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EPIDEMIOLOGY AND THE WEB OF CAUSATION: HAS ANYONE SEEN THE SPIDER?

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Abstract—'Multiple causation' is the canon of contemporary epidemiology, and its metaphor and model is the 'web of causation.' First articulated in a 1960 U.S. epidemiology textbook, the 'web' remains a widely accepted but poorly elaborated model, reflecting in part the contemporary stress on epidemiologic methods over epidemiologic theories of disease causation. This essay discusses the origins, features, and problems of the 'web,' including its hidden reliance upon the framework of biomedical individualism to guide the choice of factors incorporated in the 'web.' Posing the question of the whereabouts of the putative 'spider,' the author examines several contemporary approaches to epidemiologic theory, including those which stress biological evolution and adaptation and those which emphasize the social production of disease. To better integrate biologic and social understandings of current and changing population patterns of health and disease, the essay proposes an ecosocial framework for developing epidemiologic theory. Features of this alternative approach are discussed, a preliminary image is offered, and debate is encouraged.

Key words—ecosocial, health inequalities, philosphy of science, social class, social epidemiology

> . . . And right spang in the middle of the web there were the words 'Some Pig.' The words were woven right into the web. They were actually part of the web, Edith. I know, because I have been down there and seen them. It says, 'Some Pig,' just as clear as clear can be. There can be no mistake about it. A miracle has happened and a sign has occurred here on earth, right on our farm, and we have no ordinary pig.
>
> 'Well,' said Mrs Zuckerman, 'it seems to me you're a little off. It seems to me we have no ordinary *spider*.'
>
> E. B. White
> *Charlotte's Web* [1, p. 80]

'Multiple causation' is the canon of contemporary epidemiology, and its metaphor and model is the 'web of causation.' Expressed through the notion of 'multifactorial etiology' [2–4] and embedded in the statistical techniques of 'multivariate analysis' [3–7], the belief that population patterns of health and disease can be explained by a complex web of numerous interconnected risk and protective factors has become one of this discipline's central concepts [3, 4]. Equally entrenched is the corollary that epidemiology's power to improve the public's health rests upon its ability to identify—and predict the results of breaking—selected strands of this causal web [2–4].

The widespread adoption of a multicausal framework would suggest the existence of a well-developed body of epidemiologic literature that analyzes its essential features and explores its implications for causal reasoning about population patterns of health and disease. Instead, theoretical work in epidemiology—especially in the United States—has recently focused on methodologic issues [3–8] and debates about causation have chiefly concerned the nature and validity of causal inference [5, 8–10]. These, however, are problems for any science, not just epidemiology [11, 12]. By contrast, relatively little work has been devoted to developing the concepts and framework of what might be termed epidemiologic theory, i.e. explanations of the current and changing health status of human societies [4, 13–20]. Even less has addressed the fundamental question posed by the 'web's' suggestive imagery: who or what is the 'spider' responsible for its array of factors? [17, p. 244].

The paucity of critical reflection is not just an academic point, but cuts to the core of epidemiology as a science and profession [13–21]. As noted by several senior epidemiologists in the U.S.—most especially Susser [3, 18], Terris [4, 14, 19], Stallones [15], Lilienfeld and Lilienfeld [20] and Kuller [21], modern epidemiology often seems more concerned with intricately modeling complex relationships among risk factors than with understanding their origins and implications for public health. Reflecting this trend, graduate students in epidemiology are far more likely to be taught about study design and data analysis than they are about how to generate epidemiologic hypotheses about the societal dynamics of health and disease. Most current U.S. epidemiologic textbooks, for example, focus almost exclusively on methodologic issues and devote little, if any, space to explaining the different theories of disease causation and etiologic concepts that help epidemiologists formulate hypotheses in the first place (e.g. 'time, place, and person,' 'mode of transmission,' 'herd immunity,' 'environment,' and 'lifestyle'); even fewer discuss the

origin of these ideas or the history of epidemiology itself (Table 1).

Although the importance of learning a science's methodology is obvious, epidemiology is more than a mere amalgam of methods and study designs. Whether explicitly articulated or not, epidemiologic research embodies particular ways of seeing as well as knowing the world, with the express intent of analyzing and improving the public's health [2–4, 13–21]. Because the insights of any science—including epidemiology—are as much, if not more, dependent upon its concepts and theories than upon its specific

Table 1. Survey of U.S. epidemiologic textbooks and anthologies published since 1970: content on epidemiologic history and theory,[a] and diagram of 'web of causation'

Text	Total pages	Percentage of pages on epidemiologic History	Percentage of pages on epidemiologic Theory	Diagram of Web
Textbooks				
MacMahon B. and Pugh T. F. *Epidemiology: Principles and Methods.* Little, Brown, Boston, 1970.	302	0.0	11.6	+
Fox J. P., Hall C. E. and Elveback L. R. *Epidemiology: Man and Disease.* Macmillan, New York, 1970.	339	3.5	44.8	—
Susser M. *Causal Thinking in the Health Sciences: Concepts and Strategies of Epidemiology.* Oxford University Press, New York, 1973.	181	12.2	8.3	—
Mausner J. S. and Bahn A. K. *Epidemiology: an Introductory Text.* Saunders, Philadelphia, 1974.	377	0.0	4.0	+
Friedman G. *Primer of Epidemiology.* McGraw-Hill, New York, 1974.	230	0.0	0.9	+
White K. L. and Henderson M. (Eds) *Epidemiology as a Fundamental Science: its uses in Health Services Planning, Administration, and Evaluation.* Oxford University Press, New York, 1976.	235	0.9	0.9	—
Lilienfeld A. and Lilienfeld D. *Foundations of Epidemiology.* Oxford University Press, New York, 1980.	375	6.1	5.1	—
Kleinbaum D. G., Kupper L. L. and Morgenstern H. (Eds) *Epidemiologic Research: Principles and Quantitative Methods.* Lifetime Learning Publications, Belmont, CA, 1982.	529	0.0	1.1	—
Schlesselman J. *Case-Control Studies: Design, Conduct, Analysis.* Oxford University Press, New York, 1982.	354	0.6	0.0	—
Kahn H. A. *An Introduction to Epidemiologic Methods.* Oxford University Press, New York, 1983.	166	0.0	0.0	—
Miettinen O. S. *Theoretical Epidemiology: Principles of Occurrence Research in Medicine.* Wiley, New York, 1985.	359	0.0	1.4	—
Feinstein A. R. *Clinical Epidemiology: the Architecture of Clinical Research.* Saunders, Philadelphia, 1985	812	1.1	1.2	—
Weiss N. *Clinical Epidemiology: The Study of the Outcome of Illness.* Oxford University Press, New York, 1986.	144	0.0	0.0	—
Rothmam K. *Modern Epidemiology.* Little, Brown, Boston, 1986.	358	1.7	0.0	—
Kelsey J., Thompson W. D. and Evans A. S. *Methods in Observational Epidemiology.* Oxford University Press, New York, 1986.	366	0.0	7.4	—
Hennekens C. H. and Buring J. E. *Epidemiology in Medicine.* Little, Brown, Boston, 1987.	383	2.6	3.9	—
Abramson J. H. *Making Sense of Data: a Self-instruction Manual on the Interpretation of Epidemiologic Data.* Oxford University Press, New York, 1988.	326	0.0	0.6	—
Anthologies				
Winklestein W. Jr, French F. E. and Lane J. M. (Eds) *Basic Readings in Epidemiology.* MSS Educational Pub. Co., New York, 1970.	193	13.9	27.8	—
Greenland S. (Ed.) *Evolution of Epidemiologic Ideas: Annotated Readings on Concepts and Methods.* Epidemiology Resources, Inc., Chestnut Hill, MA, 1987.	190	7.9	0.0	—
Buck C., Llopis A., Najera E. and Terris M. (Eds) *The Challenge of Epidemiology: Issues and Selected Readings.* Pan American Health Organization, Washington, DC, 1988.	989	14.8	24.9	—
Rothman K. (Ed.) *Causal Inference.* Epidemiology Resources, Chestnut Hill, MA, 1988.	207	0.0	0.0	—

[a]Epidemiologic theory: defined as explicit discussion of theories of disease causation and/or epidemiologic concepts (e.g. 'time, place, person').

approaches to obtaining data [6, 11, 22–25], it is important to consider how the present-day multivariate framework—as embodied in the metaphor of the 'web of causation'—has influenced the etiologic explanations and research agendas that epidemiologists now pursue, especially in the United States but in other regions as well.

THE 'WEB OF CAUSATION': ORIGINS, FEATURES, AND PROBLEMS

The year 1960 marks the first mention of the 'web of causation' in the epidemiologic literature [2, 3]. Not coincidentally, it appeared in the first formal epidemiologic textbook ever published in the United States, *Epidemiologic Methods*, by Brian MacMahon, Thomas F. Pugh, and Johannes Ipsen (Fig. 1) [2]. Although the need for epidemiologists to think in terms of 'multiple causation,' was likewise stressed by the other two groundbreaking epidemiologic textbooks written in this period (Morris' *Uses of Epidemiology* [26] and *Principles of Epidemiology* by Taylor and Knowelden [27], both published in England in 1957), neither captured this concept in as compelling or succinct an image as the 'web.'

The appearance of these textbooks and their orientation to disease causation did not occur in an historical vacuum. Signaling a new phase in the professionalization of epidemiology, these texts were developed to serve as fundamental teaching tools for the growing number of epidemiology courses being offered in the United States, England, and Scotland [3, 28]. Their goal was to imbue a new generation of researchers with the etiologic and methodologic insights acquired in the 1940s and 1950s as epidemiologists increasingly added 'chronic' conditions, like cancer and heart disease, to a research agenda previously dominated by acute and chronic infectious diseases [3, 4, 26–29].

During this period, several books and essays published in the United States, with titles like *The Epidemiology of Health* [29], *Beyond the Germ Theory* [30], and 'Epidemiology—old and new' [31], advocated replacing single-agent 'germ theories' of disease

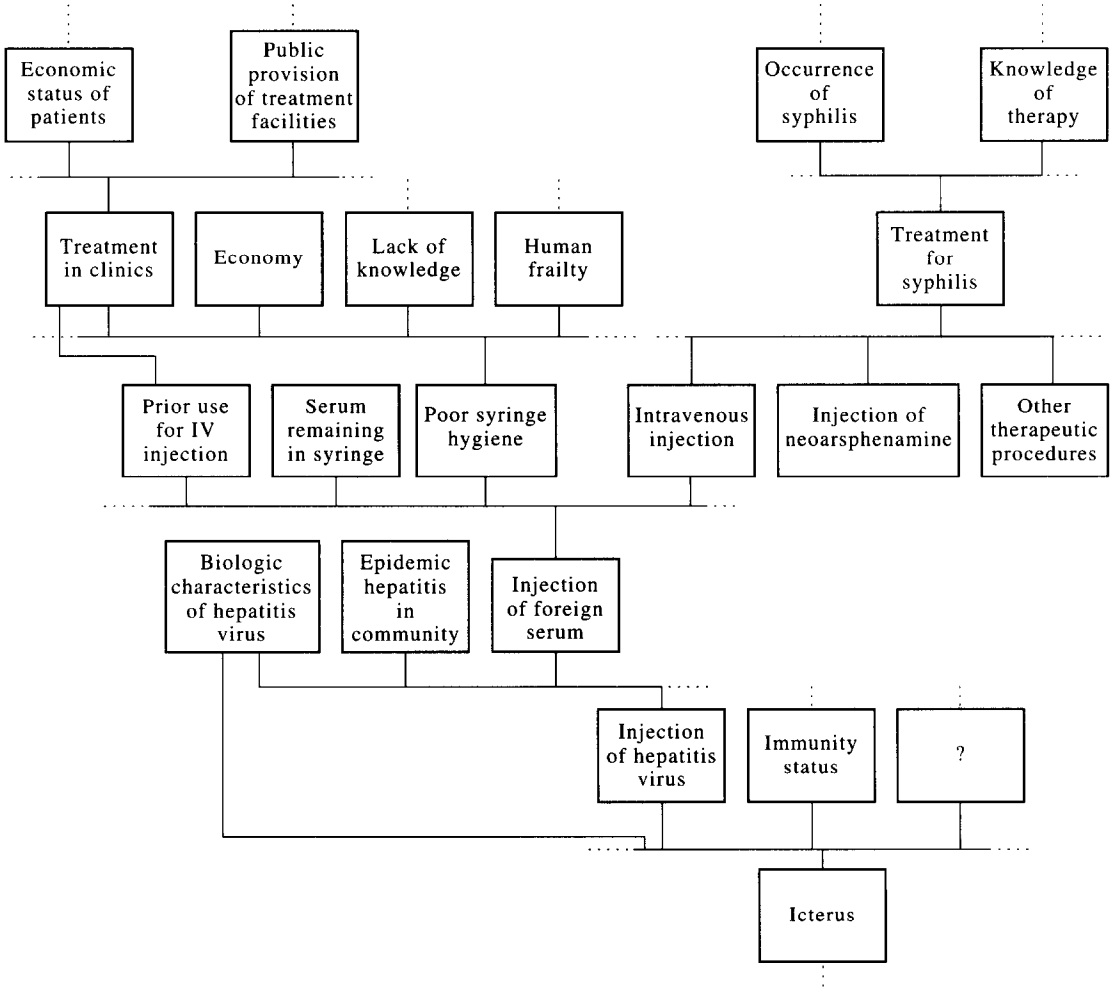


Fig. 1. McMahon *et al.*'s 'web of causation': "Some components of the association between treatment for syphilis and jaundice" [2, p. 19]. Reprinted with permission from the publisher.

with more complex models of 'host, agent, and environment.' Others, under the banner of 'social medicine,' argued in favor of explicitly examining the 'social determinants' of health [28, 32, 33]. And, as part of meeting these challenges, epidemiologists and biostatisticians refined and developed new study designs and statistical techniques, including the approximation of prospectively-determined relative risks with odds ratios derived from case-control studies [5, 34].

Other events in the 1950s also left their mark upon the development of epidemiologic theory. The discovery of the double-helix structure of DNA by Watson, Crick and Franklin in 1953 [35] augured an explosion in biological knowledge, and raised new hopes of improving understanding of gene—environment interactions. That same year, Nordling developed the first mathematical multistage-mutation model of carcinogenesis to explain the shape of cancer incidence curves [36], thereby extending epidemiologic attempts to link the micromechanisms of disease causation with population patterns of disease occurrence.

Epidemiologic thought was also stimulated by the growing use of computers to store and analyze large data sets, especially using multivariate techniques [3, 37]. The first massive civilian demonstration of this new possibility involved the 1950 U.S. census and employed the ENIAC computer, which had been built in the 1940s at the request of the U.S. War Department in World War II [38]. In 1956 the computer language FORTRAN was developed [39], and by the end of the decade, the potential of computers for cancer registries and epidemiologic studies was clearly understood [37].

It was another aftermath of World War II, however, that perhaps most strongly shaped the subsequent U.S. academic discourse about disease causation: the Cold War and its domestic corollary of McCarthyism. In a period when discussion of social class and social inequality was tantamount to heresy (even in the social sciences) [40–42], and when early civil rights activists were branded as 'subversive' (e.g. supporters of the 1955 Montgomery bus boycott) [40, 43], it is not surprising that epidemiologists (like other academics) generally eschewed dangerous speculation about the 'social determinants' of health. Instead, most pursued research based upon more biomedical and individually-oriented theories of disease causation, in which population risk was thought to reflect the sum of individuals' risks, as mediated by their 'lifestyles' and genetic predisposition to disease [3, 44, 45].

It was in this context that MacMahon *et al.* introduced the concept of the 'web of causation' [2, p. 18]. They did so in reaction to the then prevalent notion of 'chains of causation,' which they argued failed to take into account: (1) the 'complex genealogy of antecedents' of each 'component' in the 'chain' [2, p. 18], and (2) how the genealogies of diverse factors or outcomes might overlap, creating a variety of indirect as well as direct associations. Expressly challenging the still-pervasive tendency of epidemiologists to think in terms of single 'agents' causing discrete diseases, the provocative metaphor and model of the 'web' invited epidemiologists to embrace a more sophisticated view of causality.

Conceptually, the metaphor evoked the powerful image of a spider's web, an elegantly linked network of delicate strands, the multiple intersections representing specific risk factors or outcomes, and the strands symbolizing diverse causal pathways. It encouraged epidemiologists to look for multiple causes and multiple effects, to consider interaction, and to identify the many—as opposed to singular—routes by which disease could be prevented. With this metaphor in the background, epidemiologists could also treat the 'web' as a model to delineate the etiology of, and guide research about, specific health problems. More profoundly, the 'web' tapped into an intuitive sense of interconnection, one long a part of many philosphical traditions [46–48] and, during the 1950s, increasingly incorporated into other scientific disciplines, especially cybernetics and ecology [48, 49].

To illustrate their view, MacMahon *et al.* diagrammed 'some components' of the relationship between two etiologically distinct diseases, syphilis and hepatitis, whose independent 'chains' had no logical reason to intersect (Fig. 1). Asserting that "the whole genealogy might be thought of more appropriately as a web, which in its complexity and origins lies quite beyond our understanding" [2, p. 18], they proffered a picture that simultaneously detailed *how* the hepatitis virus might get into syringe needles used to treat syphilis patients, producing and outbreak of jaundice (icterus), and yet left to the reader's imagination (as indicated by the suggestive dots trailing off the edge of the page) the determinants of other factors, e.g. the 'economic status of patients,' 'human frailty,' the 'public provision of treatment facilities,' the 'occurrence of syphilis,' and 'knowledge of therapy.'

Using this model, MacMahon *et al.* drew several important inferences about prevention and research that remain part of epidemiologic thinking to this day [4, 8, 15, 18]. Arguing that "to effect preventive measures, it is not necessary to understand causal mechanisms in their entirety" [2, p. 18], they stated that "[e]ven knowledge of one small component may allow some degree of prevention," since "wherever the chain is broken the disease will be prevented" [2, p. 18].

With this image in mind of cutting strands rather than attempting to identify and alter the source(s) of the web, MacMahon *et al.* urged epidemiologists to abandon "semantic exercises aimed at hierarchic classification of causes" [2, p. 20]. They instead should seek out the 'necessary' (albeit rarely sufficient) causes most amenable to 'practical' interventions and nearest (in terms of the web's configuration) to the specified outcome. For example, to prevent infectious diseases, they observed that it "is a good

deal easier to control human water supply and to eradicate insect vectors than to breed genetically resistant populations" [2, pp. 20–21]. Noting, however, that "the alteration of any cause may be expected to have multiple effects besides the one intended", "MacMahon *et al.* cautioned that these "side effects" must be "acceptable" [2, p. 22], although they never specified acceptable to whom.

The 'web of causation' quickly gained widespread recognition as a useful concept to help orient research regarding the multifactorial etiology of disease [3, 4, 6, 15, 50]. Even so, the 'web' subsequently has appeared in only a handful of other epidemiologic texts [51–53]—in essentially the same form, and without any real elaboration (Table 1). The reason for this omission is not the 'web's' lack of utility for epidemiologic thought. Instead, notions of multiple causation and multivariate analysis are so commonplace and so embedded in modern epidemiologic reasoning that they hardly merit discussion as a model or as an approach to understanding disease [3, 54]. The 'web' as such is a 'given', and what garners attention are the analytic problems posed by the intricate concatenations of its component parts [3–8].

Guided by a 'multicausal' view, for example, epidemiologists have greatly developed their understanding of such critical phenomena as confounding and effect modification [3, 5, 8, 50]. Among these, Rothman has called for greater attention to the relationships between 'necessary' and 'component' causes [55]. To illustrate this point, in 1976 he diagrammed several hypothetical combinations of causal components sufficient to produce a hypothetical disease; whereas some component causes (necessary but not sufficient) belonged to more than one 'causal pie,' others (the necessary causes) belong to all the 'pies' (Fig. 2) [55]. Using this imagery, Rothman has shown how the strenth of an association between a component cause and a given outcome depends not simply on their specific relationship but also on the prevalence of the other component causes required to bring about the specified change [8, 55]—an insight not obvious at the time the 'web' was first proposed.

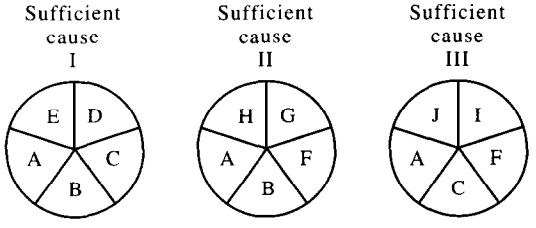


Fig. 2. Rothman's "conceptual scheme for the causes of a hypothetical disease" [55, p. 589]. Text accompanying figure: "This disease (effect) has three sufficient causal complexes, each having five component causes. In this scheme 'A' is a necessary cause, since it appears as a member of each sufficient cause. On the other hand, the component causes 'B,' 'C,' and 'F,' which each appear in more than one sufficient cause, are not necessary causes, because they fail to appear in all three sufficient causes" [55, p. 588]. Reprinted with permission from the publisher.

As critical as these developments are, it is essential to note what these views of multifactorial etiology omit: discussion of the origins—as opposed to interactions—of the multiple causes. MacMahon *et al.*, for example, never explained *why* they selected the 'components' that appear in their 'web' and left others out, nor did they offer any specific advice as to how others might elucidate the elements of other 'webs.' Having decreed the origins of the 'web' outside the bounds of epidemiologic enquiry, these authors never invoked—and essentially proscribed—the imagery of the 'spider.' Similarly, in the case of Rothman's 'pies,' the 'cook' is notably absent.

What could account for the construction of a 'spiderless' 'web'? One answer is that the 'web' was never intended to be a 'theory.' It was not elaborated to provide *explanations* of causal links, but instead was developed to enhance epidemiologists' ability to depict and study complex interrelationships between *specified* risk factors and diseases.

This answer, however, is not sufficient: models do not exist independently of theories [11, 22–25]. Theories attempt to explain *why* phenomena exist and are interrelated. By contrast, models attempt to portray *how* these connections occur and are always constructed with elements and relationships specified *by* particular theories [11, 22–25]. In ecology, for example, diverse predator/prey models may illustrate *how* the populations of two species covary, but it is evolutionary theory that directs researchers to consider *why* this relationship exists and how it developed, through reference to such phenomena and concepts as consumption, reproduction, genetic variation, natural selection, and historical contingency [24]. The absence of any discussion of the theory shaping the model of the 'web' is thus extremely problematic.

If the 'web' is a model, it follows that it must be based on or reflect some theory. Closer inspection of the elements of the 'web' in turn discloses the theoretical orientation woven into its very fabric. One cardinal feature is that, despite claims about being inclusive and non-hierarchic in its depiction of risk factors, the 'web' in fact employs a type of weighting that levels all distinctions [54, 56]. In MacMahon *et al.*'s 'web,' 'treatment in clinics' and the 'economy' occupy the same level, and rate the same kind of 'box' as 'injection of foreign serum' and 'epidemic hepatitis in community' (Fig. 1). The 'web' thus invevitably focuses attention on those risk factors 'closest' to the 'outcome' under investigation, and these in turn typically translate to the direct biologic causes of disease in individuals and/or to 'lifestyles' and other risk factors that allegedly can be addressed at the individual level through education or medical intervention.

Another important aspect of the 'web' is that it does not differentiate between determinants of disease in individuals and in populations. Stated another way, it fails to distinguish between what Rose, a

noted English epidemiologist, has aptly termed 'the causes of cases' vs 'the causes of incidence' [16, 57]. As Rose has shown, these two sets of causes are *not* necessarily the same and require difference research questions: asking "why do some individuals have hypertension?" is not equivalent to enquiring "why do some populations have much hypertension, whilst in others it is rare?" (Fig. 4). The former emphasizes individual susceptibility and interventions aimed at 'high risk' individuals, whereas the latter highlights population exposures and the need to shift the distribution of disease in the entire population (which will always have its outliers) to a healthier state [57]. And, by excluding any sense of history or origins, the 'web' sans 'spider' discourages epidemiologists from considering *why* population patterns of health and disease exist and persist or change over time.

As noted by other critics [54, 56], these aspects of the 'web' help identify its underlying theoretical framework: that of biomedical individualism [44]. This framework, often referred to as the 'biomedical model,' has three key features. It emphasizes biological determinants of disease amenable to intervention through the health care system, considers social determinants of disease to be at best secondary (if not irrelevant), and views populations simply as the sum of individuals and population patterns of disease as simply reflective of individual cases [44, 45, 54, 56, 58–63]. In this view, disease in populations is reduced to a question of disease in individuals, which in turn is reduced to a question of biological malfunctioning. This biologic substrate, divorced from its social context, thus becomes the optimal locale for interventions, which chiefly are medical in nature [44, 45, 54]. It is essential to stress that the 'web' did not challenge this basic biomedical and individualistic orientation to disease causation. What it opposed were simplistic interpretations of the doctrine of 'specific etiology,' which holds that single agents uniquely cause specific diseases [4, 6, 30, 64]. The novel feature of the 'web' was that it emphasized the need to consider, simultaneously, how diverse aspects of the host, agent and environment were implicated in the multifactorial etiology of disease.

THE INFLUENCE OF EPIDEMIOLOGIC THEORY UPON EPIDEMIOLOGIC THOUGHT

The hidden role of etiologic theory in the 'web' stands in stark contrast to the open debates about epidemiologic theory that frequently occurred during earlier periods of epidemiology's development as a discipline. Emerging in the early 1800s—alongside the rise of cell theory, sociology, and political economy [24, 38, 42, 65, 66]—epidemiology from the outset was as much a testing ground for different theories about the influence of biology and society on health as it was a practical enterprise geared toward describing population patterns of disease and influencing health policy [14, 65, 66]. Much of the research of the 19th century epidemiologists in both Europe and the United States, for example, was motivated or shaped by two central (and connected) debates: (1) whether miasma or contagion was the principal cause of epidemic disease [14, 65–70], and (2) whether poverty was the cause or result of poor health and immorality [18, 65, 66, 69, 70]. Other important polemics focused on whether women's ill health was a consequence of too much or too little education, employment, or activity [71], and, in the United States, on whether the poor health status of Black men and women was the result of slavery or their allegedly 'inferior' constitutions [62, 72]. In both cases, these disputes extended beyond narrow arguments about specific factors associated with particular diseases to fundamental debates about the *types* of causal factors—ranging from the biologic to the social—that could legitimately be invoked to explain population patterns of disease.

Epidemiology in the early 20th century also was characterized by contention between several schools of thought. Like their 19th century counterparts, these schools differed in their assessment of both the *number* and *realm* of etiologic factors and the types of concepts required to understand these factors. One school emphasized single agents (usual microbial) as *the* cause of disease, as exemplified by Charles Chapin's influential text. *The Sources and Modes of Infection* [73, 74]. Others, like Major Greenwood, used the imagery of "seed, soil, and some type of husbandry" [75] to argue that the host and environment, not just the agent, needed to be taken into account; this type of reasoning was clearly evident in Wade Hampton Frost's trenchant analyses of epidemic disease [76]. A third looked to basic features of the economy for clues about mass patterns of disease, as exemplified by Goldberger, Wheeler, and Sydenstricker's classic studies on the conjoint social and biologic etiology of pellagra [77–79]. In addition to proving pellagra was a dietary deficiency disease, they elucidated the link between pellagra's prevalence in the South and the region's cash-crop economy: whenever the cotton market crashed, the subsequent prevalence of pellagra inevitably rose.

To some degree, the prominence of one over another of these approaches paralleled gains and setbacks in the knowledge of related fields in the natural and social sciences, e.g. medicine, virology, toxicology, genetics, anthropology, and political economy. As important—if not more so—were institutional decisions regarding what types of research should be supported [28, 54, 74] and how public health researchers should be trained [28, 74, 80]. The influential Welch-Rose report of 1915 that led to the first U.S. school of public health being founded at Johns Hopkins University, for example, strongly favored its biomedical approach, as compared to the more sociological program proposed at the time by Columbia University [74]. The devastating 1918

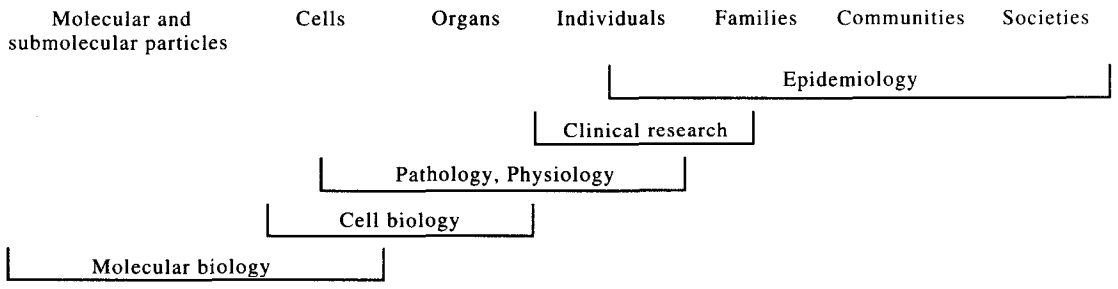


Fig. 3. Stallones' figure depicting biomedical disciplines "along a scale of biological organization" [15, p. 70]. Reprinted with permission from the publisher.

influenza pandemic and growing concern about diseases like cancer, however, impelled noted epidemiologists like Greenwood [75] and Sydenstricker [80] to question exclusive reliance upon the 'germ theory' as a framework for understanding health. They argued instead that the study and prevention of 'crowd' or 'mass' diseases—whether infectious or chronic—would require social as well as biologic reasoning.

In light of this history, it is not surprising that the present-day inattention to epidemiologic theory and preoccupation with epidemiologic methods has provoked several different types of concerned responses within the discipline. As noted earlier, several senior U.S. epidemiologists have called for renewed attention to epidemiologic theory [3, 4, 18–21, 81–84]. Some, most notably Cassel [82, 83] and Syme [84], have sought to develop psychosocial theories of disease causation to supplement or counter more biomedical approaches. Others, like Stallones [15], have attempted to bolster the theoretical underpinnings of the multifactorial framework by depicting the range of factors that epidemiologists should consider when constructing causal 'webs,' ranging from the subcellular to the societal and the microscopic to the macrosystem (Fig. 3).

Criticizing these approaches, Vandenbroucke *et al.* [85, 86] have warned against epidemiologists' tendency of invoking a vaguely-defined but often infinitely complex 'environment' to 'explain' variation in disease rates. They contend that this approach, combined with epidemiologists' widespread practice of treating disease mechanisms as a 'black box,' will soon render the discipline incapable of making meaningful contributions to understanding disease etiology. Claiming that most 'environmental' theories are but 20th-century versions of outdated 'miasmatic' arguments, Vandenbroucke has strongly argued that the future of epidemiology lies in the fast moving search for genetic markers [85], as brought about by spectacular advances in biotechnology and epitomized by the Human Genome Project [87]. Similarly, Kuller has asserted that claims about 'multifactorial etiology' may be more a sign of ignorance than insight, and has urged epidemiologists "to rethink a parsimonious approach to the etiology of disease and again to consider that each disease is caused by one agent and to evaluate confounding in terms of host, agent, and modes of transmission" [81, p. 374]. Others, however, have argued that a return to 'single agent' theories (and especially those favoring exclusively genetic explanations) will only serve to recreate the errors made in the early period of widespread enthusiasm for the germ theory of disease, which resulted in artificially narrowing the scope of etiologic explanations considered and public health interventions employed and which may also have retarded the development of research on diseases beyond its ken (e.g. cancer and cardiovascular disease) [4, 18, 54, 88–91].

To date, however, few epidemiologists have attempted to develop systematic and theoretically rigorous alternatives to the 'web of causation.' One such effort has emphasized the role of evolution, adaptation, and the 'man-made' environment in determining current and historically-changing population patterns of health and disease [64, 92–95]. This theory is best summarized in a recent book entitled *On the Origins of Human Disease* [92], written by Thomas McKeown, a well-known British epidemiologist. At its cores are three claims:

(1) for most of our history as a species, humans have lived under, and are biologically best adapted to, 'Stone Age' conditions, exemplified by the hunter/gatherer mode of existence, especially its diet and level of physical activity
(2) the transition to agriculture (*ca* 10,000 years ago) brought about vast changes in human disease, due to the concentration of larger numbers of people in more permanent settlements and also to the domestication of animals, both of which (in conjunction with

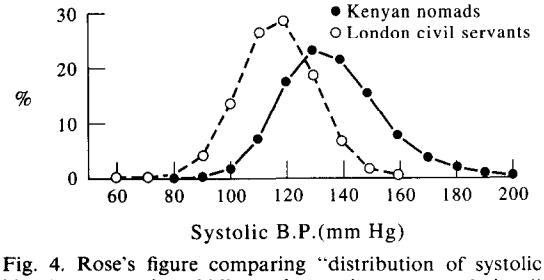


Fig. 4. Rose's figure comparing "distribution of systolic blood pressure in middle-aged men in two populations" [57, p. 33]. Reprinted with permission from the publisher.

periodic famines) enabled numerous infectious diseases to flourish

(3) the transition to industrial society (*ca* 300 years ago) led to a decline in infectious diseases and to a rise in non-communicable diseases, due to improved nutrition and increased exposure to "conditions for which we are genetically ill-equipped" [92, p. 153], e.g. refined diets, cigarette smoke, sedentary 'lifestyles,' industrial toxins, and other 'man-made' hazards

Building on these premises, McKeown argues that human diseases can best be understood and prevented if divided into three fundamental etiologic groups, defined by disease origins rather than disease mechanisms or afflicted organs [92, p. 222]. These are: (1) 'prenatal diseases' (determined at fertilization or during the course of pregnancy) [92, p. 99], (2) 'diseases of poverty' (due to deficiencies and hazards linked to the 'natural' environment, e.g. poor nutrition and poor sanitation) [92, p. 138], and (3) 'diseases of affluence' (due to maladaptation to changes in living conditions brought about by industrialization) [92, p. 140], which he nonetheless concedes now disproportionately affect the poor in both industrialized and impoverished countries [92, p. 94].

McKeown's schema is thus a far cry from MacMahon *et al.*'s [2] 'web' of assorted risk factors. Focusing on the health effects of agriculture, urbanization, and industrialization, McKeown simultaneously directs us to consider broad population shifts in disease occurrence and whether the proximate causes of a particular disease operate before or after birth and if they exert their effects via depriving the body of basic biological requirements or by exposing the body to types or levels of substances, hazards, or conditions not anticipated through 'natural selection.' Even so, McKeown's theory—like that underlying the 'web'—fails to address an important set of etiologic questions with important implications for disease prevention policies: namely, whether *all* sectors of society are equally responsible for shaping the human environment (for good or for bad), and why it is that specific sectors have not equally benefited from or been harmed by these changes [96]. Instead, the responsible parties are absent actors in a scenario where 'natural selection' plays the defining role.

In direct contrast, the other school seeking to develop epidemiologic theory has focused on those issues downplayed by McKeown: specifically, the social and political determinants of health. Building on the work of 19th and earlier 20th century public health researchers and advocates [4, 14, 26–33, 65, 66, 72, 77–80], these epidemiologists [3, 4, 15–19, 56, 62, 97–125]—along with other public health scholars and activists [58–61, 74, 78, 126–130]—have sought to develop and test the theory that past and present population patterns of health and disease primarily stem from the social organization of society, and especially its economic and social activites and inequalities.

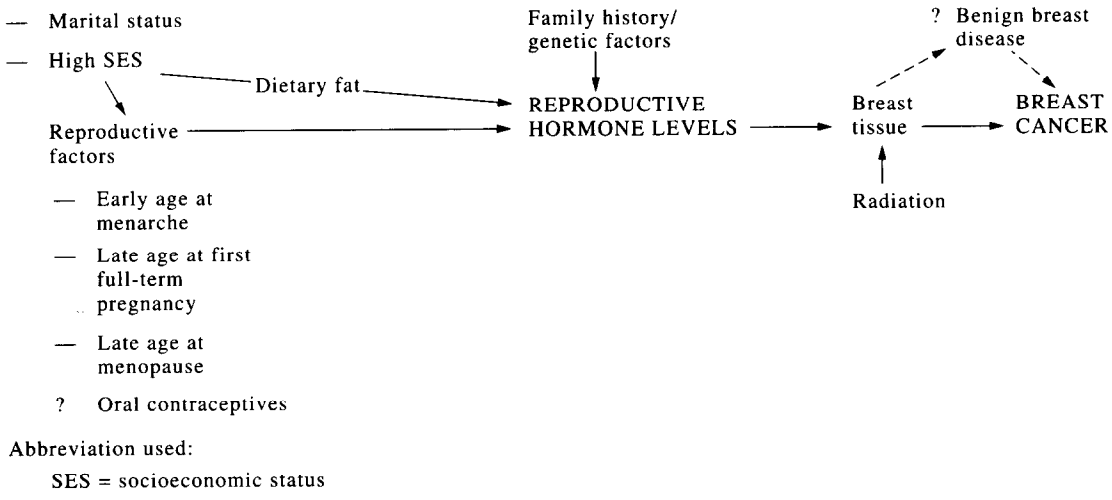
Promoting such concepts as 'the political economy of health' [58] and 'the social production of disease' [54, 56, 59–62, 97, 98, 104, 105, 123–130], proponents of this alternative view reject uniform assertions about 'man's' 'maladaptation' [92, p. 95]. Instead, they typically seek etiologic clues through comparing the health status of social groups that differentially benefit from or are harmed by the status quo, such as employers/employees, men/women, whites/people of color, heterosexuals/homosexuals, and inhabitants of economically developed/underdeveloped regions. The essential claim is that understanding patterns of health and disease *among* persons in these groups requires viewing these patterns as the consequence of the social relationships *between* the specified groups, with these relationships expressed through people's everyday living and working conditions, including daily interactions with others [98].

This perspective fundamentally differs from a stance which sees group patterns simply as the sum of individual traits and choices. It instead asks how individuals' membership in a society's historically-forged constituent groups shapes their particular health status, and how the health status of these groups in turn reflects their position within the larger society's social structure [44]. It further implies that changing these population patterns requires explicitly studying and addressing their political, economic, and ideologic determinants. According to this view, social inequalities in health are the defining problem of the discipline of epidemiology. The litmus test of any epidemiologic theory of disease causation thus is whether it can explain past and present social inequalities in health.

Yet, despite their emphasis on historical determinants of disease, few advocates of this analytic trend have extended the range of historical processes to include, as McKeown has done, the influence of biologic evolution, growth, and development on human disease processes [98]. Illustrating one such attempt is Fig. 5, which endeavors to contrast a biomedical approach to understanding breast cancer etiology to one that incorporates developmental biology within the framework of the social production of disease [122]. The first model (Fig. 5a) displays the current predominant conceptualization, which stresses the role of hormonal and genetic factors. The second model (Fig. 5b) broadens the framework of exposure and susceptibility to consider how social factors simultaneously: (1) mediate the growth and development of breast tissue (by affecting the timing of reproductive events), and (2) determine the type and timing of exposure to exogenous agents that may contribute to breast cancer risk.

As suggested by the rudimentary nature of Fig. 5(b), however, epidemiology still lacks a theoretical framework that truly integrates social and

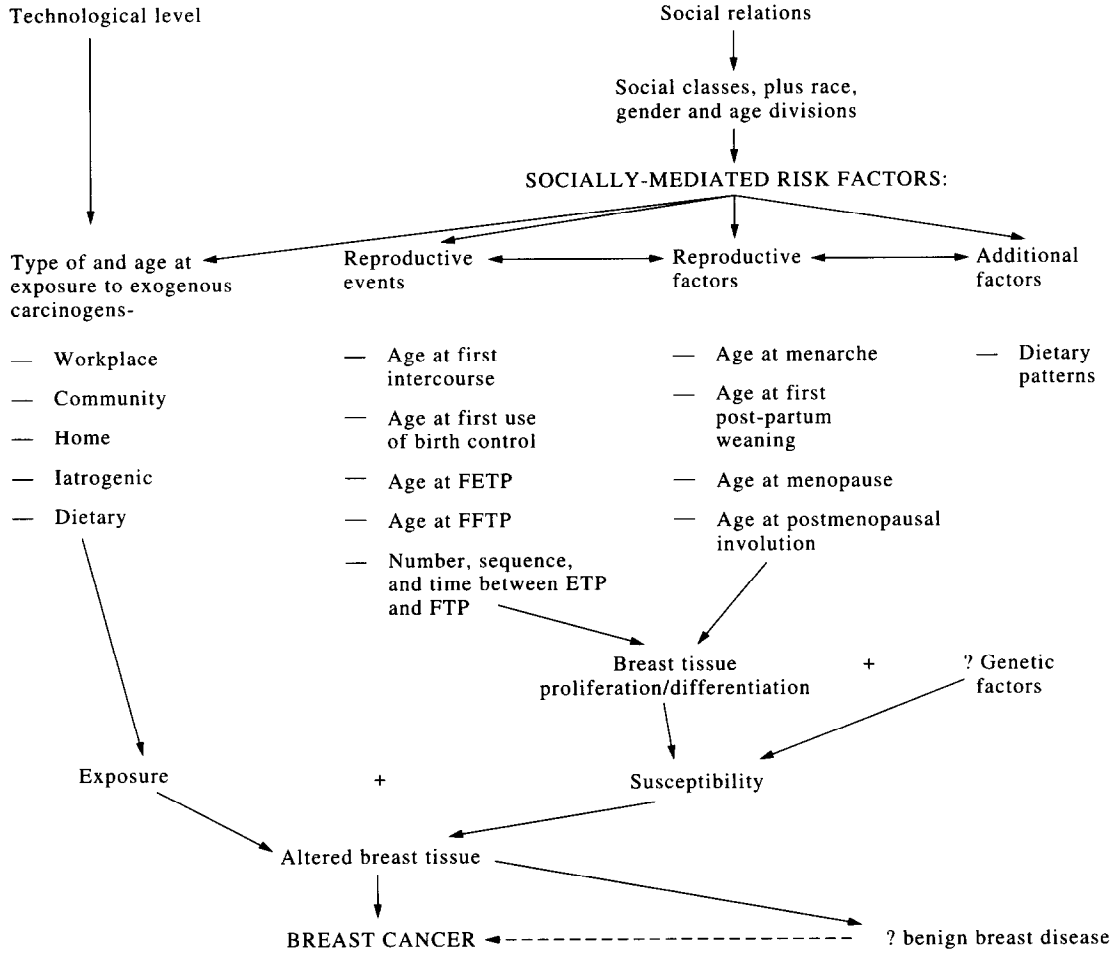
(a)



Abbreviation used:

SES = socioeconomic status

(b)



Abbreviations used:

ETP = early-terminated pregnancy

FETP = first early-terminated pregnancy

FFTP = first full-term pregnancy

FTP = full-term pregnancy

Fig. 5. Contrast of a biomedical vs social production of disease model of breast cancer pathogenesis [122, pp. 207, 209]. Reprinted with permission from the publisher.

biologic understandings of health, disease, and well-being [98]. In other words, what I would call an 'ecosocial' epidemiologic theory—one that embraces population-level thinking and rejects the underlying assumptions of biomedical individualism without discarding biology—has yet to be conceived.

MODELS, METAPHORS, AND THE DEVELOPMENT OF EPIDEMIOLOGIC THEORY: TOWARDS AN ECOSOCIAL FRAMEWORK

(1) The importance of metaphors for cognition and theory

Among the many obstacles to developing an ecosocial framework is one that has received relatively little attention: the absence of a metaphor that can succinctly capture the essence of this alternative view. Nothing comparable to the 'web' exists.

The importance of metaphors for scientific thought, though rarely acknowledged, requires greater recognition [22, 131–135]. Often viewed as purely evocative linguistic devices, metaphors in fact play an essential role in cognition [136]. Like theories, metaphors attempt to produce new understanding through constructing novel connections between seemingly disparate phenomena and/or concepts, thereby enabling the 'unknown' to be comprehended through reference to the 'known' [132, 134, 136]. To gain these insights, metaphors and theories both rely upon mental imagery; indeed, the Greek root of 'theory' means 'to view' [137, p. 3284] while that of 'metaphor' means 'to overlay meaning' [137, p.1781].

Although related, metaphors and theories are also different. Whereas theorizing implies delineating structured and testable relationships among selected entities or ideas [11, 12, 22–25], metaphors challenge us to experience one phenomenon or concept in terms of another [136, p. 5]. Through their deliberate dissonance (which distinguishes them from mere analogies [134]), metaphors jar us to 'see' relationships that otherwise would be opaque [131–135]. Less constrained than formal theories, metaphors often creatively express intuitive ideas about 'how the world works.' The metaphor of the 'web' has certainly played this role in epidemiology, as have the metaphors of 'production' and 'reproduction' for those who think in terms of the social production of disease [54, 56, 62, 105, 125].

Opening up new ways of seeing the world, metaphors can both help and hinder understanding. Consider, for example, the metaphor of 'man-as-machine,' as developed by Descartes. On the one hand, this metaphor led to new scientific knowledge by spurring non-vitalistic explanations of human biology [63, 134]. At the same time, its inherent mind/body dualism and reductionism has blocked and continues to hinder research on psychosocial determinants of health [45, 63, 135]. The power of metaphors is thus fickle: they can simultaneously free and constrain thought—and yet without them, it is unlikely that new connections will be drawn.

(2) Towards an ecosocial metaphor for epidemiology

Perhaps one step toward developing an ecosocial metaphor would be augmenting the metaphor of the 'web' with two 'spiders': one social, one biologic. They would certainly reintroduce the concepts of history and agency, and would emphasize the importance of considering the origins of both social and biologic determinants of disease. Even so, the imagery of the 'spiders' may be too simplistic, and may fail to do justice to the complex origin and nature of the 'spiders' themselves. It is of little help to posit that health and disease are socially produced within evolving and socially-conditioned biologic parameters without offering insight into why and how this occurs; reducing the 'spiders' to a new form of 'black box' would only reinforce existing limitations. Nor would introducing the 'spiders' necessarily resolve the 'web's' embodiment of a biomedical and individualistic worldview. The 'web' never was intended to and does not jar epidemiologists from the long-established practice of viewing population patterns of disease as simply the sum of individual cases; it is far from obvious that adding the 'spiders' would address this fundamental problem.

As an alternative, the closest image that comes to mind stems from marrying the metaphor of the continually-constructed 'scaffolding' of society that different social groups daily seek to reinforce or alter [136] to that of the ever-growing 'bush' of evolution [138], together defining the potential and constraints of human life. This intertwining ensemble must be understood to exist at every level, sub-cellular to societal, repeating indefinitely, like a fractal object (Fig. 6) [139–142]. Different epidemiologic profiles at the population-level would accordingly be seen as reflecting the interlinked and diverse patterns of exposure and susceptibility that are brought into play by the dynamic intertwining of these changing forms. It is an image that does *not* permit the cleavage of the social from the biologic, and the biologic from the social. It is an image that does not obscure agency. And it is an image that embraces history rather than hides it from view.

This image insists that understanding societal patterns of health and disease requires acknowledging the inextricable and ongoing intermingling—at all levels—of the social and the biologic. Through its fractal nature, it does not allow the individual to become separated from society, nor detract from people's irreducible individuality. A given branch of this intertwining structure can thus be seen as representing one set of possible epidemiologic profiles produced at a particular time by a particular combination of social structures, cultural norms, ecologic milieu, and genetic variability and similarity (among humans and among other organisms in the region). At a greater level of detail, particular groups—linked

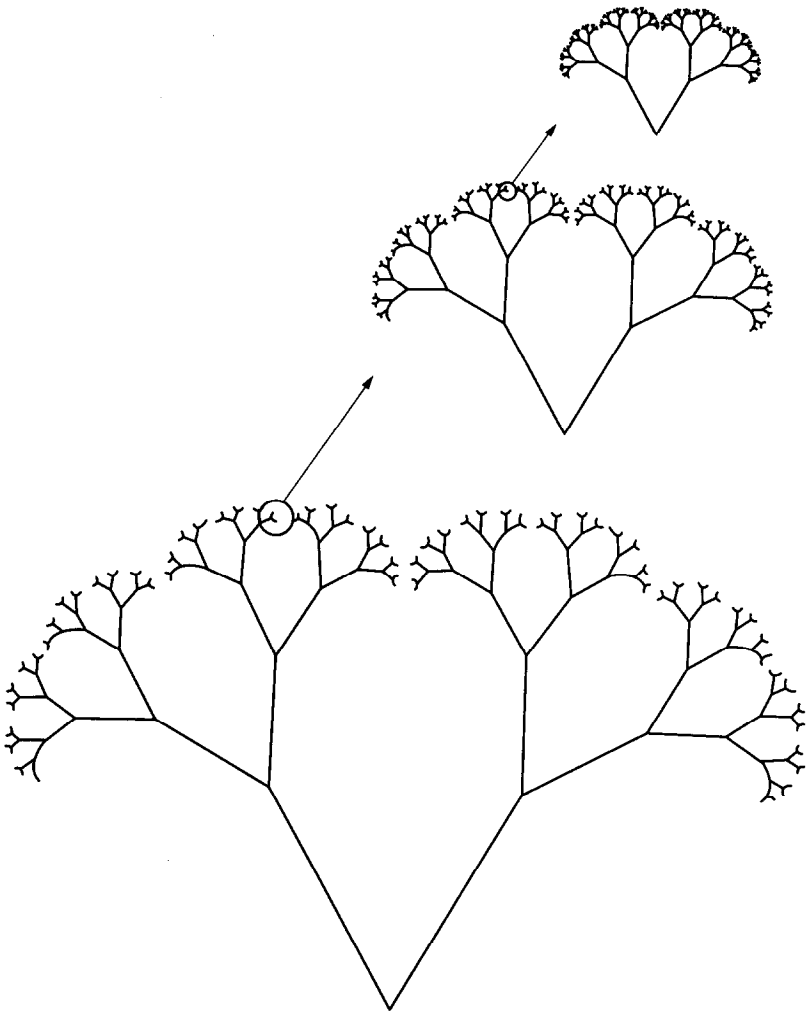


Fig. 6. Computer-generated fractal structure, illustrating self-similarity on multiple scales [141, p. 1807]. Reprinted with permission from the publisher.

by their membership in socially-defined categories—embody these characteristics, thus acquiring, in the memorable phrase of Cristina Laurell, their distinct 'class physiognomies" [105, p. 1184].

At an even greater level of detail, the particularities of health and disease among individuals become apparent, reflecting yet again the interplay of these same social and biologic influences. And, moving to yet another level, these same sets of influences can be traced through their relationship to the normal functioning and disturbances of organ systems, cellular growth and differentiation, and metabolic processes. Highlighting the inherent links between levels, this image thus *requires* considering multiple levels when seeking to understand patterns at any given level, and likewise highlights the need to frame questions broadly, regardless of the level at which any particular investigation is conducted.

The proposed image accordingly begins to offer a way to conceptualize the processes producing different epidemiologic profiles, both within and across specific societies, at a given time and over time, at a given location and across locales. It does not mandate a singular answer, applicable for all diseases, but does specify a range of questions—about social structure, cultural norms, ecologic milieu, and genetic variability—that must be systematically addressed when analyzing any specific situations. It requires population-thinking in its study of individuals, and recognition of individual variability (and similarity) in its study of populations. Beyond this, it directs epidemiologists to think about individuals in the context of their everyday lives, as shaped by their interwined histories—as members of a particular society, and as biological creatures who grow, develop, interact, and age [17, 98, 105, 112, 123]. It thus encourages asking whether the factors or processes pertaining to exposure or susceptibility are exclusively or conjointly physical, biological, or social in nature, and also exogenous, endogenous, or both. Similarly, it directs attention to whether—and if so, why and how—exposure and susceptibility vary over the course of people's lives. At issue may be social conventions regarding who does what at which age, historical

changes in living and working conditions and also interactions with other species, as well as biologic changes in tissues, organs, and systems that bear the imprint of evolutionary selection and contingent alterations [24, 112, 123, 138].

This image accordingly suggests an orientation similar to that implied by evolutionary theories, which do not specify or predict the exact fate of particular species (or groups of species) and the organisms of which they are composed, but do specify a range of questions and a set of processes that must be considered when attempting to understand their specific dynamics (extending from origin to extinction) [24, 131, 138]. And, at the same time, it suggests an orientation similar to that implied by sociological theories, which likewise do not specify or predict the exact fate of particular social groups (or societies) and the individuals of which they are composed, but similarly specify a range of questions and a set of processes that must be considered when attempting to understand their social features and behaviors [42, 61, 82]. And like both evolutionary and sociological theories, the orientation implied by this image rejects a teleological stance (implying change in a definite direction toward a specified goal), but does not do so at the expense of neglecting the critical role of human agency and accountability—at the societal as well as individual level—in shaping population patterns of health and disease.

Encouraging a social and ecologic point of view, this image also serves as a reminder that people are but one of the species that populates our planet; it thus implies that the health of all organisms is interconnected. And at the same time, by situating social groups and individuals—which, by definition, include epidemiologists—in the context of particular societies at particular times, it demands that epidemiologists consider how their social position affects the knowledge they desire and that which they produce. As such, it directs attention not only to the social production of disease, but also to the social production of science—that is, how a society's predominant view of the world and the position of scientists in this society influence the theories they develop, the research questions they ask, the data they collect, the analytic methods they employ, and the ways they interpret and report their results [45, 54, 63, 72, 74, 99, 143–145]. This stance acknowledges that science is at one—and indivisibly—objective and partisan [99]. And, by accepting these *inherent* features of science—the objectivity that arises out of commonly employed methodologies used by different investigators in comparable situation *and* the subjectivity that arises out of the values and worldviews that promote some questions and silence others—this image encourages epidemiologists (and other scientists) to avoid the trap of conflating scientific assumptions with reality and to embrace a humility that counters the apparent hubris which often accompanies the biomedical approach [44, 45, 61, 117].

(3) What problems could an ecosocial framework address?

One reason for pursuing the development of an ecosocial metaphor and theory premised upon this initial image is that such a framework could offer a helpful way of thinking about several striking features of epidemiologic data and research in the United States today. One is the stunning and long-standing acceptance of the absence of social class data in U.S. vital statistics and most disease registries [99, 116], along with paucity of good data for most minority racial/ethnic groups [99, 146]. These gaps contrast sharply with the ever expanding knowledge about epidemiologic methods (Table 1), genetic susceptibility [87], and 'lifestyle' risk factors [146]. It is also both striking and telling that the U.S. National Institutes of Health recently resorted to issuing directives requiring epidemiologists and clinical researchers to include women [147] and minorities [148] in their study populations. This change occurred only because of the growing political influence of women and people of color and their insistence upon health research being relevant to their lives. That groups outside of epidemiology triggered these changes speaks volumes about the provincialism and elitism sanctioned by the current framework guiding research today, one developed chiefly by privileged persons (mainly white, mainly men) trained in the biomedical sciences.

Such a framework could also spur greater precision in epidemiologic concepts about etiology than presently exists. By challenging the biomedical individualism underlying the construction of the epidemiologic triads of 'race, age, and sex' and of 'time, place, and person,' it would make clear that these phenomena are neither simply 'natural' nor—in the case of personal characteristics—individually innate. It would promote recognition of the fact that 'race' is a spurious biologic concept [63, 98, 115, 149] and would instead direct attention to how racism affects health—overall, and of people on both sides of the color line [98, 109–116, 149]. An ecosocial framework would likewise require considering how the process of aging cannot be separated from the social conditions in which people are born, live, work and retire [112, 123]. It would not confuse biological 'sex' with culturally-determined 'gender' and would promote questions about how the health of not only women but men is shaped by gender-based (and often sexist) assumptions [71, 98, 109, 117–123]. 'Social class' would be considered a fundamental category [56, 58–61, 97–108, 117, 123], and the term 'person' would not be used when what really is meant is 'social group.' Such a framework would thus demand that epidemiologists eschew terms like 'special populations'—now routinely used by U.S. federal health agencies to describe women, the poor, and people

of color [146]—and would instead directly expose what makes these populations 'special': their enforced marginalization from positions of power, coupled with the assumption that white, economically-secure men are allegedly the 'norm.'

An ecosocial approach would also encourage reformulating, if not rejecting, other loosely-defined terms so prevalent in the epidemiologic literature—such as 'lifestyle' [150] and 'environment' [151]—so as to end the practice of obscuring or misclassifying agency. It is more than a misnomer, for example, to imply that it is simply a persons's freely-chosen 'lifestyle' to eat poorly when supermarkets have fled the neighborhood, or to have a child early or late in life or not at all, without considering economic circumstances and job demands [44, 54, 58–61, 97, 98]. An ecosocial framework would thus require situating the social context of such health 'behaviors' if they are to be comprehended, let alone changed. And, with regard to prevention, it would encourage research on not only those factors deemed amenable to intervention through the medical care system, the work of public health departments, or the effort of solo individuals, but also on the broader determinants of health that can be changed only through more widespread social action.

An ecosocial approach would thus challenge current definitions of the 'environment' as that which is 'exogenous to the organism' (cf. *A Dictionary of Epidemiology*) [151], as well as the uses of such allegedly complementary phrases as the 'natural' and 'social' 'environment.' In rejecting this analogy, it would make clear that social conditions are not 'natural' but are constructed by people, with purpose in mind and accountability an option. Social conditions are conceptually and categorically distinct from the 'natural environment,' that is, the interplay of ecologies and global geologic and climactic forces, which humans can effect (and even destroy) but which we certainly have not created. Confounding of concepts can muddy analyses as much as confounding of risk factors; epidemiologists must be as rigorous about categories of thought as approaches to analyzing data.

Additionally, an ecosocial framework would challenge the current rigid distinction between individual- and group-level analyses. Directing attention to the health effects of collective phenomena that cannot be reduced to individual attributes [98, 152, 153], it captures why what has been termed the 'individualistic fallacy' [152, 154]—i.e. the assumption that individual-level data are sufficient to explain group-level phenomena—is as much of a liability as the 'ecologic fallacy' [152, 154, 155], which results from confounding introduced by the grouping process. Tying together the macro and the micro, it would encourage use of what has been termed 'contextual' or 'multilevel' analysis [98, 152, 153, 156–158], which combines individual- and group-level data in a clearly specified and theoretically justified manner. This approach has been used in other fields [156–158], and only recently has been explicitly introduced into the epidemiologic repertoire [152, 153], e.g. considering the conjoint effects of neighborhood- and individual-level social class upon health [152]. An ecosocial framework thus has the potential to raise new conceptual and methodologic questions about the shaping of human health, much as the 'evolutionary synthesis' of the 1940s generated considerable intellectual ferment and promoted new discoveries by integrating genetic and species-level approaches to understanding and studying biological evolution [24].

CONSTRUCTING EPIDEMIOLOGIC THEORY: A NECESSARY AND VITAL CHALLENGE

The field of epidemiology today suffers from the absence of not only a clearly articulated and comprehensive epidemiologic theory, but, it seems, even the awareness that it lacks such a theory. The science instead is taught and viewed as a collection of methods to be applied to particular problems involving human diseases and health [3, 4, 15, 20, 21].

To counter this state of affairs, the image proposed in this paper is intended to spur discussion about important aspects of epidemiology's purpose and domain, as the science that seeks to explain and generate knowledge to improve population patterns of health, disease, and well-being. Attempting to advance an ecosocial framework for the development of epidemiologic theory, this image makes clear that although the biologic may set the basis for the existence of humans and hence our social life, it is this social life that sets the path along which the biologic may flourish—or wilt. As such, it emphasizes why epidemiologists must look first and foremost to the link between social divisions and disease to understand etiology and to improve the public's health, and in doing so exposes the incomplete and biased slant of epidemiologic theories reliant upon a biomedical and individualistic world-view.

Despite its appeal, this image remains only that—an image. It is not a developed metaphor. Nor is it a substitute for a well-articulated ecosocial theory of epidemiology. And it remains open to question whether this particular image could help give rise to a more concise metaphor or provide useful debate about the current status of epidemiologic theory. The essential point, however, is that the 'multivariate' framework so widespread in epidemiology today, as expressed by metaphor of the 'web of causation,' represents an approach to epidemiologic theory that is deeply flawed. To forge a better theory, it may still be worthwhile to search for the 'spider'—whether one or many—but this can be determined only if this task is pursued. Otherwise, like Mr Zuckerman in E. B. White's classic tale, *Charlotte's Web* [1], epidemiologists will continue to mistake Wilbur the pig for the miracle of the web and the work of the spider. We will thereby miss the full story.

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REFERENCES

1. White E. B. *Charlotte's Web.* HarperCollins, New York, 1952.
2. MacMahon B., Pugh T. F. and Ipsen J. *Epidemiologic methods.* Little Brown and Co., Boston, 1960.
3. Susser M. Epidemiology in the United States after World War II: the evolution of technique. *Epidemiol. Rev.* **7,** 147–177, 1985.
4. Buck C., Llopis A., Najera E. and Terris M. Discussion. In *The Challenge of Epidemiology: Issues and Selected Readings* (Edited by Buck C., Llopis A., Najera E. and Terris M.), pp. 149–153. PAHO/WHO, Washington, DC, 1988.
5. Greenland S. (Ed.) *Evolution of Epidemiologic Ideas: Annotated Readings on Concepts and Methods.* Epidemiology Resources, Chestnut Hill, MA, 1987.
6. Susser M. *Causal Thinking in the Health Sciences: Concepts and Strategies of Epidemiology.* Oxford University Press, New York, 1987.
7. Miettinin O. S. *Theoretical Epidemiology: Principles of Occurrence Research in Medicine.* Wiley, New York, 1985.
8. Rothman K. *Modern Epidemiology.* Little & Brown, Boston, 1986.
9. Rothman K. J. (ed.) *Causal Inference.* Epidemiology Resources, Chestnut Hill, MA, 1988.
10. Susser M. What is a cause and how do we know it? A grammer for a pragmatic epidemiology. *Am. J. Epidemiol.* **133,** 635–648, 1991.
11. Ziman J. *Reliable Knowledge: an Exploration of the Grounds for Belief in Science.* Cambridge University Press, Cambridge, 1978.
12. Wisdom J. *Challengeability in Modern Science.* Avebury Press, Aldershot, 1987.
13. Cassel J. M. Potentialities and limitations of epidemiology. In *Health and the Community: Readings in the Philosophy and Sciences of Public Health* (Edited by Katz A. and Felton J.), pp. 432–435. The Free Press, New York, 1965.
14. Terris M. The epidemiological tradition. *Pub. Hlth Rep.* **94,** 203–209, 1979.
15. Stallones R. A. To advance epidemiology. *Ann. Rev. publ. Hlth* **1,** 69–82, 1980.
16. Rose G. *The Strategy of Preventive Medicine.* Oxford University Press, Oxford, 1992.
17. Krieger N. *Race, Class, and Health: Studies of Breast Cancer and Hypertension.* Dissertation, University of California at Berkeley, pp. 233–266, 1989.
18. Susser M. Epidemiology today: 'a thought-tormented world.' *Int. J. Epidemiol.* **18,** 481–488, 1989.
19. Terris M. The Society for Epidemiologic Research (SER) and the future of epidemiology. *Am. J. Epidemiol.* **136,** 909–915, 1992.
20. Lilienfeld A. M. and Lilienfeld D. E. Epidemiology and the public health movement: a historical perspective. *J. publ. Hlth Policy* **3,** 140–149, 1982.
21. Kuller L. H. Epidemiology is the study of 'epidemics' and their prevention. *Am. J. Epidemiol.* **134,** 1051–1056, 1991.
22. Fleck L. *Genesis and Development of a Scientific Fact.* University of Chicago Press, Chicago (1935).
23. Kuhn T. S. *The Structure of Scientific Revolutions,* 2nd edn. enlarged. University of Chicago Press, Chicago (1962).
24. Mayr E. *The Growth of Biological Thought: Diversity, Evolution, and Inheritance.* The Belknap Press of Harvard University Press, Cambridge, MA, 1982.
25. Holton G. *The Thematic Origins of Scientific Thought: From Kepler to Einstein,* revised edn. Harvard University Press, Cambridge, MA, 1988.
26. Morris J. N. *Uses of Epidemiology.* E & S Livingston, Edinburgh, 1957.
27. Taylor I. and Knowelden J. *Principles of Epidemiology.* J & A Churchill, London, 1957.
28. Fee E. and Acheson R. M. (Eds) *A History of Education in Public Health: Health that Mocks the Doctors' Rules.* Oxford University Press, Oxford, 1991.
29. Galdston I. (Ed.) *The Epidemiology of Health.* Health Education Council, New York Academy of Medicine, New York, 1953.
30. Galdston I. (Ed.) *Beyond the Germ Theory: the Role of Deprivation and Stress in Health and Disease.* Health Education Council, New York Academy of Medicine, New York, 1954.
31. Gordon J. E. Epidemiology—old and new. *J. Michigan St. Med. Society* **49,** 194–199, 1950.
32. Rosen G. What is social medicine?—a genetic analysis of the concept (1947). In *From Medical Police to Social Medicine.* (Edited by Rosen G.), pp. 60–119. Science History Publications, New York, 1974.
33. Galdston I. (Ed.) *Social Medicine: its Derivatives and Objectives.* The Commonwealth Fund, New York, 1949.
34. Cornfield J. A method of estimating comparative rates from clinical data: applications to cancer of the lung, breast, and cervix. *J. natn. Cancer Inst.* **11,** 1269–1275, 1951.
35. Sayre A. *Rosalind Franklin and DNA.* W. W. Norton & Co., New York, 1975.
36. Nordling C. O. A new theory on the cancer-inducing mechanism. *Br. J. Cancer* **7,** 68–72, 1953.
37. Skeet R. G. The impact of the computer on the cancer registry. *Gann Monograph on Cancer Res.* **33,** 89–95, 1987.
38. Anderson M. J. *The American Census: a Social History.* Yale University Press, New Haven, CT, 1988.
39. Vallis F. (Ed.) *Ribbons of Time,* p. 115. Weidenfeld & Nicolson, New York, 1988.
40. Zinn H. *A People's History of the United States.* Harper Colophon, New York, 1980.
41. Blumberg P. *Inequality in an Age of Decline.* Oxford University Press, New York, 1980.
42. Bell D. *The Social Sciences Since the Second World War.* Transaction Books, New Brunswick, NJ, 1982.
43. Branch T. *Parting the Waters: America in the King Years, 1954–1963.* Simon & Shuster, New York, 1988.
44. Fee E. and Krieger N. Understanding AIDS: historial interpretations and the limitations of biomedical individualism. *Am. J. publ. Hlth* **83,** 1477–1486, 1993.
45. Lock M. and Gordon D. (Eds) *Biomedicine Examined.* Kluwer Academic, Dordrecht, 1988.
46. Watts A. *Tao: The Watercourse Way.* Pantheon Books, New York, 1975.
47. Bannerman R. H., Burton J. and Wen-Chieh C. (Eds) *Traditional Medicine and Health Care Coverage: a Reader for Health Administrators and Practitioners.* World Health Organization, Geneva, 1983.
48. Bernal J. D. *Science in History,* Vol. 1–4. MIT Press, Cambridge, MA, (1954).
49. Berman M. *The Disenchantment of the World.* Cornell University Press, Ithaca, NY, 1981.
50. Weiss N. S. and Liff J. M. Accounting for the multicausal nature of disease in the design and analysis of epidemiologic studies. *Am. J. Epidemiol.* **117,** 14–18, 1983.
51. Mausner J. S. and Bahn A. K. *Epidemiology: an Introductory Text.* Saunders, Philadelphia, 1974.
52. Friedman G. *Primer of Epidemiology.* McGraw-Hill, New York, 1974.

53. Duncan D. F., Gold R. J., Basch C. E. and Markellis V. C. *Epidemiology: Basis for Disease Prevention and Health Promotion.* Macmillan, New York, 1988.
54. Tesh S. *Hidden Arguments: Political Ideology and Disease Prevention Policy.* Rutgers University Press, New Brunswick, NJ, 1988.
55. Rothman K. J. Causes. *Am. J. Epidemiol.* **104,** 587–592, 1976.
56. Breilh J. *Epidemiologia Economia Medicina y Politica.* Distribuciones Fontamara, Mexico City, (1979)
57. Rose G. Sick individuals and sick populations. *Int. J. Epidemiol.* **14,** 32–38, 1985.
58. Doyal L. *The Political Economy of Health.* Pluto Press, London, 1979.
59. Navarro V. *Crisis, Health, and Medicine: a Social Critique.* Tavistock, New York, 1986.
60. Waitzkin H. *The Second Sickness: Contradictions of Capitalist Health Care.* The Free Press, New York, 1983.
61. Conrad P. and Kern R. (Eds) *The Sociology of Health and Illness: Critical Perspectives.* St Martin's Press, New York, 1981.
62. Krieger N. and Bassett M. The health of black folk: disease, class and ideology in science. *Monthly Rev.* **38,** 74–85, 1986.
63. Lewontin R. C., Rose S. and Kamin L. J. *Not in our Genes: Biology, Ideology, and Human Nature.* Pantheon Books, New York, 1984.
64. Dubos R. *Man Adapting.* Yale University Press, New Haven, CT, 1965.
65. Rosen G. *A History of Public Health.* MD Publications, New York, 1958.
66. Coleman W. *Death is a Social Disease: Public Health and Political Economy in Early Industrial France.* University of Wisconsin Press, Madison, WI, 1982.
67. Ackerknecht E. H. Anticontagionism between 1821 and 1867. *Bull. Hist. Med.* **22,** 562–593, 1948.
68. Pelling M. *Cholera, Fever, and English Medicine 1825–1865.* Oxford University Press, Oxford, 1978.
69. Rosenberg C. D. *The Cholera Years: the United States in 1832, 1849, and 1866.* University of Chicago Press, Chicago (1962).
70. Rosenkrantz B. G. *Public Health and the State: Changing Views in Massachusetts, 1842–1936.* Harvard University Press, Cambridge, MA, 1972.
71. Apple R. D. (Ed.) *Women, Health & Medicine in America: a Historical Handbook.* Rutgers University Press, New Brunswick, NJ, 1992.
72. Krieger N. Shades of difference: theoretical underpinnings of the medical controversy on black/white differences in the United States, 1830–1870. *Int. J. Hlth Services* **17,** 259–278, 1987.
73. Chapin C. V. *The Sources and Modes of Infection.* Wiley, New York, 1910.
74. Fee E. *Disease and Discovery: a History of the Johns Hopkins School of Hygiene and Public Health, 1916–1939.* Johns Hopkins University Press, Baltimore, MD, 1987.
75. Greenwood M. *Epidemics and Crowd Diseases: An Introduction to the Study of Epidemiology*, p. 359. Williams & Northgate, London, 1935.
76. Frost W. H. Epidemiology. In *Papers of Wade Hampton Frost, MD: a Contribution to Epidemiologic Method* (Edited by Maxcy K. F.), pp. 439–542. The Commonwealth Fund, New York, 1941.
77. Goldberger J., Wheeler G. A. and Sydenstricker E. A study of the relation of family income and other economic factors to pellagra incidence in seven cotton-mill villages of South Carolina in 1916. *Publ. Hlth Rep.* **35,** 2673–2714, 1920.
78. Etheridge E. W. *The Butterfly Caste: a Social History of Pellegra in the South.* Greenwood, Westport, CT, 1972.
79. Terris M. (Ed.) *Goldberger on Pellagra.* Louisiana State University Press, Baton Rouge, LA, 1964.
80. Sydenstricker E. *Health and Environment.* McGraw Hill, New York, 1933.
81. Kuller L. H. Relationship between acute and chronic disease epidemiology. *Yale J. Biol. Med.* **60,** 363–376, 1987.
82. Cassell J. Social science theory as a source of hypotheses in epidemiologic research. *Am. J. publ. Hlth* **54,** 1482–1488, 1964.
83. Cassell J. The contribution of the social environment to host resistance. *Am. J. Epidemiol.* **104,** 107–123, 1976.
84. Syme S. L. Social determinants of disease. In *Maxcy-Rosenau-Last Public Health & Preventive Medicine.* 13th edn. (Edited by Last J. M. and Wallace R. W.), pp. 687–700. Appleton & Lange, Norwalk, CT, 1992.
85. Vandenbroucke J. P. Is 'the causes of cancer' a miasma theory for the end of the twentieth century? *Int. J. Epidemiol* **17,** 708–709, 1988.
86. Vandenbroucke J. P., Eelkman R. H. M. and Brukers H. Who made John Snow a hero? *Am. J. Epidemiol.* **133,** 967–973, 1992.
87. Hulka B. S., Wilcosky T. and Griffith J. D. (Eds) *Biological Markers in Epidemiology.* Oxford University Press, New York, 1990.
88. Loomis D. and Wing S. Is molecular epidemiology a germ theory for the end of the twentieth century? *Int. J. Epidemiol.* **19,** 1–3, 1990.
89. Yack D. Biological markers: broadening or narrowing the scope of epidemiology? *J. clin. Epidemiol.* **43,** 309–310, 1990.
90. Hubbard R. and Wald E. *Exploding the Gene Myth.* Beacon Press, Boston, 1993.
91. Lippman A. Led (astray) by genetic maps: the cartography of the Human Genome and health care. *Soc. Sci. Med.* **35,** 1469–1476, 1992.
92. McKeown T. *The Origins of Human Disease.* Blackwell, Oxford, 1988.
93. Boyden S. F. (Ed.) *The Impact of Civilization on the Biology of Man.* Toronto University Press, Toronto, 1970.
94. Fiennes R. N. *The Environment of Man.* St Martin's Press, New York, 1978.
95. Cohen M. N. *Health and the Rise of Civilization.* Yale University Press, New Haven, CT, 1989.
96. Szreter S. The importance of social intervention in Britain's mortality decline *ca* 1850–1914: a reinterpretation of the role of public health. *Soc. Hist. Med.* **1,** 1–38, 1988.
97. Black D., Morris J. N., Smith C. and Townsend P. *Inequalities in Health: the Black Report.* Penguin Books, Harmondsworth, 1985.
98. Krieger N., Rowley D., Hermann A. A., Avery B. and Phillips M. T. Racism, sexism, and social class: implications for studies of health, disease, and well-being. *Am. J. Prev. Med.* **9,** suppl 6, 82–122, 1993.
99. Krieger N. The making of public health data: paradigms, politics, and policy. *J. publ. Hlth Policy* **13,** 412–427, 1992.
100. Haan M., Kaplan G. A. and Camacho T. Poverty and health: prospective evidence from the Alameda county study. *Am. J. Epidemiol.* **125,** 989–998, 1987.
101. Marmot M. G., Smith G. D., Stansfeld S. *et al.* Health inequalities among British civil servants: the Whitehall II study. *Lancet* **337,** 1387–1393, 1991.
102. Whitehead M. *The Health Divide.* Penguin Books, Harmondsworth, 1987.
103. Svensson P. G. Health inequities in Europe—introduction. *Soc. Sci. Med.* **31,** 225–226, 1990.
104. Sanders D. *The Struggle for Health: Medicine and the Politics of Underdevelopment.* MacMillan, London, 1985.

105. Laurell A. C. Social analysis of collective health in Latin America. *Soc. Sci. Med.* **28,** 1183–1191, 1989.
106. Franco S., Nunes E., Duarte E., Breilh J., Granda E., Yepez J., Costales P. and Laurell C. *Debates en Medicina Social.* Organizacion Panamericana de la Salud-ALAMES, Quito, Ecuador, 1991.
107. Johnson J. V. Collective control: strategies for survival in the workplace. *Int. J. Hlth Services* **3,** 469–480, 1989.
108. Karasek R. The political implication of psychosocial work redesign: a model of the psychosocial class structure. *Int. J. Hlth Services* **3,** 481–508, 1989.
109. Krieger N. Racial and gender discrimination: risk factors for high blood pressure? *Soc. Sci. Med.* **30,** 1273–1281, 1990.
110. Armstead C. A., Lawler K. A., Gordon G., Cross J. and Gibbons J. Relationship of racial stressors to blood pressure and anger expression in black college students. *Hlth Psychol.* **8,** 541–556, 1989.
111. James S. A. LaCroix A. Z., Kleinbaum D. G. and Strogatz D. S. John Henryism and blood pressure differences among black men. II. The role of occupational stressors. *J. Behav. Med.* **7,** 259–275, 1983.
112. Geronimus A. T. The weathering hypothesis and the health of African-American women and infants: evidence and speculation. *Ethnicity Dis.* **2,** 207–221, 1992.
113. Dressler W. W. *Stress and Adaptation in the Context of Culture: Depression in a Southern Black Community.* State University of New York Press, Albany, NY, 1991.
114. David R. J. and Collins J. W. Jr Bad outcomes in black babies: race or racism? *Ethnicity Dis.* **1,** 236–244, 1991.
115. Cooper R. and David R. The biological concept of race and its application to epidemiology. *J. Hlth Politics Policy Law* **11,** 87–116, 1986.
116. Navarro V. Race or class versus race and class: mortality differentials in the United States. *Lancet* **ii,** 1238–1240, 1990.
117. Roberts H. (Ed.) *Women's Health Counts.* Routledge, London, 1990.
118. Hall E. M. Double exposure: the combined impact of the home and work environments on psychosomatic strain in Swedish women and men. *Int. J. Hlth Services* **22,** 239–260, 1992.
119. Bassett M. T. and Mhloyi M. Women and AIDS in Zimbabwe: the making of an epidemic. *Int. J. Hlth Services* **21,** 143–156, 1991.
120. Krieger N. Women and social class: a methodologic study comparing individual, household, and census measures as predictors of black/white differences in reproductive history. *J. Epidemiol. Community Hlth* **45,** 35–42, 1991.
121. Krieger N. Influence of social class, race, and gender on the etiology of hypertension among women in the United States. Conference proceedings, "Women, behavior, and cardiovascular disease," National Heart, Lung, and Blood Institute, Washington, DC. In press.
122. Krieger N. Exposure, susceptibility, and breast cancer risk: a hypothesis regarding exogenous carcinogens, breast tissue development, and social gradients, including black/white differences, in breast cancer incidence. *Breast Cancer Res. Treat.* **13,** 205–223, 1989.
123. Arber S. and Ginn J. Gender and inequalities in health in later life. *Soc. Sci. Med.* **36,** 33–46, 1993.
124. PAHO. On the concept of health and disease: description and explanation of the health situation. *Epidemiol. Bull.* **10,** 1–8, 1990.
125. Organizacion PanAmericana de la Salud. La formacion en epidemiologia para el desarrollo de los servicios de salud. XIV conferencia de la Asociacion LatinoAmerican y del Caribe de educacion en salud publica (ALASEP). Taxco, Mexico, 15 al 19 Noviembre 1987, PAHO/WHO, Washington, DC, 1988.
126. Brenner M. H. *Estimating the Effects of Economic Change on National Health and Social Well-being. Joint Economic Committee of the U.S. Congress.* U.S. Government Printing Office, Washington, DC, 1984.
127. Eyer J. and Sterling P. Stress related mortality and social organization. *Rev. Radical Polit. Econ.* **9,** 1–44, 1977.
128. Stark E. The epidemic as a social event. *Int. J. Hlth Services* **7,** 681–705, 1977.
129. Turshen M. *The Politics of Public Health.* Rutgers University Press, New Brunswick, NJ, 1989.
130. Packard R. M. *White Plague, Black Labor: Tuberculosis and the Political Economy of Health and Disease in South Africa.* University of California Press, Berkeley, CA, 1989.
131. Young R. *Darwin's Metaphor.* Cambridge University Press, Cambridge, 1985.
132. Martin J. and Harre R. Metaphor in science. In *Metaphor: Problems and Perspectives* (Edited by Miall D.S.), pp. 89–105. The Harvester Press, Sussex, NJ, 1982.
133. Gould S. J. Glow, big glowworm. In *Bully for Brontosaurus* (Edited by Gould S. J.), pp. 255–268. W. W. Norton & Co., New York, 1991.
134. MacCormac E. R. *A Cognitive Theory of Metaphor.* MIT Press, Cambridge, MA, 1985.
135. Osherson S. and Amarasingham L. The machine metaphor in medicine. In *Social Contexts of Health, Illness, and Patient Care* (Edited by Mishler E. G., Amarasingham L., Hauser S. T. *et al.*), pp. 218–249. Cambridge University Press, Cambridge, 1981.
136. Lakoff G. and Johnson M. *Metaphors we Live By.* Chicago University Press, Chicago, 1980.
137. *The Compact Edition of the Oxford English Dictionary.* Oxford University Press, Glasgow, 1971.
138. Gould S. J. *Wonderful Life: the Burgess Shales and the Nature of History.* W. W. Norton & Co., New York, 1989.
139. Gleik J. *Chaos: Making A New Science.* Viking Penguin, New York, 1987.
140. Glass L. and Mackey M. C. *From Clocks to Chaos: the Rhythms of Life.* Princeton University Press, Princeton, NJ, 1988.
141. Lipsitz L. A. and Goldberger A. L. Loss of 'complexity' and aging: potential applications of fractals and chaos theory to senescence. *J.A.M.A.* **267,** 1806–1809, 1992.
142. Schaffer W. M. Chaos in ecology and epidemiology. In *Chaos in Biological Systems* (Edited by Degn H., Holden A. V. and Olsen L. F.), pp. 233–248. Plenum Press, New York, 1987.
143. Rose H. and Rose S. (Eds) *Ideology of/in the Natural Sciences.* Schenkman, Cambridge, MA, 1979.
144. Haraway D. *Primate Visions: Gender, Race, and Nature in the World of Modern Science.* Routledge, New York, 1989.
145. Hubbard R. *The Politics of Women's Biology.* Rutgers University Press, New Brunswick, NJ, 1990.
146. U.S. Department of Health Services, Public Health Service. *Healthy People 2000: National Health Promotion and Disease Prevention Objectives.* DHHS Pub. No. (PHS) 91-50123, U.S. Government Printing Office, Washington, DC, 1991.
147. NIH/ADAMHA policy concerning inclusion of women in study populations. *NIH Guide for Grants and Contracts,* October 24, 1986.
148. NIH/ADAMHA policy concerning inclusion of minorities in study populations. *NIH Guide for Grants and Contracts,* September 25, 1987.
149. Centers for Disease Control and Prevention. Use of race and ethnicity in public health surveillance. *M.M.W.R.* **42,** (No. RR-10), 1–17, 1993.

150. Coreil J., Levins J. S. and Jaco E. G. Life style: an emergent concept in the sociomedical sciences. *Culture, med. psychiat.* **9,** 423–437, 1985.
151. Last J. M. (Ed.) *A Dictionary of Epidemiology—A Handbook Sponsored by the International Epidemiological Association*, p. 32. Oxford University Press, New York, 1983.
152. Krieger N. Overcoming the absence of socioeconomic data in medical records: validation and application of a census-based methodology. *Am. J. Publ. Hlth* **82,** 703–710, 1992.
153. Von Korff M., Koepsell T., Curry S. and Diehr P. Multi-level analysis in epidemiologic research on health behaviors and outcomes. *Am. J. Epidemiol.* **135,** 1077–1082, 1992.
154. Alker H. R. Jr. A typology of ecological fallacies. In *Social Ecology* (Edited by Doggan M. and Rokkan S.), pp. 69–81. MIT Press, Cambridge, MA, 1969.
155. Robinson W. S. Ecological correlations and the behavior of individuals. *Am. Sociol. Rev.* **15,** 351–357, 1950.
156. Langbein L. I. and Lichtman A. S. *Ecological Inference.* Sage, Beverly Hills, CA, 1978.
157. Iversen G. R. *Contextual Analysis. Sage University Paper Series on Quantitative Applications in the Social Sciences. Series No. 07-081.* Sage, Newbury Park, CA, 1991.
158. Van den Eeden P. and Huttner H. J. M. Multi-level research. *Current Sociol.* **30,** 1–181, 1982.