educational qualifications – which takes into account the course of the first 20 years of their lives – then you see much larger slope coefficients than we saw with respect to social class at birth: at least a doubling of them (Table 6.2). In this case, the highest educational level is university graduation, and the lowest is ‘less than O-levels’ (O-levels were exam-based school qualifications usually obtained at age 16). The relative share of the population at the extremes of this distribution is similar to that for social



**Table 6.2 Gradients in health outcome, age 33, by educational qualifications (1958 British birth cohort)**

|                                   | Men         | Women |
|-----------------------------------|-------------|-------|
| Fair/poor self-rated health       | 5.58        | 6.11  |
| Limiting illness                  | 3.33        | 2.19  |
| Respiratory symptoms ( $\geq 1$ ) | 4.21        | 5.08  |
| High malaise score                | 5.17        | 9.54  |
| Asthma                            | 1.91 (n.s.) | 2.11  |
| BMI – obese                       | 2.82        | 2.38  |

*Note:* Numbers are odds ratios, based on the slope index.

n.s.=not significant.

*Source:* Power, 1995.

class at birth. Thus, men with ‘less than O-levels’ are 5.58 times more likely to be in only fair or poor (self-rated) health than those with university degrees. Further analysis shows a statistically independent pathway effect (Power, 1995). In other words, the life courses that people follow take them into life circumstances that are more or less supportive or undermining of health, and that is an effect over and above the latent effect.

Finally, by scoring the social class of individuals at ages 0, 7, 11, 16 and 23, it was possible to show that those people who were always in the lowest social class experienced much worse health than those who were always in the highest social class. You get a classic, cumulative, dose-response kind of relationship for the cumulative social class variable (wherein ‘16’ means always in the lowest social class and ‘4’ means always in the highest, with all other combinations in between), and this stands over and above the latent effect and the pathway effect (Figure 6.4). Thus, overall, this kind of cohort data reveals three different types of effects on a person’s health during their lifetime: latent effects, pathway effects and cumulative effects.

It is, therefore, vitally important – and I propose it as a priority for the twenty-first century – to understand the process of biological embed-

Figure 6.4 **Cumulative social class (birth–33 years) and prevalence of poor health at age 33**



Source: Power et al., 1999.

ding. That is, the process whereby systematic differences in the quality of the environments that people are exposed to during their early development can embed themselves in human biologies in ways that can create systematic differences in people's resilience/vulnerability to disease.

Early on in life many critical events, environmental experiences, are occurring in relation to the sculpting of the brain. In other words, certain synapses in the brain are reinforced and certain die away, as a result of tactile, visual, auditory, emotional and intellectual stimulation. We have come to understand over the last 15 years or so that the brain is an environmental organ, every bit as much as the lungs are environmental organs. Environments of pollution can affect the function of the lungs physiologically; the environments of stimulation in which the developing brain grows up affect the way that it is wired

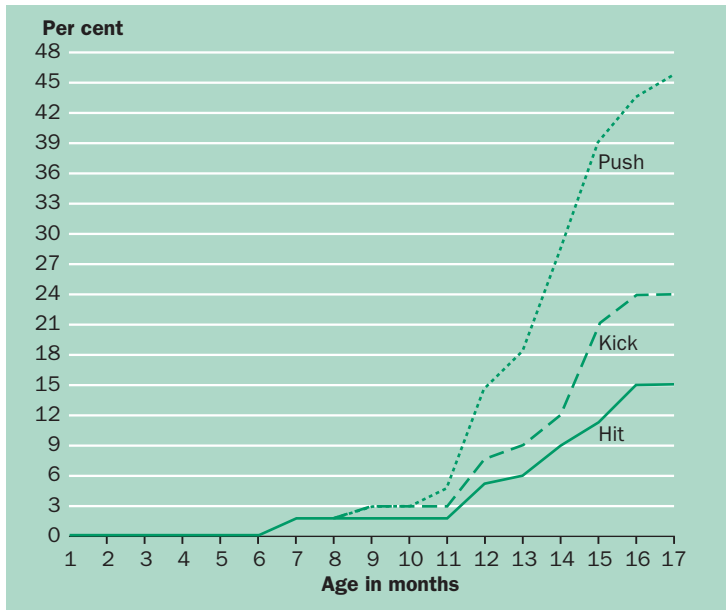
and affect its ability to function competently in the world. We are also coming to understand that those aspects of the environment that sculpt the brain for cognitive, behavioural and social functions can also affect the host defence pathways through the hypothalamic-pituitary-adrenal axis, through the sympatho-adrenal-medullary axis, and through the connections between perception, brain and the immune system. The question of appropriate brain sculpting early on in life and differential access to the conditions which will allow for appropriate brain sculpting, is therefore relevant to the development not only of well-being and competence but also of health.

One example of the issue of critical periods in brain development is understanding the problem of juvenile delinquency. People usually think of teenagers as being physically aggressive and that physical aggression begins in teenage. Even the parents of physically aggressive teenagers will usually tell you that they do not know where that behaviour came from: 'the kid was so nice when he/she was young'. However, prospective studies, following children over time, show quite clearly that physically aggressive behaviour takes off exponentially at about the end of the first year of life (Tremblay, in press – see Figure 6.5). The proportion of the population which is going to be physically aggressive in socially conflictual situations peaks at about age two and then declines dramatically, before children ever reach teenage (Tremblay, in press – see Figure 6.6). Intervention studies show that this early period of life is when interventions to moderate children's behaviour will have the greatest success. It is in that period when some children are naturally learning to start kicking, biting and hitting and other children are not, that the best chance exists to socialise these things out, as opposed to later on.

To make a very long story short, to a greater or lesser extent our societies provide differential access to children for six basic, developmental, priming environments (Ramey and Ramey, 1998):

- encouragement of exploration;
- mentoring in basic skills;
- celebration of developmental advances;

Figure 6.5 Cumulative onset of physically aggressive behaviour



Source: Tremblay, in press.

- guided rehearsal and extension of new skills;
- protection from inappropriate disapproval, teasing or punishment; and
- a rich and responsive language environment.

As a result of that, we over-produce inequality in children’s cognitive and behavioural readiness for school. There are some experiments of nature, such as the Romanian orphans’ study (Fisher et al., 1997), which show what sensory deprivation does when you do not provide children with environments of stimulation. On the other hand, there are studies like the Perry pre-school project, which show that if you take children from deprived environments and you enrich them, how much gain you can make. In this case Schweinhart and colleagues

Figure 6.6 Hitting, biting and kicking (boys and girls age 2 to 11 years)

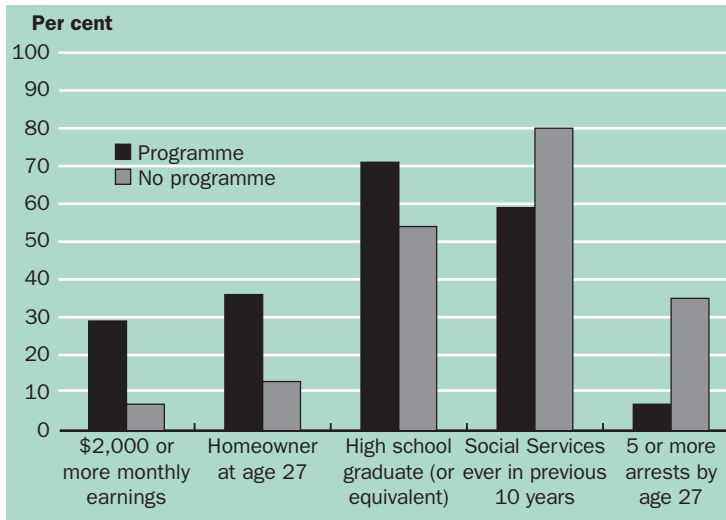


Source: Tremblay, in press.

(1993) looked at a 23-year follow-up of a year and a half enrichment programme early on in life. The results are shown in Figure 6.7. Children included in the programme as part of a randomised control trial showed greater achievements and fewer social problems than those randomised not to be included in the programme.

It is therefore not surprising, when you look at data like this, that our old friend, the socioeconomic gradient, is there in spades by the time that children reach kindergarten age, looking at measures of cognitive and behavioural development. Figure 6.8 shows data from the national longitudinal study of children in Canada (Ross and Roberts, 1999). It shows a three and a half to fourfold gradient in the proportion of children who have delayed vocabulary development in preschool, comparing the lowest household income group with the highest. This is associated with maternal education, family functioning, income per se; on the neighbourhood level, it is associated with

Figure 6.7 Perry pre-school project – major findings at age 27



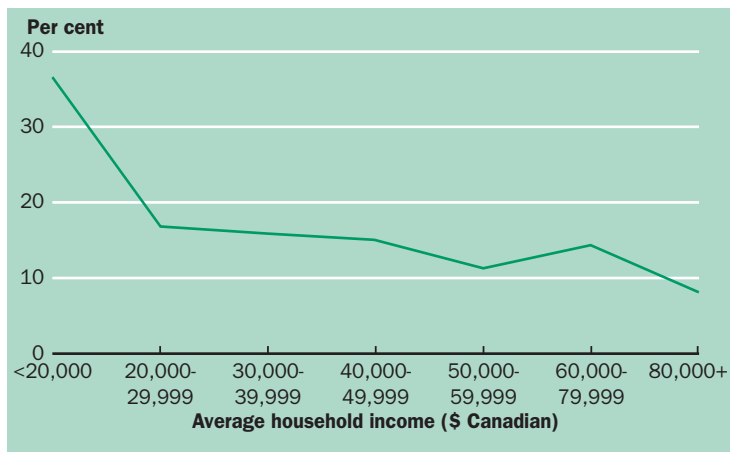
Note: All findings are significant at  $p < .05$ , two-tailed.

Source: Schweinhart et al., 1993.

neighbourhood safety, cohesion and demographic ghettoisation; and, at a broader level of social aggregation, it is associated with residential transience and access to quality child care.

The socioeconomic gradient continues throughout childhood, and thereafter. Figure 6.9 presents an international comparison of numeracy skills for young adults, according to their parents' level of education (OECD and Statistics Canada, 1995). In this case a 'level 4' score means a sophisticated use of arithmetic skills in problem skills; a 'level 1' score means bare arithmetic literacy; and 'level 3' is the level at which an individual is competent to handle the arithmetic of modern life. We see steeper gradients for certain countries like New Zealand and Great Britain, with quite low mean achievement levels. For countries like Canada and the US there are moderate gradients with intermediate levels of achievement. For some countries, like Sweden, there are relatively flat gradients, with much higher levels of mean achieve-

Figure 6.8 **Children with delayed vocabulary development in pre-school**



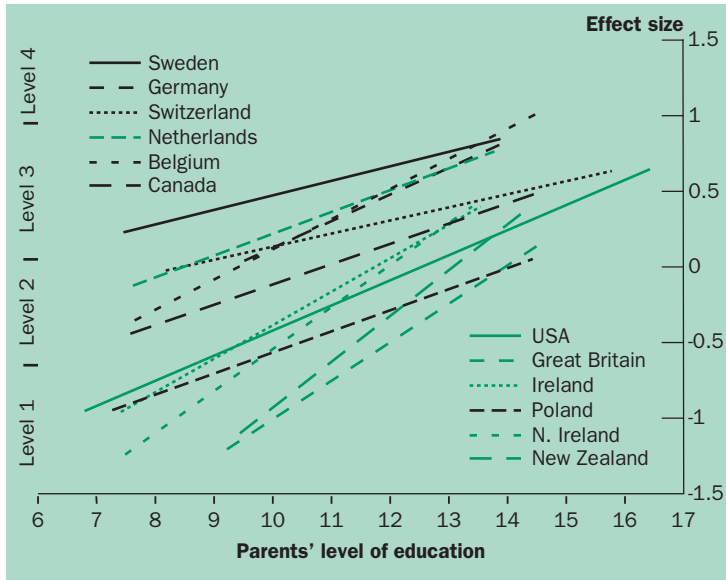
Source: Ross and Roberts, 1999.

ment. What we see in this case are gradients which seem to flatten up, in the sense that the countries with the flattest gradients seem to have the highest means. We also see a ‘fan closed’ pattern, i.e. as you move up to the more privileged end of the spectrum the curves converge. The most privileged people in society seem to be able to come up with similar outcomes whatever country they are in, but as you go down the socioeconomic spectrum the differential level of vulnerability produces larger and larger differences in outcome.

## Conclusion

Thus, I would propose that the psychosocial, socioeconomic and developmental determinants of health are all of a piece. We can use developmental trajectories as a kind of a surrogate for health futures. Embarking on national efforts to look at early child development, cognitive, social and behavioural readiness to learn, and using these as a proxy for the quality of social arrangements we have for people

Figure 6.9 Numeracy scores for young adults aged 16-25. International adult literacy study, 1994



Source: OECD and Statistics Canada, 1995.

in our society, is a good place to begin. Starting from there will enable us to understand how to make health improvements within each society and how to reduce the differences in health status that exist between wealthy countries.

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## Chapter 7

# Conclusions: how the relative importance of population health determinants may change

MICHAEL MARMOT

### Introduction

McKeown was clearly a good scientist. He was perhaps not a great scientist in the sense that Darwin was, because all scientists are Darwinians now and the argument seems to be amongst Darwinians about particular aspects of Darwinian theory, not about whether Darwin was right. McKeown, although not of the Darwin ilk, was nevertheless a good scientist because he set up a target and he got a lot of people busy aiming at that target. The fact that he may not have got it all exactly right does not mean that he did not do a very good job of stimulating a lot of interesting and important work. We can be grateful to him.

### The importance of context

The effort to distinguish how much of the improvement in population health during the twentieth century was due to changes in individual behaviour, which McKeown said was the most important factor, how much was due to social forces and how much was due to medical care, clearly depends on circumstances. It is a little like the gene/environment debate. I have had arguments with colleagues who say when you compare monozygotic and dizygotic twins, obesity is shown to be 80 per cent heritable. We saw figures today showing that the prevalence of obesity has doubled in England and Wales in the last 10 years. So, we must have changed the gene pool in the last 10 years, because obesity is 80 per cent heritable! If you go into a middle-class community in Bombay or Delhi, you will see many people with a lot of central adiposity – i.e. a lot of fat, middle-class Indians. If you go into an Indian village you see no central adiposity. Hence, obesity is clearly 100 per cent environmental. So it is both 80 per cent heritable and 100 per cent environmental. We need to keep

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that in mind when we are saying how much of the gain in life expectancy is due to personal behaviour, how much to medical care, and so on.

The relative sizes of the impacts of the different determinants are also likely to vary with the context. The impact of new medicines when or where no others exist is quite different from when and where they do. Furthermore, one might guess that medical care would make a bigger contribution to reducing inequalities in health in the US, where there are probably bigger inequities in provision of and access to medical care, than it would make in the Netherlands or the UK.

### How much room for further improvement?

The second point I want to make about the McKeown analysis relates to the question: how far can we go? McKeown showed that there was only a very modest improvement in the mortality rate of the 45 to 64 age group in England and Wales over the long period from 1838 to 1971 (see Figure 1.3 in Chapter 1 above). But this was then followed by a big reduction in this age group's mortality rate during the much shorter period from 1971 to 1997. We might have thought, up until the early 1970s, that we had gone about as far as we could go; that the mortality rate for 45-64 year olds was not going to get significantly better. But then, suddenly, there is a big improvement. So, whatever our current level of mortality rates are, we should not assume that there is no further room for improvement. The same lesson can be drawn by comparing changes in life expectancy in the UK and Japan. We saw from Jon Sussex and Peter Yuen in Chapter 1 that life expectancy for men in the mid-1990s was 74 in England and Wales. In Japan it is 77. Back in the 1960s, life expectancy in Japan for men was about 64 compared with 67 in England and Wales. Japan, in 30 years from the mid-1960s to the mid-1990s, has thus seen life expectancy for men increase by 13 years, from 64 to 77. It is a striking improvement and emphasises that we can never be sure how far we can go.

### Social cohesion

The other general point to make is that the two decades in the twentieth century that have seen the biggest improvement in life expectancy in England and Wales (other than for combatants) were 1910-1919, which embraced the First World War, and 1940-1949, which includes nearly the whole of the Second World War. People have certainly pointed to the second period as a time of improved nutrition, when inequalities in nutrition were drastically reduced because of rationing and the realisation that we could not afford 'luxury food' but that we did need to feed the whole population. Some people have suggested that the improvement in life expectancy was due to improved nutrition and equalisation of nutritional standards. However, Sen speculates, and it is a speculation with which I am very sympathetic, that the improvement could be due to improved social cohesion; that in the decades that embraced the two world wars social cohesion may well have increased (Sen, 1999).

### An artificial distinction?

However, the question of whether it is social conditions or nutrition or medical care that have produced most of the fall in mortality may be trying to make an artificial distinction. Improved social conditions may be what allow better medical care to grow up and flourish. McKeown was perhaps wrong in trying to separate out social forces from personal behaviour, and trying to say which was more important.

The Black Report on inequalities in health did something of the same, in talking about artefactual explanations and selection for inequalities (Black et al., 1980). It highlighted social conditions and rather dismissed lifestyle and behaviour. However, lifestyle and behaviour are determined by social conditions.

We may have once thought that smoking was simply an individual behaviour that was somehow divorced from the environment in which people found themselves. But I would hope that we no longer think about framing health policy as if smoking is just an individually

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chosen behaviour that comes from nowhere. My colleague, Martin Jarvis, used General Household Survey data classifying the degree of deprivation of area of residence, and showed a straight-line relationship between degree of deprivation and smoking levels (Jarvis, 1997). In the most deprived areas up to 80 per cent of men and women were smoking.

With respect to alcohol consumption, the 'wetness' of the drinking culture affects individual behaviour. There is a relationship between the prevalence of heavy drinking and the mean level of consumption.

We can make it easier or harder for people to take exercise. We can create the conditions where it is easier for people to build exercise into their daily lives. You do not just say, 'Don't be a couch potato. Take exercise'. If cycling and walking are acceptable modes of transport, it is then easy for people to exercise. Mark McCarthy also made the point, in Chapter 5 above, that transport can promote or undermine social interaction.

I could cite many other examples, but the general point is that people's behaviour is influenced by social forces. Clyde Hertzman has shown that social forces do not act only through individual behaviours, however. There are other mechanisms by which they can act.

### **Priorities and economics**

Economists want to prioritise. Shall we spend money on this or on that? The problem is that we do not have estimates of the relative costs and benefits of many of the different ways of improving population health. Where we do have estimates, they may be subject to very wide confidence limits. I would argue that a policy option should not simply be ignored if there is no estimate of the relative costs and benefits. In that case, is it appropriate to try and prioritise? Even if we did have cost and benefit estimates for all options, it could be politically very difficult to reject the apparently less cost-effective ones. For example, if there had just been a fatal railway accident, no politician would be willing to tell the public that there would be no additional investments in rail safety improvements, regardless of

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whether economic analysis were to suggest that more lives could be saved by investing the same money elsewhere. This is not an argument not to know the costs of things and not to know the potential benefits, but it is an argument not to delude ourselves that knowing those things will provide the answer for us.

### The twenty-first century

If we ask what is going to affect population health in developed countries in the twenty-first century, then clearly the social gradient will be a major influence. The socioeconomic gradient shows no sign of abating and indeed in many Western countries the gradient has become steeper in recent years. The health gap between the most advantaged and the least advantaged has become wider. The challenge to policy is that it is not simply an issue of the existence of deprivation and ill health in parts of the population, but that the presence of a social gradient affects health however rich the country is on average. The social gradient means that people in the middle of the hierarchy have worse health than people at the top, and people at the bottom have worse health than people in the middle.

This leads to consideration of psychosocial factors. As we solve a large proportion of our material environmental problems, e.g. by no longer, by and large, having people dying because of poor housing, increasingly our problems will be psychosocial ones. How do we organise our lives, and the conditions in which people live and work, to promote well-being? Do we yet have the knowledge and understanding of psychosocial factors and what to do about them?

My final point relates to the periods of life. Clyde Hertzman (Chapter 6) rightly made a call to action regarding the importance of early childhood and its role in promoting health and well-being in later life. However, I want to finish on a note about the ageing population. We all know about the increase in the elderly population in both the developing and the developed countries. We are working fewer hours during our working life. If you look at exit from the labour force in Britain, as in most other European countries, it is becoming earlier and earlier. By age 60 in Britain, fewer than half of men are still in

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the labour force. There are a variety of ways in which people get out of the labour force – disability, premature retirement, and so on – but we are working fewer hours, we are working for fewer years, and we are living longer. The result is a great increase in the amount of so-called ‘discretionary time’ available.

Isolated older people with no role in life have worse health. Do we want a society where we have a lot of people with disabilities who are dependent? Given how much of our total adult life is ‘discretionary time’ outside paid employment, and how this is likely to increase during the twenty-first century, we should be trying to harness that time. We need the imagination to think about how we could improve health and well-being for the elderly, and at the same time try to solve some of the other problems in society, by putting these two issues together.

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