

Traditional Epidemiology, Modern Epidemiology, and Public Health

ABSTRACT

There have been significant developments in epidemiologic methodology during the past century, including changes in basic concepts, methods of data analysis, and methods of exposure measurement. However, the rise of modern epidemiology has been a mixed blessing, and the new paradigm has major shortcomings, both in public health and in scientific terms. The changes in the paradigm have not been neutral but have rather helped change—and have reflected changes in—the way in which epidemiologists think about health and disease. The key issue has been the shift in the level of analysis from the population to the individual. Epidemiology has largely ceased to function as part of a multidisciplinary approach to understanding the causation of disease in populations and has become a set of generic methods for measuring associations of exposure and disease in individuals. This reductionist approach focuses on the individual, blames the victim, and produces interventions that can be harmful. We seem to be using more and more advanced technology to study more and more trivial issues, while the major causes of disease are ignored. Epidemiology must reintegrate itself into public health and must rediscover the population perspective. (*Am J Public Health*. 1996;86:678–683)

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Introduction

In this article I consider some of the key features of what has become known as modern epidemiology, and I contrast these with more traditional approaches. I do not intend to present a detailed historical review,^{1–3} nor to present a comprehensive review of current approaches. Rather, I will focus on some of the key changes in epidemiology over the past few decades, and I will consider the concepts of causality involved and their ideological and practical consequences. I will argue that the rise of modern epidemiology has been a mixed blessing and that the new paradigm has major shortcomings, both in public health and in scientific terms.⁴ The recent changes in epidemiologic methodology have not been neutral, but rather (in combination with other influences) they have changed—and have reflected changes in—the way in which epidemiologists think about health and disease.⁵ The key issue has been the shift in the level of analysis from the population to the individual (S. Wing, Concepts in modern epidemiology: population, risk, dose–response, and confounding, unpublished manuscript). This is typified by the current lack of interest in population factors as causes of disease, the lack of interest in the history of epidemiology, and the lack of integration with other public health activities. I will give particular emphasis to the current neglect of social, economic, cultural, historical, political, and other population factors, and I will refer to these using the general term of “socioeconomic factors.”

Of course, traditional epidemiology was not a monolith. A wide variety of approaches were used, and there is a danger of setting up caricatures of ideal types. It should also be emphasized that traditional epidemiology gave rise to

modern epidemiology; therefore, they have many features in common. Nevertheless, there are some important differences between the traditional and the modern approaches, particularly the loss of the population perspective in recent decades. I therefore will discuss some of the reasons the population perspective has been lost and the implications of this paradigm shift. Then, I will discuss some of the key issues in developing new forms of epidemiology that restore the population perspective while making use of recent methodological advances. (I am tempted to use the term “postmodern epidemiology” to provide a contrast with modern epidemiology and because some postmodernist concepts are relevant to my arguments; however, the use of this term could imply an uncritical advocacy of postmodernism, which has its own epistemological and practical shortcomings.)

Traditional and Modern Epidemiology

In the first week of their training, most epidemiologists usually learn a little about the history of public health. In anglophone countries they learn about the work of Chadwick, Engels, Snow, and others, who exposed the appalling social conditions during the industrial revolution, and the work of Farr and others, who revealed major socioeconomic differences in disease in the 19th century. At that time, epidemiology was a branch of public

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Editor's Note. See related editorial by Winkelstein (p 621) in this issue.

health and focused on the causes and prevention of disease in populations, in contrast to the clinical sciences, which were branches of medicine and focused on disease pathology and treatment of individuals (S. Wing, Concepts in modern epidemiology: population, risk, dose-response, and confounding, unpublished manuscript). Thus, the emphasis was on the prevention of disease and the health needs of the population as a whole. The dramatic decline in infectious diseases that has occurred since the mid-19th century has been attributed to improvements in nutrition, sanitation, and general living conditions,⁶ although it has been argued that specific public health interventions regarding factors such as urban congestion actually played the major role.⁷

There are still major socioeconomic differences in health, and the relative differences are continuing to increase.^{8,9} Nevertheless, modern epidemiologists rarely consider socioeconomic factors and the population perspective, except perhaps to occasionally adjust for social class in analyses of the health effects of tobacco smoke, diet, and other lifestyle factors in individuals. For example, studies in most industrialized countries have repeatedly found strong associations between social class and cancer,¹⁰ yet social class did not feature, except for a brief mention as a confounder, in the most comprehensive review of the causes of cancer in the United States,¹¹ and one leading epidemiology text states that "social class is presumably related causally to few if any diseases."¹²

Traditional epidemiology has become unfashionable and is treated somewhat disparagingly in modern epidemiology texts, which have rewritten the history of epidemiology in their own image. In particular, there has been a strong focus on statistical issues and paradigms¹³ and an ignorance of the other modes of thought that were integral to the work of 19th-century pioneers such as Snow.¹⁴ One is left with the impression that 19th-century epidemiologists used ad hoc methods that have now been placed on a sounder foundation through recent developments in methods of study design (e.g., the theory of case-control studies), data analysis (e.g., logistic regression), and exposure measurement (e.g., new molecular biology techniques). Epidemiology has become a set of generic methods for the measurement of disease occurrence, and there has been a concomitant lack of distinctive theory to permit an understand-

TABLE 1—Epidemiological Paradigms

	Traditional Epidemiology	Modern Epidemiology
Motivation	Public health	Science
Level of study	Population	Individual/organ/tissue/ cell/molecule
Context of study	Historical/cultural	Context free
Paradigms	Demography/social science	Clinical trial
Epistemological approach	Realist	Positivist
Epistemological strategy	Top down (structural)	Bottom up (reductionist)
Level of intervention	Population (upstream)	Individual (downstream)

ing of the population patterns of disease occurrence.¹⁵

These methodological developments have been paralleled by, and have reflected, a shift in the level of analysis from the population to the individual. Most modern epidemiologists still do studies in populations, but they do so in order to study decontextualized individual risk factors, rather than to study population factors in their social and historical context. McKinlay¹⁶ argued that what is now regarded as established epidemiology is characterized by biophysiological reductionism, absorption by biomedicine, a lack of real theory about disease causation, dichotomous thinking about disease (everyone is either healthy or sick), a maze of risk factors, confusion of observational associations with causality, dogmatism about which study designs are acceptable, and excessive repetition of studies. He argued that this approach diverts limited resources, blames the victim, produces a lifestyle approach to social policy, decontextualizes risk behaviors, seldom assesses the relative contribution of nonmodifiable genetic factors and modifiable social and behavioral factors, and produces interventions that can be harmful. These trends are particularly noticeable in the recent rise of molecular epidemiology,^{17,18} especially in the renewed emphasis on issues of individual susceptibility.

The Decline of Population Epidemiology

As Vandenbroucke¹⁹ noted somewhat disapprovingly, social explanations for illness won't go away and are rediscovered with each new generation of epidemiologists. However, rather than ask, as Vandenbroucke does, why the population perspective keeps resurfacing, it is perhaps more useful to ask why it keeps

sinking. Some of the reasons for the current lack of interest in the population perspective may lie in the personal and professional situations of epidemiologists. In most countries the main sources of funding are government or voluntary agencies that have little interest in, or sympathy for, studies of socioeconomic factors and health. In the last decade, Western countries, particularly anglophone countries, have increasingly placed emphasis on individual responsibility, typified by the famous statement by Margaret Thatcher²⁰ that "there is no such thing as society, there are only families and individuals." Governments and funding agencies have been most supportive of studies that focus on individual lifestyle, and epidemiologists, either through choice or through necessity, have tended to go "where the money is."

A related issue is that socioeconomic factors are "not easily modifiable" and are "too political." However, the decision not to study socioeconomic factors is itself a political decision to focus on what is politically acceptable rather than what is most important in scientific and public health terms.²¹ Governments have repeatedly shown that social and economic differences are not God-given but are directly affected by government policies, albeit in unexpected ways.²²⁻²⁴ For example, there is some preliminary evidence that inequitable distribution of the gross national product can have a more significant impact on overall national mortality rates than the actual level of the gross national product²⁵; in some countries, a large increase in gross national product has been accompanied by little benefit in terms of health, whereas some relatively poor countries (e.g. China, Jamaica, Costa Rica) have made major improvements in health care and life expectancy.²⁶ Public health measures that aim to address the

health effects of poverty may ultimately find themselves in conflict with government policies, or may even have the ultimate policy impact of changing the government, but this does not make the role of social factors any less important or less worthy of study.

A further issue may be that epidemiologists tend to be most interested in risk factors that they can relate to, or may even be exposed to. Epidemiologists are frequently at risk from tobacco smoke, alcohol, diet, viruses, and even some occupational chemical exposures, but they are rarely at risk of being poor. The poor may be occasionally encountered in random population surveys or after taking the wrong exit from the autoroute; in daily life they are mostly invisible, although they are becoming harder to avoid as problems of homelessness and exclusion increase.

The Rise of Risk Factor Epidemiology

However, perhaps the main reason that socioeconomic factors currently receive little attention in epidemiology is that they are not considered to be real causes. Of course, many 19th-century epidemiologists also considered that socioeconomic factors were not the real causes; rather, they studied disease at the population level because the relevant biologic agents were at that time unknown.²⁷ When these agents were discovered, attention then shifted toward addressing these "real causes," and the Henle-Koch postulates displaced the population-based approach of the 19th-century epidemiologists.¹

The decline in infectious disease and the rise in relative importance of noncommunicable disease led to the development of a new epidemiologic paradigm in the mid-20th century; this involved not only a shift in the object of study and a recognition of the role of multiple causes, but also the development of new techniques of study design and data analysis.¹ In a certain sense, this new paradigm represented both a significant advance and a step back to the future, as it restored some of the population-based inferences that had fallen into decline in the late 19th century and the first half of the 20th century because of the successes of the germ theory.¹ Some key figures in the new paradigm espoused a holistic view of disease, recognized the need for a multidisciplinary approach (social, biologic, statistical), and specified the population group

as the unit of study.¹ However, the new risk factors that were studied were often conceptualized in individual terms, and individual lifestyle has received increasing emphasis during the last few decades.

Epidemiology became widely recognized with the discovery of tobacco smoking as a cause of lung cancer in the early 1950s. Subsequent decades have seen major discoveries relating to other causes of noncommunicable disease such as asbestos, ionizing radiation, and dietary factors. These epidemiologic successes have in some cases led to successful preventive interventions without the need for major social or political change. For example, occupational carcinogens can, with some difficulty, be controlled through regulatory measures, and exposures to known occupational carcinogens have been reduced in industrialized countries in recent decades. Another example is the successful World Health Organization campaign against smallpox.²⁷ More recently, some countries have passed legislation to restrict advertising of tobacco and smoking in public places and have adopted health promotion programs aimed at changes in lifestyle.

However, the success of risk factor epidemiology has been more temporary and more limited than might have been expected (S. Wing, Concepts in modern epidemiology: population, risk, dose-response and confounding, unpublished manuscript).²⁸ It is one thing to discover that tobacco smoke is the major cause of lung cancer, but redressing this situation is a different problem entirely. For example, smoking can be viewed as a strategy enabling women to cope with stress,²⁹ while at the same time undermining their health and that of their children.³⁰ Any meaningful public health intervention regarding tobacco must also consider why manual workers smoke more than nonmanual workers and find it more difficult to give smoking up³¹ and why most physicians have responded to the epidemiologic evidence and given up smoking, whereas nurses continue to smoke in great numbers. Moreover, it can be argued that the fundamental problem of tobacco lies in its production rather than in its consumption.³² The limited success of legislative measures in industrialized countries has led the tobacco industry to shift its promotional activities to developing countries, so that more people are exposed to tobacco smoke than ever before.^{33,34} Similar shifts have occurred for some occupational carcinogens.³⁵ Thus, on a global basis the

"achievement" of the public health movement has often been to move public health problems from rich countries to poor countries and from rich to poor populations within the industrialized countries. Of course, this is not solely the fault of epidemiologists. However, when a public health problem is studied in individual terms (e.g., tobacco smoking) rather than in population terms (e.g., tobacco production, advertising, and distribution, and the social and economic influences on consumption), then it is very likely that the solution will also be defined in individual terms and the resulting public health action will merely move the problem rather than solve it.

Levels of Analysis

The apparently competing explanations for disease causation (e.g. tobacco smoking in individuals vs socioeconomic factors in populations) can be reconciled by recognizing that these explanations operate at different levels of analysis. Just as the occurrence of disease within a population can be studied at many different levels³⁶—including populations, individuals, organs, tissue, cells, and molecules—the causes of disease can also be studied at these different levels, including socioeconomic factors, lifestyle, the organ burden of a carcinogen, and DNA adducts.³⁷ Although specific risk factors may appear to operate at the individual level, exposure and susceptibility^{38,39} may occur due to a wide range of political, economic, and social factors. For example, Millard discussed the factors leading to high child mortality rates in developing countries and identified three main tiers: the *proximate tier* includes the immediate biomedical conditions that result in death (involving interactions of malnutrition and infection); the *intermediate tier* includes child care practices and other behaviors that increase the exposure of children to factors on the proximate tier; and the *ultimate tier* encompasses "the broad social, economic and cultural processes and structures that lead to the differential distribution of basic necessities, especially food, shelter and sanitation."⁴⁰

Top Down and Bottom Up

Thus, any meaningful analysis of the causes of disease in populations must integrate the individual-biologic and population levels of analysis without collapsing one into the other or denying the exist-

tence of either.⁴¹ Nevertheless, it is necessary to start somewhere.

So what is the most appropriate level at which to commence to study the causes of disease in a population? Most researchers will immediately answer that their own discipline has it right, and all of the others have it wrong (what McKinlay¹⁶ described as “hardening of the categories”). Molecular biologists will focus on the etiologic process at the molecular level, much recent epidemiologic research has focused on individual lifestyle, and some epidemiologists, demographers, and social scientists have continued to conduct studies at the population level.²¹

These various pathways to understanding the disease process fall into two main approaches that mirror wider scientific debates in recent centuries. The *bottom-up* approach is inherently reductionist and positivist (i.e., it assumes that knowledge consists only of events [facts] that come from sense perception). This approach focuses on understanding the individual components of a process at the lowest possible level and using this information as the building blocks to gain knowledge about higher levels of organization. One current example is molecular epidemiology, which attempts to understand disease at the molecular level and then ultimately to use this knowledge in public health policy (e.g., by screening populations for individual susceptibility to specific carcinogens). This approach stems from the clinical tradition and is typified by an emphasis on the individual, on specific risk factors, and on the use of the randomized clinical trial as a paradigm (a variety of study designs are used, but the randomized trial is the gold standard to which the other study designs aspire). It is implicit in some recent definitions of epidemiology^{12,42} and yields useful information about the level under study (e.g., the molecular level), but it is debatable whether the bottom-up approach is an effective and efficient long-term strategy to gain knowledge or prevent disease at the population level.¹⁸

The bottom-up approach lacks distinctive theory regarding the occurrence of disease at the population level⁴³ (modern epidemiologic studies are conducted in populations, but the implicit etiologic theory is usually based at the individual-biologic level), and its products can be likened to “a vast stockpile of almost surgically clean data untouched by human thought.”²⁴ Although it has an air of scientific purity, this approach is in fact rarely used in other sciences or related

disciplines; for example, no one would attempt to predict the weather or the motion of the planets from measurements of individual molecules. Not only is such an approach impossible in practice because of the infinitely large amount of information required, but recent work in chaos theory has shown that such an approach is also impossible in theory because small inaccuracies can produce huge effects in nonlinear systems.⁴⁴

On the other hand, the *top-down* approach (variants of which include the structural approach,²⁸ the dialectical approach,⁴⁵ and the “upstream” approach¹⁶) stems in part from the demographic tradition and starts at the population level in order to ascertain the main factors that influence health status within the population. It implicitly uses a structural model of causation rather than a behavioral model or a biomedical model.⁴⁶ This approach is inherently realist⁴⁷ (i.e., it holds that the objects of study exist and—for the most part—act independently of scientists, but it differs from positivism in that the object of scientific inquiry is not patterns of events but rather the underlying processes and structures that cause these events to occur). Causation is seen as resulting from mechanisms that are internal to the population under study and that operate dialectically, rather than involving regular associations between externally related independent objects.⁴⁸ The top-down population approach is implicit in traditional definitions of epidemiology that commonly refer to “the study of the distribution and determinants of health-related states or events in specified populations, and the application of this study to control of health problems.”⁴⁹ It has been supported in a recent editorial⁵⁰ that argued for the “need to move away from the almost exclusive focus of research on individual risk, toward the social structures and processes within which ill-health originates, and which will be more amenable to modification.”⁵¹

Beyond Modern Epidemiology

So how can we go “back to the future”¹⁶ and develop new forms of epidemiology that restore the population perspective but that appropriately use recent methodologic advances?

The key issue is that epidemiology is first and foremost a branch of public health.⁵² This view is not contradictory to the view of epidemiology as a branch of science; in fact, it is necessary to take a

scientific approach to discover the major causes of disease in populations. However, if the goal is to understand and prevent the causation of disease in populations, then epidemiology should start at the population level and should address the major determinants of health and disease at this level. For example, the recent Leeds declaration⁴ emphasized the need to refocus upstream and to use research methods that are appropriate to the level at which intervention will take place. Epidemiologic techniques can be used in other settings (e.g., clinical epidemiology) and for other purposes (e.g., studies of disease progression and prognosis), but the key contribution of epidemiology to public health is its population focus.

Of course, epidemiologic studies in populations involve individuals who have specific exposures, but the important distinction is whether or not the etiologic framework is conceptualized at the population level and whether or not these exposures are placed in their social and historical context.⁵ For example, just as a variety of health effects in various organ systems (e.g., various types of cancer) may have a common contributing cause (e.g., tobacco smoking) at the level of the individual, a variety of individual exposures (e.g., smoking, diet) may have common socioeconomic causes at the population level (e.g., poverty). On the other hand, the need to analyze the causes of disease at the population level does not mean that public health action should only be taken at the population level. Indeed, there is a danger of a social engineering approach to public health that itself reinforces the social structures that cause disease. Just as it is important to understand the causation of disease at both the population and the individual levels, it is also essential to take action at both the population and the individual levels in ways that increase rather than decrease individuals’ control over their environment.⁵³

It is also important to recognize that the “populations” that epidemiologists study are not just collections of individuals that are conveniently grouped for the purposes of study.²¹ Every population has its own history, culture, organization, and economic and social divisions, which influence how and why people are exposed to particular factors. For example, Terris argues that

the causes of cholera in India today go back hundreds of years in India’s history, to the British invasion and destruc-

tion of once-flourishing textile industries; the maintenance of archaic systems of land ownership and tillage; the persistence of the caste system and the unbelievable poverty, hunger, and crowding; the consequent inability to afford the development of safe water supplies and sewage disposal systems; and, almost incidentally, the presence of cholera vibrios.⁵⁴

Thus, epidemiology is inevitably entangled with society, and it is not feasible or desirable to study the causes of disease in the abstract.⁵ To understand the causation of disease in a population, it is essential to understand the historical and social context and to emphasize the importance of diversity and local knowledge rather than only searching for universal relationships.⁵⁵ This requires a greater involvement from the social sciences and a more multidisciplinary approach.^{56,57} Epidemiology is just one of the approaches by which the major determinants of health in a population can be addressed, and it should be complemented by other quantitative approaches from the social sciences, as well as qualitative and historical studies. The emphasis should be on using appropriate methodology⁵¹ rather than making the problem fit the method.

Rose⁵⁸ also noted that entire populations may be exposed to a particular risk factor, and there is usually a continuum of disease risk (rather than a clear distinction between the sick and the healthy) across the population. Small improvements in the health of a "sick population" may be more effective than attempts to treat or prevent illness in "sick individuals."⁵⁸ Studying these phenomena often requires the use of ecologic studies, since group variables may be important apart from individual-level variables⁵⁹ (although they may not be an adequate substitute for the individual-level variables, especially for confounding control⁶⁰). For example, analysis at the individual level cannot explain epidemic spread at the group level and cannot even fully explain the spread of infections between individuals. The problems specific to ecologic analysis⁶¹ arise only when one extrapolates downward from the population to the individual level; many criticisms of ecologic studies are based on the questionable assumption that the individual level of analysis is most appropriate.

It should also be emphasized that the strength, and even the direction, of associations between risk factors and disease will vary between populations and over time. For example, coronary heart disease

was at one time a disease of the affluent, but it has become a disease of the poor as smoking and eating habits have changed over time.⁶² Thus, appropriate preventive measures at the population level will differ widely between populations. Furthermore, although many specific risk factors play an important role in any population, their contribution to disease risk is modified by the baseline disease risk and the presence of various cofactors, making it impossible to assume a universal dose-response relationship.²¹ A related issue is the importance of considering interrelationships between causes rather than considering each cause in isolation.⁶³

Finally, the randomized clinical trial may be an appropriate paradigm in many epidemiologic studies of specific risk factors, but it often is inappropriate in studies that require a consideration of the historical and social context. The danger is that attempting to eliminate the influence of all other causes of diseases—in an attempt to control confounding—strips away the essential historical and social context,²¹ as well as the multiple moderating influences that constitute true causation.⁶³ Thus, the tendency to only study factors that fit the clinical trial paradigm should be resisted, and appropriate study designs should be chosen (or developed) to fit the public health question that is being addressed.⁶⁴

Epidemiology has become a set of generic methods for *measuring* associations of exposure and disease in individuals, rather than functioning as part of a multidisciplinary approach to *understanding* the causation of disease in populations. These methodologic changes have not been neutral, but rather, in combination with other influences, they have changed—and have reflected changes in—the way in which epidemiologists think about health and disease. We seem to be using more and more advanced technology to study more and more trivial issues, while the major population causes of disease are ignored. Epidemiology must reintegrate itself into public health, and must rediscover the population perspective. □

Acknowledgments

This work was funded by a Senior Research Fellowship of the Health Research Council of New Zealand.

I would like to thank Warwick Armstrong, Robert Beaglehole, Paolo Boffetta, Sunny Collings, Fiona Cram, Peter Davis, Isabelle Godin, Philippa Howden-Chapman, Rod Jackson, Ichiro Kawachi, Manolis Kogevi-

nas, Meri Koivusalo, John McKinlay, Tony McMichael, Timo Partanen, Charlotte Paul, Steve Wing, and Karen Witten for their comments on the draft manuscript.

References

1. Susser M. Epidemiology in the United States after World War II: the evolution of technique. *Epidemiol Rev.* 1985;7:147-177.
2. Lilienfeld D, Lilienfeld A. Epidemiology: a retrospective study. *Am J Epidemiol.* 1977; 106:445-449.
3. Terris M. Epidemiology and the public health movement. *J Public Health Policy.* 1987;7:315-329.
4. *Directions for Health: New Approaches to Population Health Research and Practice.* The Leeds Declaration. Leeds, England: Nuffield Institute for Health, University of Leeds; 1993.
5. Susser M. Epidemiology today: "a thought-tormented world." *Int J Epidemiol.* 1989;18: 481-488.
6. McKeown T. *The Role of Medicine.* Princeton, NJ: Princeton University Press; 1979.
7. Szreter S. The importance of social intervention in Britain's mortality decline c. 1850-1914: a reinterpretation of the role of public health. *Soc Hist Med.* 1988;1:1-37.
8. Smith GD, Morris J. Increasing inequalities in the health of the nation. *BMJ.* 1994;309:1453-1454.
9. Pappas G, Queen S, Hadden W, Fisher G. The increasing disparity in mortality between socioeconomic groups in the United States, 1960 and 1986. *N Engl J Med.* 1993;329:103-109.
10. Logan WPD. *Cancer Mortality by Occupation and Social Class, 1951-1971.* Lyon, France: International Agency for Research on Cancer; 1982.
11. Doll R, Peto R. *The Causes of Cancer.* Oxford, England: Oxford University Press; 1981.
12. Rothman KJ. *Modern Epidemiology.* Boston, Mass: Little, Brown; 1986.
13. Stallones S. To advance epidemiology. *Annu Rev Public Health.* 1980;1:69-82.
14. Cameron D, Jones IG. John Snow, the Broad Street pump and modern epidemiology. *Int J Epidemiol.* 1983;12:393-396.
15. Krieger N. Epidemiology and the web of causation: has anyone seen the spider? *Soc Sci Med.* 1994;39:889-903.
16. McKinlay JB. Towards appropriate levels of analysis, research methods and health public policy. Presented at the International Symposium on Quality of Life and Health: Theoretical and Methodological Considerations; May 25-27, 1994; Berlin, Germany.
17. McMichael AJ. "Molecular epidemiology." New pathway or new travelling companion? *Am J Epidemiol.* 1994;140: 1-11.
18. Pearce N, san Jose S, Boffetta P, et al. Limitations of biomarkers of exposure in cancer epidemiology. *Epidemiology.* 1995;6: 190-194.
19. Vandembroucke JP. New public health and old rhetoric. *BMJ.* 1994;308:994-995.
20. Thatcher M. *Womans Own.* October 1987.
21. Wing S. Limits of epidemiology. *Med Global Survival.* 1994;1:74-86.
22. Black D. Deprivation and health. *BMJ.* 1993;307:1630-1631.

23. Hewlett SA. *Child Neglect in Rich Nations*. New York, NY: UNICEF, 1993.
24. Pearce N. Economic policy and health in The Year of the Family. *NZ Med J*. 1994;107:379-381.
25. Wilkinson R. National mortality rates: the impact of inequality. *Am J Public Health*. 1992;82:1082-1084.
26. Sen A. *Levels of Poverty: Policy and Change*. Washington, DC: The World Bank; World Bank Staff Working Paper No. 401. 1980.
27. Tesh SN. *Hidden Arguments: Political Ideology and Disease Prevention Policy*. London, England: Rutgers; 1988.
28. Loomis D, Wing S. Is molecular epidemiology a germ theory for the end of the twentieth century? *Int J Epidemiol*. 1991;19:1-3.
29. Jacobson B. *The Lady Killers: Why Smoking Is a Feminist Issue*. London, England: Pluto Press; 1981.
30. Power C, Manor O, Fox J. *Health and Class: The Early Years*. London, England: Chapman Hall; 1991.
31. Pearce N, Davis PB, Smith AH, Foster FH. Social class, ethnic group, and male mortality in New Zealand, 1974-8. *J Epidemiol Community Health*. 1985;39:9-14.
32. Reid P, Pouwhare R. *Te Taonga mai Tawhiti: The Gift from a Distant Place*. Auckland, New Zealand: Niho Taniha; 1991.
33. Barry M. The influence of the U.S. tobacco industry on the health, economy, and environment of developing countries. *N Engl J Med*. 1991;324:917-920.
34. Tominaga S. Spread of smoking to the developing countries. In: Zaridze D, Peto R, eds. *Tobacco: A Major International Health Hazard*. Lyon, France: International Agency for Research on Cancer; 1986:125-133.
35. Pearce NE, Matos E, Vainio H, Boffetta P, Kogevinas M, eds. *Occupational Cancer in Developing Countries*. Lyon, France: International Agency for Research on Cancer; 1994.
36. Susser M. *Causal Thinking in the Health Sciences*. New York, NY: Oxford University Press; 1973.
37. Potter JD. Reconciling the epidemiology, physiology, and molecular biology of colon cancer. *JAMA*. 1992;268:1573-1577.
38. Syme SL, Berkman LF. Social class, susceptibility and sickness. *Am J Epidemiol*. 1976;104:1-8.
39. Cassel J. The contribution of the social environment to host resistance. *Am J Epidemiol*. 1976;104:107-123.
40. Millard AV. A causal model of high rates of child mortality. *Soc Sci Med*. 1994;38:253-268.
41. Rose S, Lewontin RC, Kamin LJ. *Not in Our Genes: Biology, Ideology and Human Nature*. London, England: Penguin Books; 1990.
42. Miettinen OS. *Theoretical Epidemiology: Principles of Occurrence Research*. New York, NY: Wiley; 1985.
43. Smith A. The epidemiological basis of community medicine. In: Smith A, ed. *Recent Advances in Community Medicine 3*. Edinburgh, Scotland: Churchill Livingstone; 1985:1-10.
44. Firth WJ. Chaos—predicting the unpredictable. *BMJ*. 1991;303:1565-1568.
45. Levins R, Lewontin R. *The Dialectical Biologist*. Cambridge, Mass: Harvard University Press; 1985.
46. Armstrong D. *An Outline of Sociology as Applied to Medicine*. Bristol, England: Wright; 1980.
47. Bhaskar R. *Reclaiming Reality*. London, England: Verso; 1989.
48. Jones K, Moon G. *Health, Disease and Society: A Critical Medical Geography*. London, England: Routledge & Kegan Paul; 1987.
49. Last JM, ed. *A Dictionary of Epidemiology*. New York, NY: Oxford University Press; 1988.
50. Anon. Population health looking upstream. *Lancet*. 1994;343:429-430. Editorial.
51. McKinlay JB. The promotion of health through planned sociopolitical change: challenges for research and policy. *Soc Sci Med*. 1993;36:109-117.
52. Lilienfeld A, Lilienfeld DE. Epidemiology and the public health movement. *J Public Health Policy*. 1982;3:140-149.
53. Adler NE, Boyce WT, Chesney MA, Folkman S, Syme SL. Socioeconomic inequalities in health: no easy solution. *JAMA*. 1993;269:3140-3145.
54. Terris M. The epidemiologic tradition. *Public Health Rep*. 1979;94:204.
55. Kunitz S. *Disease and Social Diversity*. New York, NY: Oxford University Press; 1994.
56. Susser M, Watson W, Hopper K. *Sociology in Medicine*. 3rd ed. New York, NY: Oxford University Press; 1985.
57. Connolly J. Social sciences provide a framework for epidemiology. *BMJ*. 1994;308:1569. Letter.
58. Rose G. *The Strategy of Preventive Medicine*. Oxford, England: Oxford University Press; 1992.
59. Koopman JS, Longini IM. The ecological effects of individual exposures and nonlinear disease dynamics in populations. *Am J Public Health*. 1994;84:836-842.
60. Greenland S, Robins J. Ecologic studies—biases, misconceptions, and counterexamples. *Am J Epidemiol*. 1994;139:747-760.
61. Susser M. The logic in ecological: II. the logic of design. *Am J Public Health*. 1994;84:830-835.
62. Wing S. Social inequalities in the decline of coronary mortality. *Am J Public Health*. 1988;78:1415-1416.
63. Dean K. Creating a new knowledge base for the new public health. *J Epidemiol Community Health*. 1994;48:217-219.
64. McMichael AJ. Persons, populations and planets: a full world brings epidemiology full circle. *Epidemiology*. In press.