The uses of 'Uses of Epidemiology'

George Davey Smith

In 1944 the Association for Education in Citizenship published a pamphlet entitled '*Health*' by Major Jerry Morris[1] (of the Royal Army Medical Corps), as one of a series of *Handbooks for Discussion Groups*. The preface to the pamphlets in this series said that they were intended to be used by discussion circles concerned with the social, economic and political problems that arose from the Second World War. The central problem the pamphlet dealt with was that of health as a social function: statistics on the current state of ill-health in Britain were presented (against the background of considerable improvement from the mid-19th century), the problem of socioeconomic differentials in health outlined and arguments in favour of a universal tax-funded health service presented. By way of a conclusion readers were exhorted to involve themselves in understanding—and through this improving—the health of the communities in which they lived (Box 1).

In 1948—and now as a civilian—Jerry Morris became Director of the Medical Research Council Social Medicine Research Unit.[2] This unit contributed importantly to the development of the methodology and practice of the epidemiology of chronic disease and in 1955 the Director published an article entitled '*Uses of Epidemiology*' in the *British Medical Journal*.[3] This article was expanded into a book[4] which, on publication in 1957, became one of the pioneering texts of 20th century epidemiology. Major Greenwood's 1935 *Epidemics and Crowd Diseases*[5] had discussed occupational disease, psychological influences on morbidity, the importance of nutrition and the epidemiology of cancer, but it was largely concerned with infectious disease, as was the other pioneering text of 1957, *Principles of Epidemiology* by Taylor and Knowelden.[6] What was new about Jerry Morris's book was illustrated in its characteristically modest preface,

which pointed out that a more accurate title would be 'Some uses of epidemiology in the study of non-communicable disease'.

The book appeared at a time when epidemiology was undergoing a fundamental change. While exemplars of non-communicable disease epidemiology could, of course, be cited—the work of Goldberger and Sydenstricker on pellagra is the classic example—no systematic approach to the population aspects of non-communicable disease existed at the end of the Second World War.[7] *Uses of Epidemiology*, therefore, helped create the field that it documented.

The area of concern of *Uses of Epidemiology* was similar to that of the *Handbook for Discussion Groups*, although the presentation was more attuned to an academic audience. There were seven uses of epidemiology in the 1955 *British Medical Journal* paper and there remained seven uses in the third edition of the book in 1975[8] (although the order of the fifth and sixth uses switched between the second[9] and third[8] editions). Box 2 reproduces the 'recapitulation' from the first edition,[4] and summarizes the way these uses were conceptualized. Epidemiology was seen as contributing to understanding the burden of disease in the community, changes in this over time (and perhaps projections of future burdens of disease), the characteristics of the health problems involved (their cause, their course, their nature and their response—or non-response—to health care) and the implications of this understanding for the health prospects of individuals.

Uses of Epidemiology contained a wealth of prescient ideas, which became a cornerstone for epidemiology as it developed over the second half of the 20th century. The book was certainly well received, as the quotes from the reviews in several journals (including *Nature* and the *British Medical Journal*) on the paper

---

Box 1

TO THE READER

Investigation

The best way to make a further study of these subjects is to find out all you can about health conditions and health services in your own community. How many doctors and dentists? How are they distributed? How many clinics, how many hospitals? What is it like to be an out-patient? How many factories have medical officers? How many children are immunized against diphtheria or smallpox? How much of the milk is pasteurized or tuberculin tested? What does your council spend on health services? What has it done with its permissive powers? How much smoke in the air? How much overcrowding? How many parks, swimming pools, playing fields? How much does it cost and how long does it take to get into the country for a day's outing? How many factories and shops give holidays with pay? What are the local death-rates—infant mortality, tuberculosis in young persons, diphtheria, etc? In all these respects how does your community compare with neighbouring districts, with the whole country, with the best area? Why are there such differences?

Taken from Morris JN, *Health.*[1]

Department of Social Medicine, University of Bristol, Canynge Hall, Whiteladies Road, Bristol BS8 2PR, UK. E-mail: zetkin@bristol.ac.uk

---

Box 2

RECAPITULATION; GENERAL

Epidemiology is the only way of asking some questions in medicine, one way of asking others (and no way at all to ask many). Seven 'uses' of epidemiology have been described:

1. In *historical study* of the health of the community and of the rise and fall of diseases in the population; useful 'projections' into the future may also be possible.
2. For *community diagnosis* of the presence, nature and distribution of health and disease among the population, and the dimensions of these in incidence, prevalence, and mortality; taking into account that society is changing and health problems are changing.
3. To study the *workings of health services*. This begins with the determination of needs and resources, proceeds to analysis of services in action and, finally, attempts to appraise. Such studies can be comparative between various populations.
4. To estimate, from the common experience, *the individual's chances* and risks of disease.
5. To *help complete the clinical picture* by including all types of cases in proportion; by relating clinical disease to the subclinical; by observing secular changes in the character of disease, and its picture in other countries.
6. In *identifying syndromes* from the distribution of clinical phenomena among sections of the population.
7. In the *search for causes* of health and disease, starting with the discovery of groups with high and low rates, studying these differences in relation to differences in ways of living; and, where possible, testing these notions in the actual practice among populations.

These various uses, it may be said, all stem from the fact that in epidemiology the group is studied and not merely particular individuals or cases in the group. The definition of groups involves accounting for all members; and this has immediate uses in the study of the natural history of disease. Describing group experience of health, disease and their circumstances is useful in itself, and it permits manifold comparisons in time, place and society.

Epidemiology is today the cinderella of the medical sciences. Nevertheless, there have been advances during recent years in the study of lung and other cancers, dental caries, pneumoconiosis, of atherosclerosis, ischaemic heart disease, hypertension, of rheumatism, schizophrenia, the congenital malformations—to mention some examples. New ground is being broken in the investigation of health, in the determination of physiological norms, in studies of morbidity, in family studies, in application to genetics, in the study of psychological aspects. There have been improvements in techniques of sampling and surveys, diagnostic and screening devices, methods of prediction, in the estimation of observer validity and reliability, the treatment of qualitative data. The prospective study of cohorts, the combination of survey with case studies, international comparisons and field experiments are being increasingly used. The proposition might be advanced that Public Health needs more epidemiology; so does medicine in general; and, it may be said, society at large.

Public Health needs more epidemiology—this cannot be doubted since epidemiology is the most likely basis for its further intellectual growth.

Medicine as a whole needs more epidemiology because it is a social science as well as human biology and the epidemiological is the main method of studying the social aspects of health and disease. Moreover, epidemiology is rich with suggestions for clinical and laboratory research and it offers many possibilities for testing hypotheses emerging from these. The main relations of epidemiology with *clinical medicine* may be restated thus:

Epidemiology is the study of populations and all cases that can be defined in them. These cases will often include, and in their due proportion, cases differing in type from those presenting to particular clinical attention (early disease, minor, the symptomless cases, the somehow peculiar). The epidemiological method can also be used to identify subclinical manifestations and again in proper proportion to the clinical. Epidemiology thus *helps to complete the clinical picture* and natural history of disease.

Epidemiology *supplements the clinical picture* by asking questions that cannot be asked in clinical medicine about the health of the community and of sections of it, present and past: it provides a different view of the world of medicine. Clinical problems are set in community perspective; health problems are revealed and indication may be given where among the population they might best be studied. Measurements can be made of the need for clinical services and how the needs are being met, thus providing an indicator of the quality of medical care.

Finally, epidemiology by identifying harmful ways of living, and by pointing the road to healthier ways, *helps to abolish the clinical picture*. One of the most urgent *social* needs of the day is to identify rules of healthy living that might reduce the burden of the metabolic, malignant and 'degenerative' diseases which are so characteristic a feature of our society. This is the main field today for the use of epidemiology.

Taken from Morris JN, *Uses of Epidemiology.*[4]

---

cover of the second edition[9] attest. It was said that if read by clinicians the book would provide a new and fresh outlook on clinical problems, that it was one of the most significant contributions to the progress of preventive medicine in recent years, approached the stature of a minor classic, made exciting reading for the epidemiologist or any medical graduate and was a gold mine to the post-graduate research worker looking for a subject or a cause.

Many ideas in *Uses* are simply included *en passant,* in a manner that suggests the author had considerable respect and confidence in his readers' ability to make connections that were perhaps less than self-evident. Discussion of many of these ideas has been expanded greatly length-wise by others subsequently, although perhaps the profundity of the ideas has sometimes not increased in the process. While depth of thinking in epidemiology may not have increased greatly since the *Uses* first appeared, the nature of epidemiology textbooks has generally been transformed. The concern of most recent books is almost exclusively methodological[10–14]—the health of populations has become a footnote to a detailed exposition of how to calculate a multivariably adjusted effect estimate from a study with appropriate sampling, and then how to apply a billiard-ball view of causation to your study results. In 1980 Reuel Stallones had already noticed that recent work in epidemiology demonstrated a 'continuing concern for methods, and especially the dissection of risk assessment, that would do credit to a Talmudic scholar and that threatens at times to bury all that is good and beautiful in epidemiology under an avalanche of mathematical trivia and neologisms',[15] a view Jerry Morris clearly shares.[16]

Working of health services

In some ways the most revolutionary part of *Uses* was the chapter on 'Working of health services'. Stimulated by what he was able to observe of 'operational research' during the war, this chapter viewed medical care as a legitimate focus for epidemiology. Research in this tradition existed before the mid-century—for example in 1930 RA Bolt published a study relating public health expenditure to infant mortality rates across cities in the US.[17] However, Thomas McCarthy and Kerr White have recently suggested that a 1952 conference held in Chapel Hill on research requirements for health and medical care, at which Jerry Morris was the keynote speaker, represented a landmark in the arrival of health services research in the US.[18] His talk covered the principles of randomization and population-based studies, and considered the three possible locations for such research: the laboratory, the clinical encounter and the population.[18] Morris certainly had little truck with the view that health services could necessarily only have a minimal impact on population health;[19] therefore health care needed to be considered in any enterprise concerned with why some people (and peoples) were healthy and some were not. In the chapter 'Working of health services' in the first edition of *Uses* there were many examples of what would now be considered to be 'health services research'.

The potential impact of medical therapies was illustrated with data on the changing social class distribution of diabetes deaths at age 20–34 years, when most cases would be type 1 (or insulin-dependent) diabetes (Table 1). In the early 1920s mortality was higher in social classes I and II than in social classes IV and V. Over the subsequent decade, mortality rates fell in all social classes, but to a much greater extent for social classes I and II than for social classes IV and V, leading to a cross-over in social class patterning of diabetes mortality. The suggestion here is that the more privileged social groups benefit at an earlier stage from the introduction of insulin, and that while insulin had a dramatic effect on diabetes mortality, some benefit much more than others.

Differences in the quality of medical care were examined through case-fatality rates (Table 2), which were considerably lower in teaching hospitals than non-teaching hospitals for several important health problems where treatment manifestly could affect outcome. While differences in characteristics of the patients (and their diseases) could account for much of this, it suggested that differences in medical care resources and procedures also produced differences in health outcomes.

Gross variation in medical practice was utilized as a way of indicating that, in at least some places, optimal care was not being delivered. Thus Morris suggested that the substantial variation in tonsillectomy rates (Table 3) indicated over-treatment in some places, which could be contributing to wasted health service expenditure.

Morris considered that it was important to quantify the need for health care in the population. Population studies—of people's needs for health care and their demands for it—were said to be required. Here we see the beginnings of research into

Table 1 Impact of a new therapy. Death rates per million from diabetes at 20–35 years of age. Males, England and Wales

Social class	1921–1923	1930–1932
I and II	64	26
III	50	25
IV and V	46	35

Source: Morris.[4]

Table 2 Case-mortality in teaching and non-teaching hospitals[a]. England and Wales, 1951

Condition	Teaching hospitals			Non-teaching hospitals		
	Cases	Deaths	%	Cases	Deaths	%
Age 45–64 years						
Coronary heart disease	755	143	19	183	52	28
Perforated peptic ulcer	379	25	7	131	13	10
Fractured skull and other head injuries	499	27	5	117	14	12
Age 65–74 years						
Hyperplasia of prostate	660	41	6	280	48	17
All ages						
Appendicitis	3478	24	0.7	1466	22	1.5

[a] Fifty per cent national sample of teaching hospitals; 3% sample of non-teaching hospitals. Males.
Source: Morris.[4]

Table 3 'Glover' phenomenon

Tonsillectomy rates per 1000 school pupils 1936–1938 (annual averages)			
Manchester	11	Leeds	38
Bradford	12	Leicester	36
Gloucester	12	Exeter	40
Birkenhead	3	West Hartlepool	39
Isle of Ely	4	Soke of Peterborough	55
Cambridge	13	Oxford	40

Source: Morris.[4]

what Stephen Frankel has called the 'epidemiology of indications',[20] in which the important parameter is the number of people in a population who can benefit from medical treatment, rather than the number of people with a particular condition.

Health services were not seen as being outside of the remit of epidemiology, and indications, process, outcome and costs could, in Morris's view, be quantified. As he said in the third edition of *Uses*, 'myself I have an old-fashioned faith in saturating the services with *facts*'.[8] The current state of the debate regarding rationing of health services in the UK—carried out mainly in data-free fashion[21]—reflects how this optimism has failed to be realized, largely through a failure of imagination, rather than because it is impossible in principle to treat the need for health services as an empirical problem.

Prescient ideas

Woven throughout *Uses* are a myriad of examples of insightful thinking about epidemiology which have been incorporated in the later development of the discipline. Here there is room for just a few examples.

Population approach

The great potential of population-based approaches to disease prevention—as opposed to interventions targeting the relatively small number of high-risk individuals—has been given considerable emphasis in recent public health policy.[22] The importance of the population approach was elegantly summarized in *Uses*: 'The stakes are high: quite small shifts in population distributions of blood pressure or blood cholesterol to the left … could well confer substantial benefits on community health, diminish suffering and lighten the burden on services out of all proportion'.[8]

Large enough trials

Recognition of the fact that clinical trials have tended to be too small to detect plausible effects of medical treatments which, while not great, may have substantial population impact, has had considerable influence in recent years.[23] Morris recognized this, pointing out that the contribution of health services to health 'may be quite small compared with other factors, personal, environmental and unknown, a situation that requires the perfect and often still unachieved clinical trial for an answer'.[8] Morris was involved in establishing one of the first of these 'mega-trials'; the World Health Organization trial of clofibrate in the primary prevention of ischaemic heart disease, which randomized over 10 000 participants.[24] Perhaps because this trial produced an unexpected small increase in mortality in

the clofibrate treated group compared to the control group, its pioneering nature has been under-recognized.

Individual and group risks

There has been considerable recent interest in the concept that groups possess properties over and above the sum of the properties of individuals, and that these may influence disease risk.[25] Conceptualization of the group-level properties which cannot be summarized with individual-level data is being explored, and the concept of herd immunity in infectious disease epidemiology has been invoked to stress the need for 'population systems epidemiology' in non-infectious disease situations.[26] Morris discussed this with respect to mining accidents (Figure 1) in the first edition of *Uses*.[4] 'This figure makes a point which has scarcely been mentioned before: that groups possess properties as groups which (like herd immunity to infectious disease) are not merely the sum of the properties of the individuals who constitute the groups. Up to now in the present exposition we have been dealing with what are technically known as 'aggregates' having little or no systematic interaction among the individuals in them, and often defined specially for the purposes of the study. But this figure postulates a function of the group as a whole, in this instance psychological morale.' (Durkheim was, however, not referenced until the second edition.) In related fashion Morris considered that we needed an 'ecological view' if we were to understand that the 'chronic diseases are products of the interaction between people and their place in the world, of causes jointly in these'.[8] This view is reflected in the recent models which have been advanced for epidemiology, such as the eco-epidemiology of Susser and Susser[27] and the ecosocial framework proposed by Nancy Krieger.[28]

In search of causes

While the seven functions of epidemiology were treated comprehensively in *Uses*, the key issue in epidemiology was seen to be the uncovering of causes of disease. In the first edition, the chapter on aetiology covered about a third of the book, which had increased to about a half by the third edition. Furthermore,
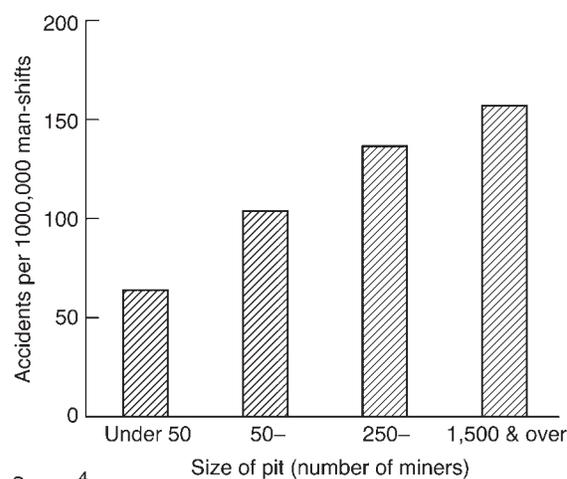

Source:[4]

Figure 1 Frequency of mining accidents in relation to size of pit—number of miners. Source: Morris.[4]

much of the material in the chapters regarding the other uses of epidemiology refers to how they can contribute to understanding the causes of disease. For example, social class differences in disease are covered in the chapter on 'community diagnosis', where it is pointed out that these differences can also provide useful clues to disease aetiology.

One concern of the *Uses of Epidemiology* that has tended to atrophy in more recent epidemiological textbooks is with the history and geography of disease. The book starts out with a lively summation of disease trends in Britain. It was particularly concerned with the increasing male-female disparity in death rates, (Figure 2) with little indication of any improvement in male death rates from the 1930s through to the 1960s, a period during which female death rates declined consistently. The important contribution of ischaemic heart disease and lung cancer to this increasing disparity was made clear. These two conditions—together with peptic ulcer—were causes of an increasing proportion of deaths from the mid-century onwards, and therefore received much attention in the book and influenced its thinking. Regarding the causes of disease, the large-scale historical changes and differences between countries were considered key indicators of whether factors were plausible aetiological agents: 'to survive, a hypothesis on aetiology must be consistent with such facts of life'.[8]

Changes in disease rates were also taken to indicate the environmental dependency of disease burden. Addressing the data on male and female mortality (Figure 2) Morris rhetorically asked: 'What are the *social* changes that underlie the *biological* changes expressed in the figure'.[4] This notion of how the social literally becomes biological should be at the heart of the epidemiological enterprise, although it remains relatively unexplored. Morris's own lifetime research on the 'modern epidemic' of coronary heart disease and physical activity of work, then exercise in leisure-time in the increasingly sedentary population, illustrates this.

Lifecourse epidemiology

While many of the problems facing epidemiology in the mid-century have been solved, some remain resolutely intractable.
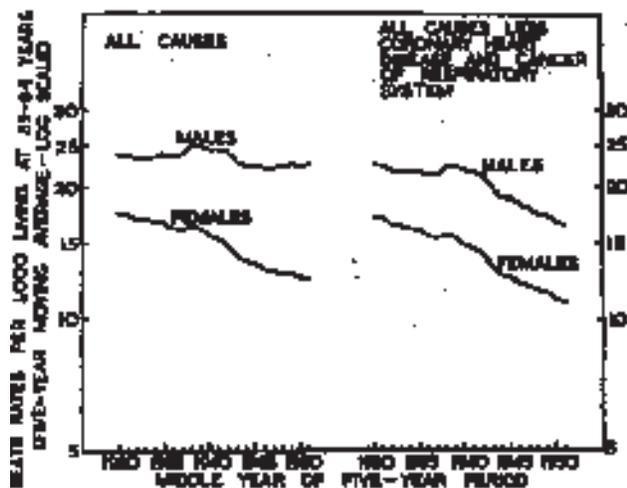

Figure 2 Mortality in middle-age, 1928–1953. The contribution of coronary heart diseases and lung cancer. England and Wales. Source: Morris.[4]

One of the striking findings reported in *Uses* related to the changing prevalence of coronary atheroma during the period when deaths from ischaemic heart disease increased dramatically. If anything, there was a decrease in the level of atheroma over this period. This led to the hypothesis that factors relating to blood clotting were of importance.[29] More recently a study of adults undergoing angiography suggested no decline in coronary atherosclerosis in the US during the period when coronary mortality declined dramatically.[30] However, looking at an early stage of life across the cohorts in the US who showed falling ischaemic heart disease mortality rates, it is possible to detect a decline in atherosclerosis. Table 4 presents data from autopsies of young men dying in the Korean war (early 1950s) and Vietnam war (late 1960s). These data have been frequently cited as demonstrating the high prevalence of atherosclerosis in early adulthood, and the importance of early intervention. It has less often been noted that there was a substantial decline in prevalence between the early 1950s and late 1960s. The data suggest that the recent decline in adult ischaemic heart disease mortality could have been influenced by changes in onset of the early stages of the disease in childhood. This notion was discussed in several places in *Uses*. For example, we are told that 'on all counts, the notion of hypertension, atherosclerosis and coronary heart disease as "paediatric problems" represents a hopeful advance';[8] that disease of later life may be laid down in childhood;[8] that 'the "physiological" failures reflected in *perinatal* mortality reflect the lifetime experience of the mother;[8] and even that there was 'the programming of adult disease in childhood'.[8] The explosion of interest in the past 15 years in the early-life origins of adult disease—whether in prenatal development[31] or childhood[32]—demonstrates how these insights have been developed. Table 5 shows how social circumstances in childhood—indexed by father's social class—specifically influence the risk of cardiovascular disease mortality in later life. These data come from students attending Jerry Morris's *alma mater*, Glasgow University. These students—attending between 1948 and 1969—will have become a privileged socioeconomic group in adulthood; less than 5% of school leavers entered university over this period of time. Therefore continuity between childhood and adulthood social circumstances is unlikely to account for the association. Furthermore, other socially patterned causes of death do not show the same association as cardiovascular disease, suggesting that lifestyle and socioeconomic factors in adulthood—which influence other causes of death in addition to cardiovascular disease—do not generate this association.[33]

Table 4 Coronary artery disease in young US war fatalities

Korean war—early 1950s
200 autopsied combatants, mean age = 22 years
77% evidence of atherosclerosis
15% clinically significant narrowing of vessel(s)
Vietnam war—late 1960s
105 autopsied combatants, mean age = 22 years
45% evidence of atherosclerosis
5% clinically significant narrowing of vessel(s)

Source: Enos *et al.*[53] and McNamara *et al.*[54]

Table 5 Age-adjusted relative risks (95% CI) of mortality

Father's social class	Cause of death				
	All causes (n = 866)	CVD[a] (n = 339)	Cancer (n = 305)	Other (n = 222)	CVD[b] (n = 339)
I	1.0	1.0	1.0	1.0	1.0
II	1.13 (0.94–1.38)	1.51 (1.08–2.11)	1.11 (0.81–1.51)	0.81 (0.56–1.16)	1.46 (1.05–2.05)
III	1.22 (1.00–1.47)	1.63 (1.17–2.27)	1.07 (0.78–1.46)	1.00 (0.70–1.42)	1.66 (1.19–2.32)
IV	1.24 (0.90–1.70)	1.85 (1.12–3.07)	1.11 (0.65–1.91)	0.81 (0.42–1.57)	1.91 (1.15–3.17)
V	1.32 (0.78–2.24)	2.36 (1.11–4.99)	0.47 (0.11–1.91)	1.34 (0.53–3.37)	2.31 (1.09–4.89)
P-value for trend	0.038	0.002	0.90	0.74	0.001

[a] Cardiovascular disease.

[b] CVD adjusted for systolic blood pressure and smoking.

All analyses adjusted for quintile of year of birth.

Source: Davey Smith *et al.*[33]

Another study from Scotland has demonstrated that different causes of death show different relative strengths of association with deprivation in childhood and adulthood.[32] Stomach cancer and stroke are strongly related to childhood social circumstances, with little contribution from adulthood social circumstances over and above this,[32] while coronary heart disease and respiratory disease demonstrate a cumulative influence of social circumstances across the lifecourse, and lung cancer and accidents and violence show a predominate influence of adulthood social circumstances. For some of these associations we have a reasonable basis for judging why the findings are as they are. For example, in this study smoking was more strongly associated with adulthood social circumstances than childhood circumstances,[34] and as smoking is the major determinant of lung cancer risk, the disease would be expected to be strongly socially patterned by adulthood social class. Conversely, stomach cancer is related to *Helicobacter pylori* infection, an infection generally acquired in childhood and related to overcrowded housing, large family size, absence of running water or an indoor toilet, and the inability to maintain adequate hygiene practices. Thus childhood social circumstances would be expected to influence the risk of stomach cancer in adulthood, as was found.

The associations seen at an individual level in prospective epidemiological studies can be considered with respect to the historical and geographical trends in disease, as advocated by Jerry Morris. It is noteworthy that stomach cancer and stroke—both diseases related to deprivation in childhood—have shown markedly declining rates over the century in Britain, in tandem with improving material circumstances, falling family size and reduced overcrowding. The risk of mortality from these diseases declines as cohorts who experienced improved conditions in their childhood become older adults. It is not surprising that the declines in stomach cancer and stroke mortality in several countries demonstrate cohort effects. Furthermore, when an indicator of socio-environmental conditions—infant mortality rate—from 70 years previously is examined in relation to mortality rates across countries, strong correlations are seen for those causes of death known to be influenced by childhood deprivation: stroke, stomach cancer and tuberculosis (Table 6).[35] This demonstrates that individual level risk associations may

Table 6 Relation of adult mortality (age 65–74 years in 1991–1993) with infant mortality at time of birth and at time of death for 27 countries

	Infant mortality 1921–1923				Infant mortality 1991–1993			
	Males		Females		Males		Females	
Pearson correlation coefficients (and *P* values)								
All causes	0.52	(0.005)	0.51	(0.007)	0.58	(0.002)	0.63	(<0.001)
Respiratory TB	0.77	(<0.001)	0.73	(<0.001)	0.40	(0.04)	0.33	(0.09)
Stomach cancer	0.83	(<0.001)	0.82	(<0.001)	0.39	(0.04)	0.44	(0.02)
Lung cancer	−0.10	(0.61)	−0.48	(0.01)	−0.02	(0.91)	−0.23	(0.24)
Coronary heart disease	−0.05	(0.81)	0.16	(0.42)	0.13	(0.53)	0.28	(0.16)
Stroke	0.66	(<0.001)	0.63	(<0.001)	0.61	(<0.001)	0.64	(<0.001)
Partial correlation coefficients[a] (and *P* values)								
All causes	0.32	(0.11)	0.28	(0.17)	0.42	(0.03)	0.50	(0.009)
Respiratory TB	0.71	(<0.001)	0.69	(<0.001)	0.01	(0.96)	−0.07	(0.72)
Stomach cancer	0.80	(<0.001)	0.77	(<0.001)	−0.08	(0.71)	0.04	(0.87)
Lung cancer	−0.10	(0.60)	−0.43	(0.03)	0.04	(0.86)	0.02	(0.92)
Coronary heart disease	−0.13	(0.52)	0.03	(0.90)	0.18	(0.39)	0.23	(0.27)
Stroke	0.51	(0.008)	0.45	(0.02)	0.42	(0.03)	0.48	(0.01)

Sex- and cause-specific correlations of adult mortality with infant mortality in one period adjusted for infant mortality in the other period.

The 27 countries in the analyses were: Australia, Austria, Belgium, Bulgaria, Canada, Chile, Czechoslovakia, Denmark, Finland, France, Greece, Hungary, Ireland, Italy, Japan, Netherlands, New Zealand, Norway, Poland, Portugal, Romania, Russian Federation, Spain, Sweden, Switzerland, UK, USA.

underlie geographical and historical differentials in disease which, as Morris suggested, is an important indication that they are likely to be of importance with respect to population health.

Multiple causality and general susceptibility

The paradigm attached to chronic disease epidemiology after the Second World War was that of what Morris called 'multiple causality' of disease.[4] He referred to the 'notion that non-specific adrenal-cortical processes, as well as processes which are specific to the particular condition, produced the clinical picture of disease'. It is noticeable how this reference from 1957 to these non-specific adrenal cortical processes has developed little over subsequent decades and is still invoked to account, for example, for social class differences in disease.[36] These general processes are sometimes taken to explain why there is increased susceptibility to disease in general amongst those in adverse social circumstances. Indeed, the demonstration that one process increases risk across a wide range of health outcomes among disadvantaged socio-demographic groups would relegate disease-specific investigations to being of secondary importance, with the key task being the identification and characterization of the underlying increased susceptibility. This was the strategy suggested by John Cassell, considered by many to be the father of social epidemiology in the US, in his influential paper from 1976, 'The contribution of the social environment to host resistance'.[37]

Data from several sources suggest that such a focus would miss the true complexity of socioeconomic differentials in health. When particular causes of ill-health and death are examined there is a considerable degree of heterogeneity in their association with socioeconomic position. Figure 3 presents data relating to cancer from the Whitehall study of London civil servants, among whom there was a marked gradient in the association between employment grade and all-cause mortality.[38] For overall cancer mortality the lower grade civil servants (clerical and manual) had a 48% higher risk than the higher grades (administrators, professionals and executives). However, for the 13 specific cancer sites examined grade-related risk varied by site. The low-grade civil servants had a greater mortality risk for seven of the cancer sites, the higher grades had a greater risk for six.[38] The greater burden of cancer morbidity and mortality amongst the lower grade civil servants is because the cancers causing most deaths tend to be those that demonstrate strong gradients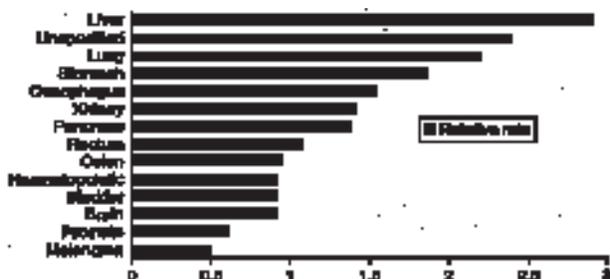 of increasing risk with less favourable socioeconomic circumstances, such as lung cancer. Similar findings with respect to the heterogeneity of site-specific cancer risk with socioeconomic position have come from other studies.[39,40]

In Table 7, data for a wider range of causes of death are presented from a mortality follow-up of a third of a million men in the US.[41] Relative risks are given for mortality associated with $10 000 lower median income of the area of residence (Zip Code areas being used for this purpose). For some causes of death—including AIDS, homicide, respiratory disease, diabetes and rheumatic heart disease—there are large differentials, with relative risks greater than 1.5 per $10 000 lower Zip Code income. The bottom decile income group had mortality rates 2–6 times higher than the rates for the highest decile income

Table 7 Relative risk (RR) of cause-specific mortality for $10 000 lower median income of area of residence (Zip Code) in US men screened in the MRFIT study

Relative risk	Cause of death
RR > 1.50	AIDS
	Diabetes
	Rheumatic heart disease
	Heart failure
	COPD[a]
	Pneumonia/influenza
	Homicide
RR 1.21–1.50	Infection
	Coronary heart disease
	Stroke
	Cirrhosis
	Genitourinary disease
	Symptoms/signs
	Accidents
	Lung cancer
	Liver cancer
	Colorectal cancer
RR 1.00–1.20	Aortic aneurysm
	Suicide
	Nervous system disease
	Oesophageal cancer
	Stomach cancer
	Pancreatic cancer
	Prostate cancer
	Bladder cancer
	Kidney cancer
	Brain cancer
	Myeloma
	Leukaemia
RR < 1.00	Blood disease
	Motor neurone disease
	Flying accidents
	Lymphoma
	Hodgkin's disease
	Melanoma
	Bone/connective tissue cancer

[a] Chronic obstructive pulmonary disease.

Source: Davey Smith et al.[41]

Figure 3 Cancer in the Whitehall Study: relative rate for low employment grade versus high employment grade.

Source: Davey Smith et al.[38]

group for these causes. For other causes of death—including such major contributors to all-cause mortality as coronary heart disease, lung cancer and stroke—the relative risks associated with $10 000 lower income were in the range 1.21–1.50. For these causes the bottom income decile had mortality rates between 60% higher and more than twice those of the top income decile. For a large number of causes of death—many of them relatively minor contributors to all-cause mortality—there were weak or reversed gradients between income and risk. For example, dying in flying accidents was markedly more likely for higher income men—presumably because those who earned more could afford to fly more. The marked heterogeneity in the strength and even direction of the associations between socio-economic position and cause-specific mortality draws attention to the need for explanatory models which account both for the overall and specific health effects of socioeconomic position, by considering how lifetime socioeconomic position structures the distribution of risk factors for a range of outcomes over time, and how this can vary by geographical location and birth cohort.

A striking phenomenon, mentioned above, is the tendency for the most important causes of death to demonstrate the most marked socioeconomic gradients. Indeed, as particular causes of death have become more important health problems over the course of this century, the tendency for them to be concentrated among the most deprived tends to become greater. Table 8 presents data on male lung cancer from 1931 to 1991. In 1931 when lung cancer caused one per cent of deaths it showed no social class gradient; by 1991 there was a marked gradient—with the mortality rate in social class V men 4.6 times that of social class I men. A similar picture is seen with respect to social class differences in coronary heart disease during the period of rapid increase in this condition as a cause of death. It reflects the ability of favourable social circumstances to allow some people to avoid identified noxious exposures. The influence of these exposures occurs against the background of less avoidable exposures (for example poor growth, health and development in childhood) to determine the overall pattern of disease. It should be remembered in this regard that even lung cancer—a disease for which a particularly important adult risk factor can be identified—may show socio-demographic differentials over and above those created by smoking.[42,43]

While 'general susceptibility' as a unitary biological phenomenon does not appear to underlie health inequalities it is certainly possible to identify social processes which lead to unfavourable exposures being concentrated on those in less privileged social circumstances, from birth to death. Human bodies in different social locations become crystallized reflections of the social experiences within which they have developed. The socially patterned nutritional, health and environmental experiences of the parents and of the individuals concerned influence birthweight, height, weight and lung function, for example, which are in turn important indicators of future health prospects. These biological aspects of bodies (and the histories of bodies) should be viewed as frozen social relations, rather than as asocial explanations of health inequalities which, once accepted, exclude the social from consideration.[44] The life-course approach to health inequalities views the physical and the social as being mutually constitutive, since aspects of bodily form can influence social trajectory in the same way as social experiences become embodied. Comprehending the ways in which the social becomes biological—and the biological in turn becomes part of the social world—must be a central aspect of an agenda aimed at improved understanding of how health inequalities arise and how they can potentially be reduced.

Peptic ulcer—travelling imaginatively?

The first edition of *Uses*[4] was much concerned with an increase in peptic ulcer in Britain,[4] and the marked international differences in the prevalence of peptic ulcer were also noted. Morris considered that this was a field that was not being exploited and that 'there may well be gold awaiting the imaginative traveller'.[4] By the mid-1950s peptic ulcer rates began to fall and by the second edition of *Uses* a mysterious decline was noted.[9] Morris discussed the work of Mervyn Susser and Zena Stein,[45–47] which identified clear birth cohort patterns in the rise (and then fall) of peptic ulcer disease in Britain. An analysis of data from 19 countries showed similar cohort patterns in all countries, with some variation between countries in when the rises and falls started.[48]

In 1967 Susser concluded that the apparent multifactorial aetiology of peptic ulcer—with contributions from diet, alcohol, cigarette smoking, emotional strain, personality and genotype did not 'exclude the possibility that a major single causal factor waits discovery'. However, much of the research carried out in the 1950s, 1960s and 1970s on peptic ulcer related to psychological factors: it was the classic stress-related disease. In the third edition of *Uses*, Morris recognized that there was no better theory for peptic ulcer than stress hypotheses at the time, but was clearly very dissatisfied with these. He pointed out that there was a true decline in incidence in the disease and that this 'would suggest to anyone in sympathy with "psychosomatic" theories … that the type of personality disposed to the disease is less common—unfortunately not a testable proposition; [or] that the environment is less of a strain—which is scarcely conceivable'.[8] In retrospect both Susser and Morris were correct, imaginative travellers demonstrated that *H. pylori* infection is a cause of peptic ulcer. The prevalence of infection is declining in a cohort-specific fashion in countries with declining peptic ulcer incidence.[49] Eradication of the infection successfully treats symptoms and promotes ulcer healing[50] and the adoption of this radical—i.e. non-palliative—treatment reduces health care expenditure.[51] The identification of *H. pylori* therefore represents a major advance in understanding and controlling an important disease. This advance was made by a pathologist and a clinician, with no input from the extensive body of epidemiological research on this important public health topic.[52]

As epidemiology enters the 21st century its traditional uses remain of considerable importance in the post-genome world.

Table 8 Lung cancer mortality 1931–1991: social class differences and contribution to total mortality among men of working age. Relative rates (with overall mortality rate among men of working age at each time point as baseline)

	Social class							% all deaths
	I	II	IIIn		IIIm	IV	V	
1931	1.07	0.96		1.01		0.91	1.12	1.0
1951	0.81	0.82		1.07		0.91	1.18	2.5
1971	0.53	0.68	0.84		1.18	1.23	1.43	11.7
1991	0.45	0.61	0.87		1.38	1.32	2.06	9.9

Source: Logan[55] and Drever and Whitehead.[56]

The importance of the style of thinking advocated by Jerry Morris is increased by the tendency of epidemiology to concentrate more and more at the individual rather than population level. Putting together individuals and their historical and social contexts still has much to offer to the imaginative traveller.
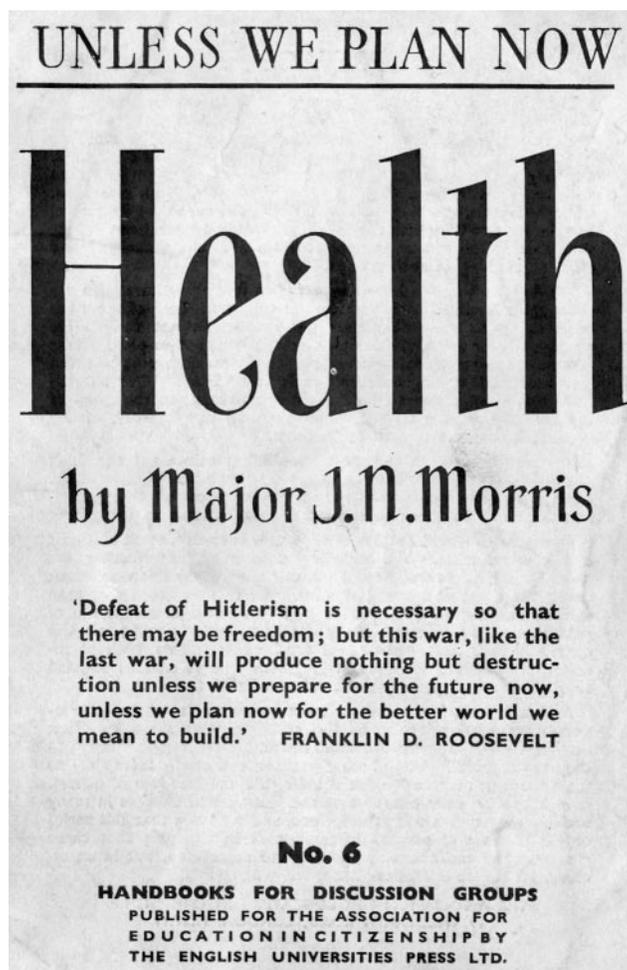
Acknowledgments

References

[1] Morris JN. *Health.* London: English Universities Press, 1944.

[2] Murphy S. The early days of the MRC Social Medicine Research Unit. *Soc Hist Med* 1999;**12:**389–406.

[3] Morris JN. Uses of epidemiology. *Br Med J* 1955;**Aug 13:**395–401.

[4] Morris JN. *Uses of Epidemiology.* Edinburgh: Livingstone, 1957.

[5] Greenwood M. *Epidemics and Crowd Diseases: An Introduction to the Study of Epidemiology.* London: Williams and Norgate Ltd, 1935.

[6] Taylor I, Knowelden J. *Principles of Epidemiology.* London: J & A Churchill, 1957.

[7] Susser M. Epidemiology in the United States after World War II: the evolution of technique. *Epidemiol Rev* 1985;**7:**147–77.

[8] Morris JN. *Uses of Epidemiology. 3rd Edn.* Edinburgh: Churchill Livingstone, 1975.

[9] Morris JN. *Uses of Epidemiology. 2nd Edn.* Edinburgh: Livingstone, 1964.

[10] Ahlbom A, Norell S. *Introduction to Modern Epidemiology.* Chestnut Hill: Epidemiology Resources Inc., 1984.

[11] Rothman KL. *Modern Epidemiology.* Boston: Little, Brown and Co., 1986.

[12] Norell SE. *Workbook of Epidemiology.* New York: Oxford University Press, 1995.

[13] Page RM, Cole GE, Timmreck TC. *Basic Epidemiological Methods and Biostatistics.* Boston: Jones and Bartlett Publishers, 1995.

[14] Szklo M, Nieto FJ. *Epidemiology: Beyond the Basics.* Gaithersburg, Maryland: Aspen Publishers, 2000.

[15] Stallones RA. To advance epidemiology. *Annu Rev Public Health* 1980;**1:**69–82.

[16] Morris JN. Modern epidemiology? *J Epidemiol Community Health* 1988;**42:**100.

[17] Bolt RA. Municipal expenditures for public health in cities of the United States of 70,000 population and over for the year 1923 in relation to their infant mortality rates. *Am J Hyg* 1930;**11:**601–18.

[18] McCarthy T, White KL. Origins of health services research. *Health Serv Res* 2000;**35:**375–87.

[19] Davey Smith G. The UK National Health Service and the national health: 1948–98. *Crit Public Health* 1999;**9:**69–74.

[20] Frankel S. The epidemiology of indications. *J Epidemiol Community Health* 1991;**45:**257–59.

[21] Frankel S, Ebrahim S, Davey Smith G. The limits to demand for health care. *Br Med J* 2000;**321:**40–44.

[22] Rose G. Sick individuals and sick populations. *Int J Epidemiol* 1985;**14:**32–38.

[23] Yusef S, Collins R, Peto R. Why do we need some large, simple randomised trials? *Stat Med* 1984;**3:**409–20.

[24] Committee of Principal Investigators. A co-operative trial in the primary prevention of ischaemic heart disease using clofibrate. *Br Heart J* 1978;**40:**1069–118.

[25] Diez-Roux AV. Multilevel analysis in public health research. *Annu Rev Public Health* 2000;**21:**171–92.

[26] Koopman JS, Lynch JW. Individual causal models and population system models in epidemiology. *Am J Public Health* 1999;**89:**1170–74.

[27] Susser M, Susser E. Choosing a future for epidemiology: II. From black box to Chinese boxes and eco-epidemiology. *Am J Public Health* 1996;**86:**674–77.

[28] Krieger N. Epidemiology and the web of causation: has anyone seen the spider? *Soc Sci Med* 1994;**39:**887–903.

[29] Meade TW, Chakrabarti R. Arterial-disease research: observation or intervention? *Lancet* 1972;**ii:**913–16.

[30] Enriquez Sarano M, Klodas R, Garratt KN, Bailey KR, Tajik AJ, Holmes DR Jr. Secular trends in coronary atherosclerosis—analysis in patients with valvular regurgitation. *N Engl J Med* 1996;**335:**316–22.

[31] Barker DJP. Mother's, babies and health in later life. London: Churchill Livingstone, 1998.

[32] Davey Smith G, Hart C, Blane D, Hole D. Adverse socioeconomic conditions in childhood and cause-specific adult mortality: prospective observational study. *Br Med J* 1998;**316:**1631–35.

[33] Davey Smith G, McCarron P, Okasha M, McEwen J. Social circumstances in childhood and cardiovascular disease mortality: prospective observational study of Glasgow University students. *J Epidemiol Community Health* 2001;**55:**340–41.

[34] Blane D, Hart CL, Davey Smith G, Gillis CR, Hole DJ, Hawthorne VM. Association of cardiovascular disease risk factors with socioeconomic position during childhood and during adulthood. *Br Med J* 1996;**313:**1434–38.

[35] Leon D, Davey Smith G. Infant mortality, stomach cancer, stroke, and coronary heart disease: ecological analysis. *Br Med J* 2000;**320:**1705–06.

[36] Brunner E. Stress and the biology of inequality. *Br Med J* 1997;**314:**1472–76.

[37] Cassell J. The contribution of the social environment to host resistance. *Am J Epidemiol* 1995;**141:**798–814 (originally published in 1976).

[38] Davey Smith G, Leon D, Shipley MJ, Rose G. Socioeconomic differentials in cancer among men. *Int J Epidemiol* 1991;**20:**339–45.

[39] Faggiano F, Partanen T, Kogevinas M, Boffetta P. Socioeconomic differences in cancer incidence and mortality. In: Kogevinas M, Pearce N, Susser M, Boffeta P (eds). *Social Inequalities in Cancer.* IARC Scientific Publications No. 138. Lyon: IARC, 1997, pp.65–176.

[40] Fernandez E, Borrell C. Cancer mortality by educational level in the city of Barcelona. *Br J Cancer* 1999;**79:**684–89.

[41] Davey Smith G, Neaton JD, Wentworth D, Stamler R, Stamler J. Socioeconomic differentials in mortality risk among men screened for the Multiple Risk Factor Intervention Trial: I. White Men. *Am J Public Health* 1996;**86:**486–96.

[42] Hart CL, Hole DJ, Gillis CR, Davey Smith G, Watt GCM, Hawthorne VW. Social class differences in lung cancer mortality: risk factor explanations using two Scottish cohort studies. *Int J Epidemiol* 2001;**30:**268–74.

[43] Davey Smith G, Shipley M, Hole D *et al.* Explaining male mortality differentials between the west of Scotland and the south of England. *J Epidemiol Community Health* 1995;**49:**541.

[44] Najman JM, Davey Smith G. The embodiment of class-related and health inequalities: Australian policies. *Aust NZ J Public Health* 2000;**24:**3–4.

[45] Susser M, Stein Z. Civilization and peptic ulcer. *Lancet* 1962;**i:**115–19.

[46] Susser M. Causes of peptic ulcer. A selective epidemiologic review. *J Chron Dis* 1967;**20:**435–56.

[47] Susser M. Period effects, generation effects and age effects in peptic ulcer mortality. *J Chron Dis* 1982;**35:**29–40.

[48] Sonnenberg A, Muller H, Pace F. Birth cohort analysis of peptic ulcer mortality in Europe. *J Chron Dis* 1985;**38:**309–17.

[49] Banatvala N, Mayo K, Megraud F, Jennings R, Deeks JJ, Feldman RA. The cohort effect and *Helicobacter pylori. J Infect Dis* 1993;**168:**219–21.

[50] Hosking SW, Ling TK, Chung SC *et al.* Duodenal ulcer healing by eradication of *Helicobacter pylori* without anti-acid treatment: randomised controlled trial. *Lancet* 1994;**343:**508–10.

[51] Murphy S. Does new technology increase or decrease health care costs? The treatment of peptic ulceration. *J Health Serv Res Policy* 1998; **3:**215–18.

[52] Thagard P. Ulcers and bacteria I: discovery and acceptance. *Stud Hist Phil Biol Biomed Sci* 1998;**29:**107–36.

[53] Enos WF, Holmes RH, Beyer J. Coronary disease among United States soldiers killed in action in Korea. *JAMA* 1953;**152:**1090–93.

[54] McNamara JJ, Molot MA, Stremple JF, Cutting RT. Coronary artery disease in combat casualties in Vietnam. *JAMA* 1971;**216:**1185–87.

[55] Logan WPD. *Cancer Mortality by Occupation and Social Class 1851–1971.* London: HMSO, 1982.

[56] Drever F, Whitehead M (eds). *Health Inequalities: Decennial Supplement.* London: The Stationery Office, 1997.

Cover of Health by JN Morris. First published by the Association for Education in Citizenship, 1944; reproduced with permission of the author.