COMMENTARY

On Genes, Individuals, Society, and Epidemiology

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Over their long history, and depending on the theory of disease causation prevalent at the time, the fields of epidemiology and public health have focused on different factors as potential causes of disease (1–3). In the late 19th century, miasmatic theories of disease causation, in which ill health resulted from foul emanations of the soil, air, and water, gave way to the germ theory and the doctrine of specific etiology. Subsequently, with the growing importance of chronic diseases, emphasis shifted to the characteristics of individuals, behaviors, and lifestyles.

Today, epidemiology may be at the brink of a new paradigm, the genetic paradigm. The advent of new technologies, and the accompanying interest in identifying genes (and creating genetic screening tests) for particular diseases, has led to explosive growth in research on the genetics of disease and its molecular mechanisms (4–8). This trend has been accompanied by the publication of several textbooks on genetic epidemiology (9–11), and genes have recently been put forward as important factors in the genesis not only of several common diseases but also of behaviors such as physical activity (12) or psychosocial characteristics such as social support (13). Simultaneously with the interest in genes as causes of disease, there appears to have been a resurgence of research and interest in the social origins of disease—as evidenced, for example, by the increase in publications examining social class differences in health (14), and by several recent commentaries on the role and future of epidemiology which have emphasized the importance of social factors in studying and understanding the distribution of diseases (3, 15–17).

The coexistence of alternate explanations on the origins of disease is, of course, not new. Throughout the history of epidemiology and public health, there has been a steady current of social medicine and social epidemiology intent on investigating and emphasizing the links between how societies are organized and the patterns of death and disease in their populations (18–27). These “social” explanations have often competed with alternate explanations emphasizing biologic and behavioral factors; but perhaps today, the contrast between levels of explanation as diverse as genetic and social has become more evident than ever. The fragmentation of the field is further evidenced by the emergence of different “types” of epidemiology: There is “social” epidemiology, “risk factor” epidemiology, and “genetic” epidemiology, each with its own literature.

As in other scientific fields, research in public health and epidemiology has been strongly influenced by the notion that the whole can be understood by breaking it down and understanding its component parts. This strategy, known as “reductionism,” has been defined as the attempt to “explain the properties of complex wholes—molecules, say, or societies—in terms of the units of which those molecules or societies are composed” (28, p. 5). The idea that understanding the whole allows us to understand the whole has indeed led to many discoveries in a number of scientific fields (29).

In public health and epidemiology, we often apply this type of strategy. For example, in attempting...
to understand disease in populations, we break populations down into "independent" individuals. In attempting to understand disease in individuals, we tease apart the "independent" contributions of different factors. This approach has yielded much useful epidemiologic information. To cite just two examples, it has contributed to the identification of aspects of lifestyle and biologic factors associated with cardiovascular disease, the leading cause of death in many of today's societies, and has shown modest to the extent (31) that permeates science generally, as evidenced, for example, by the ongoing and lively debate on this topic in the field of biology (28, 33, 34). In its efforts to integrate social and biologic factors and individual- and group-level factors in the study of health, epidemiology faces many challenges, some of which will be briefly addressed below.

THEORIES AND MODELS OF DISEASE CAUSATION

A key challenge to today's epidemiology is the integration of the biologic and sociocultural factors that may influence health outcomes. One way to approach this challenge is to identify specific factors that may contribute to disease causation and their potential interactions. For example, one might consider the role of environmental factors in disease causation, such as exposure to pollutants or infectious agents. Another approach is to consider the role of social factors, such as poverty, education, and access to healthcare, in disease causation. By considering both biologic and social factors, we can better understand the complex nature of disease causation and develop effective prevention and control strategies.

POPULATIONS AS MORE THAN COLLECTIONS OF INDIVIDUALS

An important factor which has in part limited epidemiology's ability to examine the causes of disease in populations is the "individualization" of epidemiology. By "individualization" I mean the notion that the risk of disease depends exclusively on individual-level characteristics. This notion has been reflected in the behavioral model of disease (in which disease stems from the choices and behaviors of individuals, isolated from contextual influences), and it reappears today in some aspects of the biologic model (in which disease is strongly influenced by an individual's unique genetic makeup).

Although epidemiology has often been referred to as the study of the distribution of disease in populations, much of today's epidemiology conceptualizes populations merely as aggregates of individuals (useful from a statistical point of view), rather than as groups of interacting individuals with social relationships and social organizations and with group-level properties that may partly influence risk of disease (16, 41, 44). Although much has been written in epidemiologic journals on the "ecological fallacy" (the failing of inferring individual-level associations from group-level data), there has been comparatively little mention of the fallacy inherent in focusing exclusively on the individual level without taking group-level factors into account (analogous to the ecological model of disease causation (fallacy) (45). Types of group-level variables which have recently appeared in epidemiologic analyses include, for example, income inequality (46, 47), neighborhood characteristics (48), and the spread of infection in a community (50), and contact patterns between individuals (51).

A major challenge to epidemiology today is the development of models that integrate the effects of both individual-level and group-level factors in a comprehensive manner. By examining the interactions between individuals within their groups or social contexts, that examine the interacting effects of both individual-level and group-level variables, and that take into account the role of interactions between individuals in shaping the distribution of health and disease. Recent publications on multilevel analysis (45, 52, 53)—as one analytical strategy for including variables operating at multiple levels in epidemiologic analyses—and systems analysis (40, 54)—an approach that analyzes the dynamic systems which generate patterns of health and disease in populations, allowing for interactions between components and processes—reflect a growing theoretical and methodological interest in these areas.

BEYOND INDEPENDENT EFFECTS

Many of the analytical methods used in epidemiologic today focus on the need to isolate the "independ-
POTTING EPIDEMIOLOGY BACK TOGETHER AGAIN

The epidemiologic approach focused on identifying individual-level risk factors for diseases has been fruit-
ful in increasing our understanding of many factors influencing the distribution of disease in populations, including behaviors, biologic factors, and, today, genes. These methods have also been successfully used to identify social factors involved in disease. These factors, which have been elucidated primarily through studies documenting social class differences in cardiovascular disease (56–58). Breaking things down is a useful scientific tactic with which to analyze many problems. The difficulty arises when the method itself is reduced into an ontologic stance, a "true" and "complete" representation of reality (33) (i.e., "the world is like the method" rather than "the method helps us understand some aspects of the world"). An exclusive focus on individual-level and dissectioning risk may hamper our ability to test more sophisticated (and realistic) models of diseases cause.

In a sense, today’s increasing emphasis on genes as fundamental causes of disease exacerbates the biologi-
calization and individualization of epidemiology in its maximum expression (i.e., genes as the fundamental biologic substrate and genetic makeup as a unique characteristic of individuals). To a greater or lesser extent, genes will be involved in every disease. How-
ever, for most diseases, gene expression, and indeed the degree to which genetic differences are important in understanding the disease, is influenced not just by the genetic component and dissectioning risk, but also by the way we live with each other in society (59). Genetic epidemiology and traditional risk factor epidemiology hold potential for enhancing our understanding of the causes of disease, but in isolation the picture they give us is incomplete. Analogously, social explanations of the causes of disease presuppose biologic mechanisms (e.g., genetic), biological mechanisms cannot be understood by pursuing research whose ratio-
nale is to divide and isolate the components in ever greater detail” (55, p. 29). In the extreme case, as a result of the emphasis on separating out independent effects, even the "multicausal model" is forgotten, each factor is statistically abstracted from the web or pattern of factors of which it is a part (39), and the multicausal model itself is reduced to a collection of unicausal relations (44). New analytical strategies will undeniably flow from the need to investigate hypotheses based on new models of disease causation. The examination of joint and interacting (rather than merely independent) effects operating across levels will be a part of these strategies.

REFERENCES

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