Epidemiologic Reviews

Vol. 26, 2004
Printed in U.S.A.
DOI: 10.1093/epirev/mxh010

What's Wrong with Social Epidemiology, and How Can We Make It Better?

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Received for publication December 11, 2003; accepted for publication February 20, 2004.

INTRODUCTION

It is perhaps ironic that an epidemiologist who has been working in the field of social epidemiology for over a quarter of a century, and who directs a center focused on social epidemiology, should coin a title suggesting that there is something "wrong" with social epidemiology. Perhaps it is even inopportune, as it could provide ammunition to those who believe that the practice of social epidemiology is misguided, unscientific, ideological, or too overreaching (1–3). However, this title was chosen purposely with the hope that identifying some of the critical intellectual, methodological, and empirical lacunae and challenges in social epidemiology might promote continuing development of a social epidemiology that is both scientifically enlightening and useful, productive, and contributory to the public's health.

Indeed, the hope is that the "social" in epidemiology will become so integral a part of epidemiology that the term can be dropped altogether. To assert that all epidemiology is social is not an attempt at intellectual hegemony—that the problems of disease and the distribution of disease in populations over time and space can be understood from a social perspective *only* or that such information is in some sense more fundamental that other types of information about disease determinants. In the same way that our understanding of the etiology of chronic and infectious diseases benefits from knowledge of the pathobiologic processes involved in such diseases, increased understanding of social factors, broadly considered, may shed light on processes every bit as integral to our understanding of the etiology of those diseases.

There is no question that social epidemiology has come of age and that the term "social epidemiology" is being increasingly used to describe examination of the role of a broad array of social factors in the development and progression of many important health problems, and in the natural history of the risk factors for those diseases and conditions. While not all may agree with Geoffrey Rose's assertion that "the primary determinants of disease are mainly economic and social, and therefore its remedies must also be economic and social" (4, p. 129), there is no question that there has been enormous growth in the study of these economic and social forces on health and disease. Figure 1, which plots growth since 1966 in use of the term "social epidemiology" in article titles, abstracts, and keywords, graphically illustrates this increased interest. Beginning in the early 1980s, growth of such publications increased rapidly, well fit by an exponential curve. In fact, this figure is likely to dramatically underrepresent the growth of social epidemiologists' interest in the matter; a similar exponential growth pattern has been seen when studies examining socioeconomic position and health (5) and social capital and social relationships (6) were similarly totaled.

Other contributions in this volume of *Epidemiologic Reviews* take up the wide variety of topics that social epidemiologists study. In what follows, I instead touch on a series of issues that highlight some of the critical problems with which social epidemiology must grapple. Many of the issues discussed are not restricted to social epidemiology and have their analogs in other areas of epidemiology. Thus, while the focus of this review is on social epidemiology, it would be misleading to suppose that similar criticisms do not apply to other lines of research. In addition, I want to make it clear that many of the criticisms raised apply to my own work as well as to others'. Because this is an attempt at a form of self-criticism of the field, I do not focus on as extensive citation as other reviews do. Where I do cite the work of others, it should be considered illustrative, not as singling out a particular piece of work. The lessons to be learned hopefully apply to the many.

METAPHORS AND MODELS

Figures 2, 3, 4, and 5 present a few of the many diagrammatic models that have been used to illustrate recent social epidemiologic approaches to understanding the social determinants of health and health disparities (e.g., Kaplan et al. (7), Marmot (8), House (9), Lynch (10)). These models have

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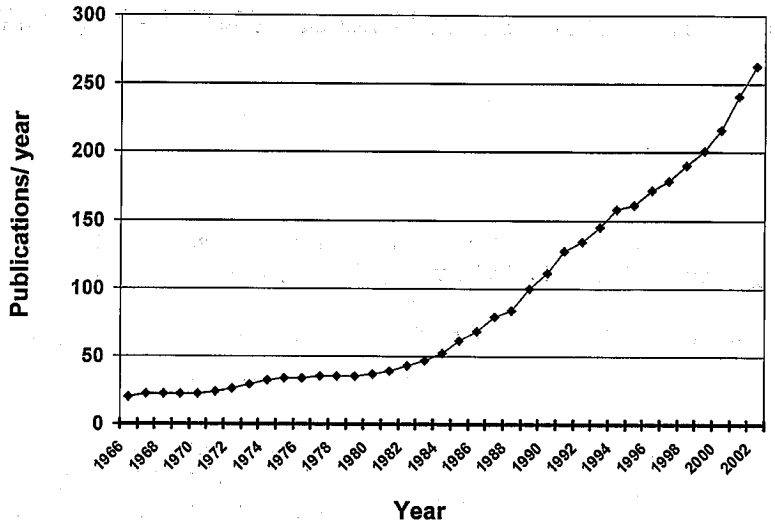

FIGURE 1. Numbers of publications included in MEDLINE, Current Contents, and PsychINFO that contain "social epidemiology" in the title, abstract, or keywords, 1966–2002.

many features in common, the most prominent being an emphasis on layered, multilevel understandings; a multiplicity of pathways; and possibilities for reciprocal influences. Such models serve as important metaphors, attempting to portray the component parts of complex processes, their interrelations, and the temporal relations between components. They act as an important caution against the potentially misleading oversimplification that comes from focusing on one level of influence, often the one most proximal to the outcome, and not the flow of information and influence represented by the totality of processes and relations. Similar to a good cartoon, they remove extra-

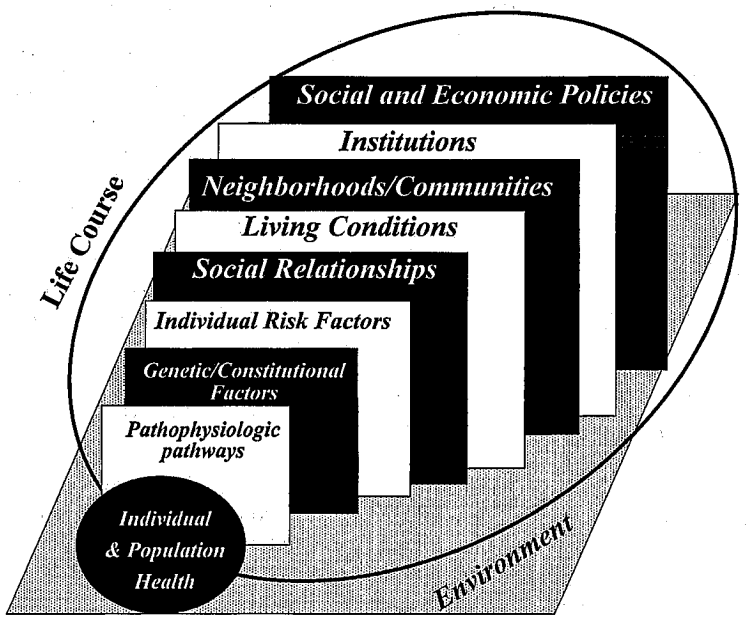

FIGURE 2. Upstream and downstream determinants of population health. Reprinted with permission from *Promoting Health: Intervention Strategies from Social and Behavioral Research*, by the National Academy of Sciences. Courtesy of the National Academies Press, Washington, DC.

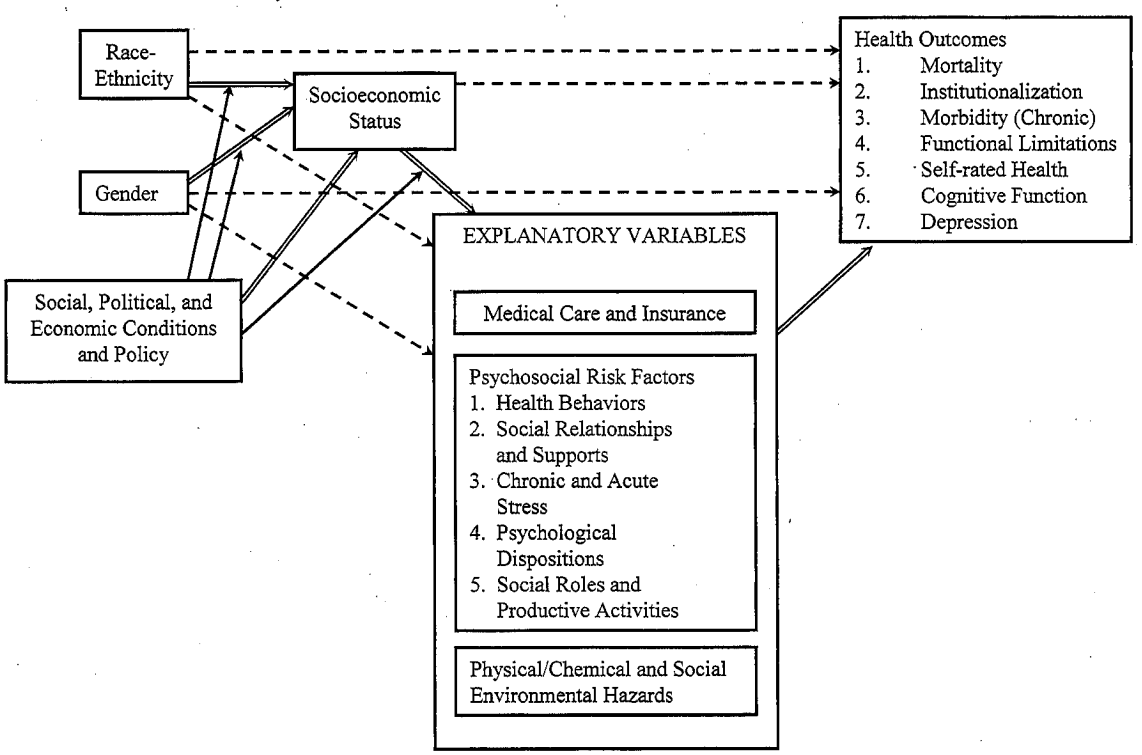

FIGURE 3. Environmental, psychosocial, and biological pathways linking socioeconomic status to diabetes mellitus (DM), coronary heart disease (CHD), and well-being. CNS, central nervous system. Reprinted with permission from the American Sociological Association. James S. House, "Understanding Social Factors and Inequalities in Health: 20th Century Progress and 21st Century Prospects," *Journal of Health and Social Behavior*, volume 43, number 2 (June 2002), pages 125–142.

neous factors and draw our attention to what is believed to be most critical.

While the heuristic utility of such models can be substantial, they do both too much and too little. Such models are common in science, but the span of factors considered in models of this type in social epidemiology—sometimes linking the most macro- and microlevel phenomena—is considerably broader than that found in many scientific pursuits (e.g., figure 6; Kaplan and Lynch (11)), creating considerable problems regarding data availability and analytical methods. Information is seldom available at the multitude of levels portrayed in such models and, where available, is often measured cross-sectionally, making the temporal influences that we consider so important in the assessment of causality opaque. Against the backdrop of these models of complex longitudinal processes, it is not unusual to attempt to use various standard multivariate statistical techniques to examine the relative contribution of one social determinant versus another to the incidence or progression of disease. Thus, the relative impact of income and race on an outcome may be compared; the independent effect of income, education, and occupation may be estimated; or the strength of the association between job control and occupational social class and some outcome may be estimated. The critical problem is that simultaneous measurement and use of these variables in multivariate models belies the historical, life-course, and temporally ordered social stratification processes that they reflect. Use of the standard toolbox of multivariate regression techniques to investigate these complex social epidemiologic models becomes even more difficult absent information related to measurement error (12); changes in exposure over time (13); the reciprocal effects of behavioral and social factors, for example, on each other over time (14); and deeper issues related to the very identifiability of certain kinds of causal effects (15).

Caught between this rock of inadequate data and the hard place of analytical limitations, are such models worthwhile? The ultimate answer will of course result from the extent to which both the heuristic use of such models and the analytical results based on them are illuminating. On the analytical side, new multilevel analytical techniques (e.g., Raudenbush and Bryk (16), Diez Roux (17)), extensions of recent advances in causal analysis and simulation (e.g., Greenland et al. (18), Wolfson (19)), and techniques borrowed from other fields (e.g., Zohoori and Savitz (20)) may prove useful.

In my opinion, the recent substantively modest, but methodologically complex contribution by Adams et al. (21) and the responses to it by economists, epidemiologists, and others in the same publication suggest a less optimistic view. While methodological rigor is always to be applauded, one is

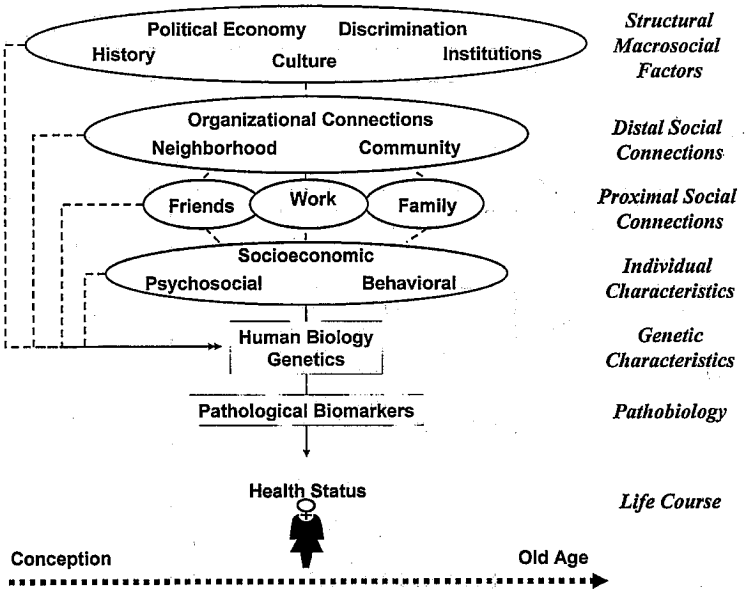

FIGURE 4. A conceptual framework for understanding social inequalities in health and aging. Solid arrows between boxes indicate presumed causal relations among variables; dotted arrows intersect solid arrows, indicating an interaction between the conditioning variables in the box at the beginning of the solid arrow in predicting variables in the box at the head of the solid arrow (10).

left with the impression that a number of the authors believe that the analysis of causal relations in observational data is so flawed as to potentially threaten even the conclusion that smoking causes lung cancer.

The fundamental need for better and more comprehensive data to "test" these models will not be solved by better statistical techniques, and there has been considerable lament regarding the ascendancy of technique over theory in epidemiology (22, 23) and its separation from basic foci of public health. Indeed, development of epidemiologic theory per se, separate from techniques for analyzing causal effects and partitioning sources of noise in data (24), may be required. However, even such developments will not substitute for better sources of data and for new methods to allow the stitching together of data from a variety of sources, levels, periods, and places. Quilts of such data stitched together by using such new techniques, and incorporating sensitivity analyses and other techniques, could considerably strengthen our ability to turn the complex models of social epidemiology into useful analytical models of disease processes in persons and populations.

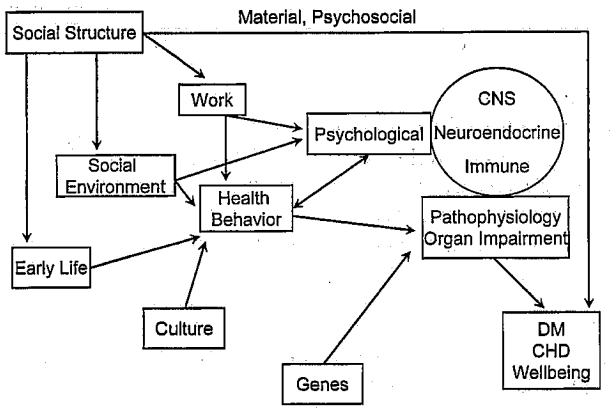

FIGURE 5. Multilevel, life-course determinants of population health. Figure 15-3 from Chapter 15: "Multilevel Approaches to Understanding Social Determinants," by Michael Marmot, from *Social Epidemiology*, edited by Lisa Berkman and Ichiro Kawachi, copyright © 1999 by Oxford University Press, Inc. Used by permission of Oxford University Press, Inc.

THE NEED FOR SOCIAL EPIDEMIOLOGIC THEORY

Perhaps nowhere is the need for social epidemiologic theory more apparent than in the study of "place" effects on health. While some have argued that it is methodologically difficult to identify the effects of context (25), and discussion of the role of context versus compositional effects continues (26), there is now an impressive array of studies from epidemiology, human development, sociology, and other disciplines suggesting an important role of place in a variety of health and developmental outcomes (27–30). For example, Haan et al. (31), using the Alameda County Study cohort, found that residence in a federally designated poverty area was associated with more than a 50 percent increased risk of death over the next 9 years. These findings were replicated at the national level by Waitzman and Smith (32), and, in the Alameda County Study, residence in the poverty area was associated with greater declines in physical activity over

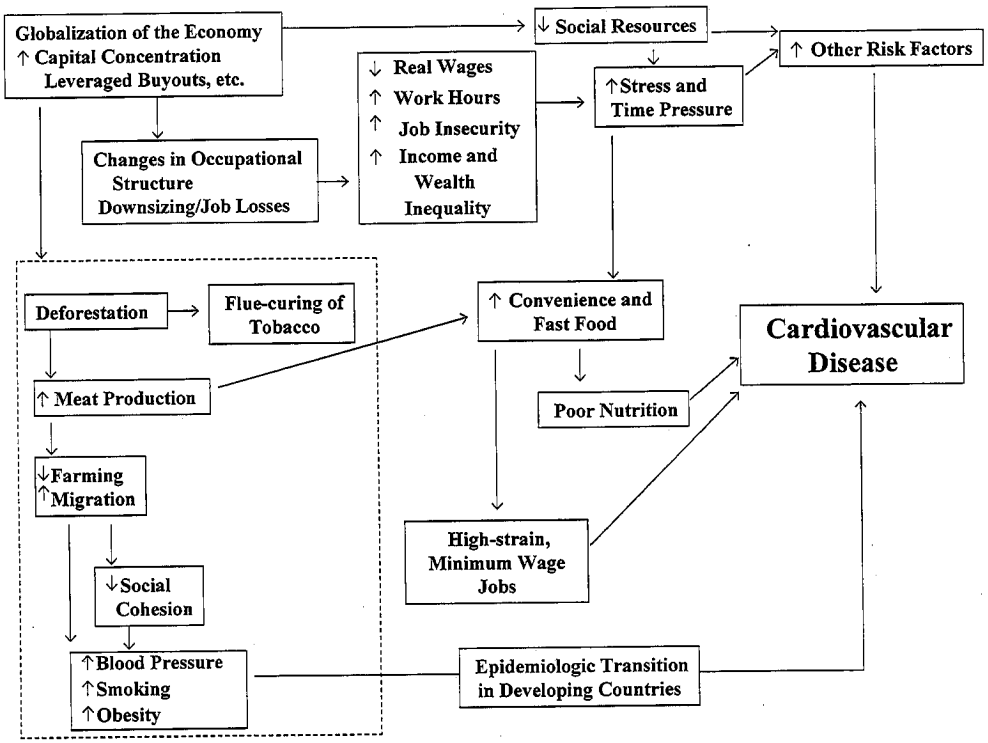

FIGURE 6. Primordial prevention of cardiovascular disease (11). Dashed lines indicate processes occurring in "developing" parts of the world. Reprinted from *Preventive Medicine*, volume 29, by G. A. Kaplan and J. W. Lynch, "Socioeconomic Considerations in the Primordial Prevention of Cardiovascular Disease," pages S30–S35, copyright 1999, with permission from Elsevier.

time and an increased incidence of depression (33, 34). Since this early study, there has been a rapid increase in studies of a variety of health and mortality effects associated with residence in areas as small as census block groups and as large as states and nations. Although use of sophisticated multilevel statistical techniques in such studies has increased tremendously, numerous conceptual and methodological questions still remain unanswered (17, 35).

Prominent among these are questions concerning what it is about residence in a particular place that conveys increased or decreased risk. In the case of more traditional environmental exposures, it is much easier to come up with an approach to exposure measurement, modeling of exposure effects, selection of possible confounders, and plausible biologic pathways. Such areas of research also point one in the direction of a particular level or scope of spatial analysis. For example, studies of children's exposure to lead-based paint would be more likely to focus on the spatial "grain" of the home and environment very proximate to the home; in contrast, studies of outdoor, airborne particulate matter would likely focus on much wider areas, perhaps incorporating knowledge of local geography and air circulation patterns. In the relative absence of such well-specified models of the level of influence of a particular social or economic factor on a particular health outcome, one is left with examples such as the literature relating income inequality to health outcomes, in which the "grain" varies from census block to country.

Similarly, the spatial scope over which determinants of specific outcomes such as all-cause mortality (36), infant mortality (37), or cardiovascular disease (38), or other outcomes, operate is less clear. Without some theory to suggest the ways in which the determinants of these outcomes are spatially embedded, one is left with a plethora of analyses of "area" effects that seem more often driven by the level of spatial data available, or other arbitrary factors, than by reasoned etiologic considerations.

This lack of clarity is not without its perils because it often leads to wide use of terms such as "neighborhood," which, while they may (or may not) invoke some shared implicit definition, are rather opaque with regard to the specific processes that are health damaging or enhancing. There are some exceptions; for example, work exists on the impact of informal social controls, operating at the small-scale neighborhood level, on the antisocial behavior of adolescents (39). Here, there are clearly stated social processes, which can be measured on a relatively small geographic scale, and a specified process that links these spatially distributed characteristics to the outcome in question.

ANALYTICAL AND METHODOLOGICAL PITFALLS IN SOCIAL EPIDEMIOLOGY

As in most areas of epidemiologic study, social epidemiology makes predominant use of observational data, with all of the attendant methodological and analytical challenges. For the most part, advances in measurement theory and causal analysis have not penetrated social epidemiologic analyses very deeply. For example, the search for social factors "independently" associated with a particular health outcome often involves use of more traditional risk factors measured rather crudely, presumably leading to considerable residual confounding. Along with the crudity of measurement, most often dictated by the data available in secondary analyses, these potential confounders are often subject to considerable change over time, which may or may not be related to the social factor under examination; therefore, baseline adjustment for them results in considerable misclassification of exposure over time.

The search for "independent" risk factors has been a dominant force in social epidemiology, and certainly risk factor epidemiology has come under fire from a number of quarters (40). As is true for much of epidemiology, claims to independent effects often conflate issues of statistical independence that are at least dependent on measurement and modeling approach with causal independence, a process not without its perils. In social epidemiology, this search for independent effects may represent more of an attempt at legitimization of a newly evolving field—identifying "new" risk factors—than something more informative. Many analyses stop with demonstrating a statistically significant association between the risk factors and outcomes in question after adjustment for known risk factors. While it could be argued that identifying new risk factors can catalyze the search for new disease mechanisms, it can also result in a plethora of new "social" risk factors that sometimes exist in an almost miasma-like fog; we observe them to be importantly related to some outcome but cannot really identify the mechanisms that explain this association.

Leaving aside the very real possibility that the measurement and analysis issues discussed earlier lead to misleading observations of independence, several examples of this problem come to mind. For example, much of the early work showing an association between socioeconomic position and mortality from cardiovascular disease or all causes statistically adjusted for potential behavioral and pathophysiologic pathways and demonstrated that adjustment for these factors did not eliminate the increased risk associated with lower socioeconomic position (41). This work has led to broad generalizations about these associations not being explained by such pathways, although some analyses do not support such a conclusion (42). Although eliminating candidate pathways can lead to identification of potentially new pathways mediating the relation, it can also represent an approach that focuses on eliminating all potential explanatory pathways. Such an approach seems less useful than one carefully demonstrating the contribution of intermediate pathways to the observed association. As a counterexample, the maturing approach to the relation between cynical hostility and cardiovascular outcomes illustrates the value (43, 44) of identifying the role of both behavioral and psychophysiologic pathways in explaining this association.

To some extent, social epidemiology, similar to much of epidemiology, would be well served by going back to careful analysis of antecedent, mediating, and confounding variables in assessing causal relations, as portrayed in Susser's book, *Causal Thinking in the Health Sciences*, published over a quarter of a century ago (45). The book carefully lays out a logic of analysis based on a careful and informed biologic and social understanding of the outcomes being studied sometimes not present in more quantitatively sophisticated approaches. While promising, it remains to be seen whether recent work on counterfactual contrasts and causal analysis (46, 47), for example, will take us substantially further than this earlier contribution.

Notwithstanding the arguments about the logic of causal analysis, the focus on multilevel determinants of disease, as illustrated in figure 2, presents new methodological and data challenges to social epidemiologists. Few data sets contain the levels of information necessary to populate empirical tests of such models, and the models are usually not specified well enough to parameterize tests of them. In analyses focused on such models, it is useful to contrast explanations concentrated on "how" and "why" (42). For example, an understanding of the association between socioeconomic position and a particular outcome clearly benefits from an understanding of the specific social, behavioral, and physiological pathways that link socioeconomic position and the outcome—the "how." However, another question remains: "why" is it that these pathways are differently distributed by socioeconomic position? Answering such a question, which, paraphrasing Geoffrey Rose, might be called the "causes of the causes" (48), requires casting a net across a broad array of determinants ranging from historical influences, to public policy, and perhaps even to genes. Although difficult to address "why," it seems clear that an explanation that focuses on only the "how" is a limited one.

THE PURSUIT AND PERILS OF INTERDISCIPLINARITY

A recent Institute of Medicine report concluded that "basic and applied research from a range of disciplines has demonstrated the importance of reciprocal interactions over time among health and biological, psychological, and social factors" (49, p. 332). In the category of biological factors, the report included "genes, neurochemical, and hormonal processes and function of the physiological systems"; in the psychological category, it included "behavioral, personality, temperamental, cognitive, and emotional" factors; and, in the social category, it included "socioeconomic status, social inequalities, social networks and support, and work conditions" (48, p. 332). With similar lists, other recent reports and volumes have come to similar conclusions, all consistent with the approaches illustrated in figures 2, 3, 4, and 5 (49–52). All reflect the widely appreciated fact that virtually all scientific endeavors are increasingly inter-, multi-, and transdisciplinary. Although such approaches fit well within the developing field of social epidemiology, they are associated with certain problems.

Because many of the topics and constructs in social epidemiology come from the social sciences, they carry with them an immediacy and familiarity not found in the world of genetics, for example. Although we may not all know what a "haplotype" is, we all think we know what "neighborhood," "poverty," "community," or "depression" means. This use of familiar terms has led to a certain conceptual looseness that may not be found in other scientific areas, reminiscent of Humpty Dumpty's statement, "When I use a word ... it means just what I choose it to mean" (53, p. 113). Thus, investigators might use the construct of "neighborhood" in describing analyses of census tracts or zip codes. For some, the term might mean an area characterized by dense, interconnected networks of people who care about each other; for others, it might mean a historically defined spatial area. In reality, what is meant is actually a spatial area constructed decades ago to represent some degree of demographic homogeneity (census tract) or an area defined by the location of postal facilities (zip codes). Similarly, the topic of "community" is often raised without any clarity as to how that community is defined or whether it has any phenomenological identity—whether the people whom the investigator locates as being part of the community actually believe they are part of it, or whether that even matters.

As in any other interdisciplinary area, studies involving many disciplines present considerable challenges. Social epidemiologists who would like to produce work that spans the biologic and social face the challenges of integrating findings from different fields, using disparate terminology and analytic methods, and understanding the typically voluminous literature in any field. To some extent, closely connected interdisciplinary groups can help to augment each other's gaps with respect to particular areas of expertise and knowledge. However, there is a considerable danger of thin glosses on areas of expertise not one's own. For example, one social epidemiologic explanation of socioeconomic inequalities in health places great emphasis on comparisons that persons make between themselves and others, focusing on the links between judgments of relative deprivation that they make in comparing themselves with others, and the psychosocial and neuroendocrine effects of such comparison. Thus, a critical component is this process of social comparisons. As it turns out, social comparison processes have been studied extensively for 50 years by social psychologists and sociologists, and a number of determinants and consequences of social comparisons have been studied (54–56). However, it is rare to see any of this literature in the social epidemiologic literature discussing relative deprivation and health.

As another example, consider the evidence linking dominance and social organization in nonhuman primates (mainly macaques) with coronary heart disease, or that linking dominance and rank with neuroendocrine function, often reflected by variations in cortisol levels. A recent systematic review by Petticrew and Davey Smith (57) suggests that many of the broad generalizations about the former work do not reflect the considerable heterogeneity in findings found in the studies; overall, the studies showed no systematic association between rank and disease endpoints. These authors suggest that considerably more reference is made to these studies in the literature on social determinants of health than is borne out by the inconsistency of the findings. Similarly, while there is also considerable citation of results indicating a consistent relation between rank and cortisol levels in nonhuman primates, a recent review of studies across a number of nonhuman primates indicates little consistency of findings (58). In fact, Sapolsky, a leading researcher in the area, commented that "a prime revisionist emphasis of this chapter has been how *little*, in fact, rank *per se* predicts any of those endpoints. Instead, it seems virtually meaningless to think about the physiological correlates of rank outside the context of a number of other modifiers" (59, p. 39; emphasis in original). The purpose of this discussion is not to single out particular investigators—indeed, there may or may not end up being consistent findings in these areas—but rather to highlight some of the potential perils of interdisciplinary work, particularly in areas of research that are fast moving. The lesson is not to shun integration across multiple disciplines, because it is critical, but to suggest that it be done in a way that maximizes the ability of each discipline to contribute information and nuance to the effort.

Personalizing populations

Although many of the discussions of the need for a social epidemiologic approach cite the work of Geoffrey Rose (60) as the rationale for a *population*-focused strategy, much of the work in social epidemiology focuses on individuals. Surprisingly, this focus on individuals is found even when studying factors such as social networks and social support, where the concepts refer to exchanges between individual persons and participation in groups, but the data used in most studies are collected from individuals (61). The literature demonstrating an important association between aspects of social relationships and health is now substantial (62), but, for the most part, there is relatively little study in social epidemiology of the population distribution of these factors or the determinants of their distribution. In some cases, such as studies linking health disadvantage to low levels of social capital, the lack of a population-based focus is particularly noticeable when social capital—conceived of by most as an attribute of a social group—is measured by individual reports (63). Indeed, some investigators (64) see the same questions about distrust and cynical hostility as reflecting individual attributes; others (63) consider them indicators of societal attributes.

While Margaret Thatcher may have said that "as you know, there is no such thing as society: there are individual men and women, and there are families" (65, p. 9), social epidemiologists who discuss the importance of population-level phenomena and determinants of health have an obligation to be articulate about the difference between a population and a Thatcherian aggregate of individual persons. To some extent, this tack has been taken in studies of income inequality and health (66) and of societal influences on human development (67). In many respects, John Cassel's (68) seminal paper, "The Contribution of the Social Environment to Host Resistance," was a step in that direction. Much more work (perhaps integrating the insights of demography, political science, sociology, and economics) needs to be

done by social epidemiologists before Rose's (69) observation—that the causes of cases within populations may be very different from the causes of differences in incidence between populations—can be fully appreciated. Social epidemiology has much to contribute to this search, but, to do so, it will need to move to a more population-based approach with respect to putative causal factors (70), notwithstanding the perils of ecologic studies (71).

Global social epidemiology

With social epidemiology's emphasis on a broad array of health determinants, it is surprising that so little work takes place in the context of global or international health. By this pursuit I mean not simply conducting studies of disease *X* or risk factor *Y* in various countries but instead using a truly comparative approach. To some degree, this approach has been attempted in research considering the association between income inequality and life expectancy or mortality (66) and in that examining differences in inequalities in health across countries (72, 73). In both cases, the work has met with some obstacles—in the first case because of the sensitivity of the results to time, period, and outcomes (74) and in the second case because the results depended on the measures of association used in the comparisons (75–77). However, the large variation between countries, and varying trends within countries, in many of the core determinants of health studied by social epidemiologists—socioeconomic position, race and ethnicity, gender, poverty and welfare policy, education, immigration, and so forth—suggests a rich field for examining both the impact of social determinants on health and the extent to which they depend on context. In carrying out such analyses, it is critical to have specific hypotheses that inform the enterprise. Rather than simply examining the health effects of a particular social factor in different countries, it is far more instructive to examine variations in the levels or distribution of the factor across countries by using between-country variations as a test of the relation. At the same time, such studies also enable examination of the ways in which context affects the relation. Thus, in countries with strong economic and social supports for the unemployed, we might expect unemployment to have less of an effect on health than in countries where such policies are weak. The growing number of systematic surveys and cohort studies across the globe mean that, in carrying out such activities, one is no longer limited to assessment of only ecologic associations.

Health versus health care

In many areas of social epidemiology, the dominant approach to health care has been to ignore or dismiss it. Thus, numerous articles argue that the contributions of medical care to the health of the population are, at best, modest (78–80). Reflecting the historical influences of McKeown (81) and others, these attempts might be seen as staking out an area of research that is independent of medical care and as legitimizing the enterprise. For example, the argument goes, one needs to look beyond the contributions of medical care to explain gradients of health by socioeconomic position (82). Although that statement is undoubtedly true, it ignores the fact that medical care is provided by institutions (figure 2) and that decisions as to who receives medical care, and the quality of that care, are shaped by social processes. A recent Institute of Medicine report (83) on racial and ethnic inequalities in health care and the consequences of lack of insurance makes this strikingly clear. Thus, rather than health care being excluded from social epidemiologic approaches, it needs to be reintroduced as one of the potential social determinants of health, with the importance of its contribution varying depending on the specific health issue being considered.

Moderating essentialism

Although Humpty Dumpty chooses to make a word mean just what he wants it to mean (52), use of certain constructs in social epidemiology carries with it an almost essentialism, an observation that has even entered the discussion of the counterfactual approach to understanding adjustment for confounding in social epidemiology (84). Consider, for example, socioeconomic position. Despite observations that the magnitude, and in some cases the direction, of the relation between socioeconomic position and health outcomes is not fixed over time or across places, the graded relation between the two has become almost reified. In fact, nothing intrinsic to income, education, or occupation, or other measures of socioeconomic position, specifies a particular association with any health outcomes. It all depends on the relation between these markers of socioeconomic position and the more proximal risk factors for the particular outcome being considered (85). Conceivably, in some circumstances (rare, to be sure), there could be no relation at all, or the relation could be in the opposite directions, as for education and breast cancer incidence. Alternatively, the nature of the association could change over time, as with the reversal of the socioeconomic gradient in coronary heart disease as the association of socioeconomic position with smoking and diet changed (86).

Furthermore, focusing on the "gradient" per se can lead to a search for a unitary factor that explains the entire range of variation across the range of socioeconomic position. In fact, there may be a variety of factors, each contributing to a greater or lesser extent to health at various socioeconomic levels (87). Discussion of "the gradient" in a reified way has tended to imply that there is a very linear relation between socioeconomic position and a health outcome when, in fact, the relation between household income and risk of death, for instance, is decidedly nonlinear (88, 89). This striking nonlinearity has important implications for both our understanding of the causal pathways involved and the public health significance of the gradient. For example, the very small increased risk associated with increases in household income above the median level makes the observation of differences in disease risk between the highest levels much less compelling and suggests that more attention should be focused on those persons at the lower ranges of socioeconomic position.

This essentialist approach also applies, strangely enough, to many social epidemiologic uses of race and ethnicity in

analyses. While most social epidemiologists would acknowledge that race and ethnicity are mainly social constructions, many studies discuss "race" or "ethnicity" effects when what they are really observing are the social, environmental, economic, and historical facts that lie underneath those terms. Indeed, notwithstanding the possible psychosocial impact of discrimination on health, socioeconomic position appears to "explain" many disparities between minority and majority group health outcomes, and there are undoubtedly other critical factors as well (90, 91). For the most part, it may not be possible to separate the multifaceted patterns of historical and contemporary exposures that have led to and underlie the social construction of race and ethnicity from the terms "race" and "ethnicity." Thus, when race effects are examined in epidemiologic studies, we risk viewing race as an essentialist category and ignoring what it stands for. A good example is the controversy surrounding the finding that college-educated African-American and White women have different birth outcomes (92). This finding has often been interpreted by some, although not by the authors, as implying that biologic differences must be responsible for the difference. This conclusion ignores the substantial differences in quality of education, differing contexts for both education and pregnancy, and the wide disparities in economic and social returns for a college education between African-American and White women. Thus, such an interpretation privileges race rather than the conditions for which it is a proxy indicator.

LOST OPPORTUNITIES

With social epidemiology's emphasis on a broad array of determinants of health, many of which are sensitive to economic and social context, it would seem that social epidemiologists would be deeply involved in examining the impact of social and economic policy on health (93). For the most part, however, such pursuits are not well represented in social epidemiology, possibly because of the difficulties involved in considering all of the hidden sources of variation that could result in misleading conclusions. However, one wonders whether this task is really any more complicated than understanding the causes of the recent decline in cardiovascular mortality (94–96) or interpreting the results of community intervention on cardiovascular disease (97). While those studying the relation between income inequality and health have been motivated by the large changes in economic inequality introduced by relatively recent economic and social changes, evaluation of the effects of social and economic policy on health has mostly fallen to other disciplines. For example, epidemiologists have not had a major role in assessing major trials focused on housing relocation (98) or poverty reduction (99), and little social epidemiologic work has examined the health effects of welfare reform (100). Some of this work can be quite compelling, such as a recent study by economists suggesting that welfare reform has led to a decrease in breastfeeding 6 months after birth (101). The lack of social epidemiologic involvement in many of these studies, particularly in their design and evaluation, has more often than not resulted in little health data being collected, even in the case of social and economic changes likely to have considerable effects on health.

WHITHER SOCIAL EPIDEMIOLOGY?

It would be foolish to suggest that social epidemiology should adopt a particular set of tools or perspectives; as in any scientific discipline, invention and discovery are best served by using a diversity of approaches. However, the areas highlighted in this review are those that present considerable opportunities for clarification and progress. The explosion of interest in social determinants of health and multilevel approaches to health determinants has opened up a broad arena for research. A few years ago, a report from the Hastings Center called for an expansion of research on social determinants of health equal to that devoted to the human genome effort (102). In all likelihood that will not happen; however, this is still an era of great opportunity for social epidemiology. Before its promise can be fulfilled, it will be necessary to wrestle with difficult conceptual, methodological, and analytical problems, some of which have been discussed in this review. Social epidemiology is poised to contribute to a new paradigm that bridges the social and the biological, the ultimate mark of success being incorporation of this expanded view of social determinants of health into a science of epidemiology that sees all that it does as embedded in the social world.

ACKNOWLEDGMENTS

Partial support for preparing this article was provided by awards from the National Institute on Aging (R37 AG11375), the National Institute of Child Health and Human Development (P50 HD38986-05), and the Robert Wood Johnson Foundation Health and Society Scholars Program.

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