

Chapter 3.

The Concept of Fundamental Causes In Explaining Social Inequities in Health

Bruce Link, Columbia University and New York State Psychiatric Institute
Jo Phelan, Columbia University

The association between socioeconomic status and disease has been remarkably consistent across time and place. In particular the association has endured despite dramatic changes in the mechanisms thought to mediate the association and in the diseases that happen to afflict humans in any particular epoch. Whereas at one time the association between socioeconomic status and disease was attributed to the dire conditions that poor sanitation and poor housing had on diseases like diphtheria, typhoid fever and tuberculosis now it is attributed to health behaviors like smoking, exercise, and diet. The persistence of the association between socioeconomic status and disease leads us to the concept of a fundamental cause. A fundamental social cause involves resources like knowledge, money, power, prestige and social connections that determine the extent to which people are able to avoid risks for morbidity and mortality. Such resources are important for avoiding risks in widely divergent situations thereby producing the conditions for a persistent association between the fundamental cause and disease. The implication of this reasoning are: 1) that fundamental causes cannot be fully understood by tracing through the mechanisms that happen to link them to disease at a particular time; and 2) that interventions targeted at intervening mechanisms cannot obliterate the effects of fundamental causes.

In their book *Sociology in Medicine*, Susser, Watson, and Hopper (1985) make a bold statement regarding the connection between sociocultural factors and the occurrence and experience of disease. They write:

Societies in part create the disease they experience and further, they materially shape the ways in which diseases are to be experienced. Cross-cultural studies of disease consistently show that the varieties of human affliction owe as much to the inventiveness of culture as they do to the vagaries of nature. If disease is seen in its full dimensions as a phenomenon besetting persons in communities, its status as a culturally constituted reality becomes apparent. (p.17).

This statement echoes similar statements made by the founders of social medicine—people like Rudolf Virchow, a 19th century German social epidemiologist, who declared that “medicine is a social science, and politics nothing but medicine on a grand scale” (Virchow 1848). These claims stand as some of the most provocative statements concerning the importance of a truly social approach to epidemiology.

But are they justified? Note the broad scope of the claims. Virchow didn’t simply assert that conditions of poverty in 19th century Germany were leading to exposure to bad water and polluted air and that these were causing social patterning of cholera, TB and so on. He said: “medicine is a social science— here, now and always.” Nor were Susser, Watson, and Hopper’s claims limited to particular situations— to particular social conditions, risks, or diseases. They said societies— not this one at this time but societies in general— create and shape patterns of disease. They assigned a fundamental status to social conditions by claiming that such conditions will produce patterns of disease no matter what —no matter what diseases or what risks happen to exist at a particular time or in a particular cultural situation.

But, if one applies the standard way of thinking about the connection between social conditions and disease, these claims seem overdrawn in their assignment of such a fundamental status to social conditions. The standard way of thinking proceeds as follows— social conditions expose people to risk factors and those risk factors cause disease, thereby producing patterns of disease in populations. From the point of view of this standard way of understanding the association between social conditions and disease, statements assigning a fundamental quality to social conditions aren’t really warranted. From this vantage point, there are reasons why social conditions are related to disease— specific risk factors link social conditions to disease. It follows that once these risk factors are uncovered and addressed through prevention or intervention, the social patterns will be addressed. In this view, social patterns are “explained by” risk factors and thus it is the “risk factors” that are of primary importance.

This standard way of thinking is evident in most attempts to understand the association between socioeconomic status (SES) and health once the association between socioeconomic status and health—once the association is observed, the critical issue turns to a search for the connecting mechanisms. What mediates the association? Is it smoking, diet, exercise, depressive symptoms, an orientation of mastery, or what? This way of thinking dominates the effort to understand the SES-disease association. It is evident, for example, in Adler et al.'s (1994) *American Psychologist* review of the graded association between SES and health, in the empirical research of Ross and Wu (1995) who seek to explain the association between education and health and in the work that one of us (BL) has undertaken in an effort to explain the association between SES and major mental disorders (Link, Dohrenwend & Skodol 1986; Link, Lennon, & Dohrenwend 1993). And of course, there is much merit in seeking to understand the mechanisms. Doing so can lead to discoveries upon which to base prevention or intervention strategies, and when such strategies are implemented, the health of the population can be improved. Moreover, given the distal position of SES in a causal chain, there must always be intervening mechanisms that provide the connecting link to disease. There is, then a reasonable and straightforward rationale for investigating mechanisms.

However, if the goal is to understand why there is an association between SES and health, we see distinct limitations to a narrow focus on mechanisms that happen to link SES to disease at a particular time. Instead, we have employed an approach that centers around a concept that we have called “fundamental social causes” of disease. Concentrating on social conditions as fundamental causes not only provides a different understanding about why the SES-health association exists, it also provides an explicit rationale for the broad and powerful claims made by Virchow and by Susser, Watson, and Hopper.

The Fundamental Cause Concept

As we define it, a fundamental social cause involves resources like knowledge, money, power, prestige, and social connections that determine the extent to which people are able to avoid risks for morbidity and mortality. Because resources are important determinants of exposure to risks, fundamental causes are linked to multiple disease outcomes through multiple risk-factor mechanisms. Moreover, because social and economic resources can be used in different ways in different situations, fundamental causes have effects on disease even when the profile of risk factors changes radically. It is because such social conditions have persistent effects on health even when there are dramatic changes in mechanisms that we call the “fundamental” social causes.

In developing the fundamental cause concept, we begin with a consideration of the association between SES and health. House et al (1990) have cogently stated the idea that SES might have a persistent effect on health despite changes in mechanisms. In discussing the results of their analysis of mechanisms linking SES to health across the life course, they caution that such mechanisms may not explain the association, because:

the variety of advantages in power, prestige, knowledge, and monetary resources that accrue to members of higher SES strata may repeatedly enable them to avoid health hazards more readily or to mobilize health protective factors, no matter what hazards or protective factors are most important at a given time (p. 406).

The history of inquiry into SES and health strongly supports this view.

Nineteenth-century physicians who founded the field of social medicine observed a strong association between indicators of poverty and ill health. At that time, the reasons for the powerful association clearly appeared to reside in the dire housing, sanitation, and work conditions of poor people. With tremendous medical advances and extensive public health initiatives, the incidence of such diseases as diphtheria, measles, typhoid fever, tuberculosis, and syphilis declined dramatically. In addition, modern welfare states have substantially increased poor people’s access to medical care. By the 1960s, many of the factors previously identified as linking SES to disease has been addressed, and one might have expected the association to wane and perhaps disappear altogether.

Indeed, this is exactly the conclusion that sociologist Charles Kadushin reached in 1964. Startled that social scientists had not recognized the demise of the SES gradient in health, Kadushin (1964) reminded his readers that most of the mechanisms thought to produce SES differences in health in the United States have been addressed and that:

...as countries advance in their standard of living, as public sanitation improves, as mass immunization proceeds and as Dr. Spock becomes even more widely read, the gross factors which intervene between social class and exposure to disease will become more and more equal for all social classes (p. 75).

As a result, Kadushin declared, Americans from the lower classes are no more likely to develop disease than those from the middle or upper classes.

Of course, Kadushin's prediction turned out to be dramatically incorrect as indicated by many recent studies that document an enduring or even an increasing association between SES and many disease outcomes (Adler et al, 1994; Pappas, 1993; Singh and Yu, 1995; Sorlie, Backlund, & Keller, 1995). But what was wrong with Kadushin's reasoning? Hadn't he engaged in logic that most of us not only accept but take for granted? Having implicitly drawn the path model with SES as the distal factor that is linked to disease by more proximal risk factors and having observed that the proximal risk factors in the model had been largely eliminated as causal agents, he concluded that the SES-disease association should have disappeared. But it didn't.

On the face of it, the reason Kadushin's 1964 prediction turned out to be wrong is readily apparent when one compares the intervening risk factors he considered to the ones identified by Adler et al (1994). . The "gross" risk factors of sanitation and immunization that Kadushin mentioned are replaced in the Adler et al. (1994) review by risk factors that include smoking, exercise, and diet, among others. Further, the evidence suggests that several of the risk factors mentioned by Adler and colleagues were not important intervening mechanisms when Kadushin wrote. For example, the association between SES and smoking emerged only during the 1960s, when people of higher SES became less likely to start smoking and more likely to quit if they had started. Thus, studies of the association between SES and disease over the past several decades reveal an important fact: as some risk factors mediating the association between SES and disease were eradicated, others emerged. Consequently, the association itself has endured.

We propose that the reason for such persistent associations, and the essential feature of fundamental social causes, is that they involve access to resources that can be used to avoid risks or minimize the consequences of disease if it occurs. We define resources broadly to include money, knowledge, power, prestige, and interpersonal resources such as social support and social networks. People who are advantaged with respect to these resources may benefit both individually, as when money allows an individual to move away from a polluted environment, and collectively, as when money directs society's resources toward the development of medical procedures that only the wealthy can afford.

In order for fundamental causes to become apparent, there must be change over time in diseases, treatments, risks and/or knowledge about risks. If no new diseases like AIDS emerged, no new risks like pollutants developed, no new knowledge about risks like cigarette smoking emerged, and no new treatments like neonatal intensive care were developed, the concept of fundamental social causes would be moot. In such a static system, if risk factors known to intervene between a social cause and disease were blocked, the association between the social cause and disease would decline in lockstep. But, of course, this is nothing like the situation humans have confronted with regard to health. In the context of a dynamic system, fundamental causes are likely to emerge. The reason is that the resources embodied in fundamental causes are transportable from one situation to another, and as health-related situations change, those who command the most resources are best able to avoid disease and their consequences. Thus, no matter what the current profile of diseases and known risks happens to be, those who are best positioned with regard to important social and economic resources will be less afflicted by disease.

As this reasoning indicates, the main prediction concerning fundamental causes is about the gradient in health outcomes. We propose that because resources are transportable from situation to situation the gradient will re-emerge no matter what the conditions are. This means that great improvements in overall levels of health can be achieved even as fundamental causes (as we have defined them), reproduce social inequalities in health. It is in no way disconfirming of our idea to show that an individually based health behavior (or any other factor) improves overall levels of health. What would be disconfirming is evidence that the effect of a fundamental cause on the gradient in health can be permanently eradicated by altering health behaviors.

Our reasoning also suggests two further attributes of fundamental causes. Because a fundamental cause involves access to broadly serviceable resources, it influences multiple risk factors and multiple disease outcomes. Consequently, the association between a fundamental cause and disease can be preserved through changes either in the mechanisms or in the outcomes. With respect to mechanisms, Lieberman (1985) proposed that some causes, which he called “basic causes,” have enduring effects on a dependent variable because, when the effect of one mechanism declines, the effect of another emerges or becomes more prominent. In this regard we have already described the emergence of smoking, diet and exercise as factors mediating the association between SES and disease. While these variables were no doubt always associated with disease, their connection to SES changed when their importance for health became known.

With respect to disease outcomes, social epidemiologist John Cassel (1976) points out that some social factors make individuals vulnerable, not to a specific disease, but to a wide array of diseases. Since most studies measure only one disease outcome, the full impact of a social cause goes unrecorded – a point that has also been made by Aneshensal, Rutter, & Lachenbruch (1991) with their distinction between sociological and socio-medical approaches. However, in addition to underestimating the full impact of social causes at any given time, a narrow focus on one disease at a time misses the possibility that changes in particular diseases outcomes can contribute to enduring associations between fundamental causes and disease overall.

When health surveillance or immunization systems fail and old diseases (e.g., tuberculosis, measles) begin to reemerge or when new diseases (e.g., AIDS) enter a population, they do so in the context of existing social conditions that are ripe environments for producing mechanisms linking fundamental social causes to those diseases. Thus, for example, before 1980, SES was linked to injection drug use, which in turn had negative health consequences. But with the emergence of AIDS, this SES-linked risk factor came to have an even more potent effect on health. Indeed, AIDS will likely become a significant contributor to SES differentials in mortality in the future because of the rapid spread of infection in low-income areas.

Theoretical Expansion of the Fundamental Cause Concept

In the foregoing exposition of the fundamental social cause concept, it is undoubtedly clear that we have SES in mind as a potential fundamental social cause. Critical to the definition of fundamental social causes is the idea of *resources* that are serviceable in many situations, and SES-- with components of education, occupational standing, and income-- tells us a great deal about the resources available to people. As a result, it serves as a useful starting point for reasoning about fundamental social causes of disease. But, powerful as it is, SES does not adequately assess the full range of social resources important to maintaining health. We suggest an expansion of our conceptualization of resources by incorporating theory relating to social class (as opposed to SES), power, prestige, and social capital.

Social class. Wright (1985) and others have developed operational measures of social class conceptualized as discrete categories formed in relation to the means of production. The categorization is based on ownership of the means of production, control of industrial assets, and ownership of scarce (credentialed) skills. Focused as the categorization is on control in the economic sphere, relatively advantaged positions in the classification are likely to carry enhanced resources of power and wealth that are not captured by SES measures of education, occupational standing, and income. Indeed, empirical studies have revealed only modest overlap between SES and class measures. Although class position is likely to have effects that generalize beyond the occupational sphere, one example of the potential health benefits of industrial ownership and control is that one can choose to avoid hazardous occupational conditions (so long as they are known), no matter what those hazards happen to be.

Power. Power may give health advantage to individuals in many ways, for example, by affording them access to superior medical care. Power, however, may be an even more important resource at a broader societal level, where it may serve to shape the health care system and research agenda to meet the health needs of powerful groups. For example, there has been concern that relatively more research and intervention effort has been accorded to cardiovascular diseases because they tend to strike men-- who are a relatively more powerful group than women-- and at an age when the men are at the peak of their careers.

Prestige. A third resource is prestige, with social honor at one end of the continuum and stigmatization at the other. As conceptualized by Goode (1978), for example, prestige is an unequally distributed resource, associated with but distinct from SES, social class, and power. There are at least two ways in which prestige might operate as the kind of general resource implied by the fundamental-cause concept. First, it may serve as a personal commodity that can be traded in social exchanges for an array of more directly health-benefiting resources. For example, doctors' own prestige may be enhanced by that of their patients, improving the quality of care available to high-prestige persons, while making it correspondingly difficult for low-prestige persons, including those from stigmatized groups, to receive adequate care (e.g., Link, 1983). Second, and potentially even more consequential, evidence from studies of humans and other primates have recently been interpreted (Adler et al 1994; Evans, Borer, & Marmor 1994) as suggesting that hierarchy per se may have an important impact on health, through processes involving stress, self-esteem and social support. This line of thinking is supported by work one of us (BL) has done on the stigma of mental illness, which demonstrates the deleterious effects of stigma on a variety of social, psychological and economic outcomes, including self esteem and social networks (e.g. Link, Cullen, Struening, Shrout, & Dohrenwend 1989).

Social Capital. According to James Coleman (1988), social capital refers to aspects of a social structure, or relations among individuals, that-- like physical and human capital-- facilitate the attainment of goals. There are several reasons to think this concept can be fruitfully applied to understanding the attainment of health and integrated with the concept of fundamental causes. First, there is substantial evidence that certain aspects of relations among individuals, such as social networks and social supports, are related to health and mortality (House, Landis, & Umberson 1988). Second, Coleman notes that social capital, once accumulated, can be appropriated to achieve goals unrelated to the purpose or context in which it was originally developed. Thus, social capital is the type of general and broadly useful resource implied by the concept of fundamental causes. For example, it is likely that social supports and the application of social norms are important in shaping health behaviors (e.g., diet), encouraging adherence to medical regimens (e.g., blood pressure medication), and dealing with life stress (House & Kahn 1985), no matter what the relevant behaviors, regimens, or stressors happen to be for a particular health outcome at a particular time.

The main point is that social resources like SES, social class, power, prestige, and social capital, because they are broadly serviceable, will shape the social distribution of disease at many time points and within the context of many societies. Moreover, the use of this broader array of resources-- broadened from SES alone-- may also help us understand other aspects of the social patterning of disease that tend to endure through time, such as by race, gender, age, and marital status. When confronted by associations between such status characteristics and health, we have the same impulse-- to search for the risk factors that “explain” the association. But to the extent that such characteristics are associated with broadly serviceable resources, their association with disease may be expected to endure through time as well. We may not be able to achieve a full understanding of these associations if we focus only on the risk-factor mechanisms that link these characteristics to disease at a particular time or in a particular context.

Validity

Having explicated our ideas about fundamental causes, we now turn to considerations that reflect on the plausibility of the fundamental-cause idea and to ways of further testing its validity. The main competitor to the fundamental cause idea is what we have called the “mechanism-based approach.” This approach calls for a broad-scale delineation of the possible mechanisms linking a social condition (like SES) to disease. In this approach, an “understanding” of the association will be achieved when we have undertaken the necessary research and found the mechanisms that explain the association. With such knowledge in hand, the mechanisms can be altered and the effects of the social condition ameliorated or eradicated. The fundamental-cause concept involves a very different understanding of the association between social conditions and disease. While the study of mechanisms-- including health behaviors, psychological orientations and biological attributes-- is clearly important in its own right, the fundamental cause concept predicts that a focus on mechanisms will ultimately fail to account for associations between fundamental causes and disease.

But how might we test the validity of these two different views? In a static system, the two perspectives are equal as explanations; they both fit the facts. The critical ingredient for separating the two ways of understanding the SES-disease association is change in risk factors, diseases or health care systems. According to our reasoning, associations between fundamental causes and diseases are reproduced under conditions of change. Thus, it is in the close observation of dramatic changes in such factors that we will find instances in which the mechanism-based approach and the fundamental-cause approach make different predictions about observable facts.

First consider changes in risk factors. According to the mechanism-based approach, the risk factors exist and simply need to be discovered and addressed through prevention or intervention. According to the fundamental-cause concept, associations between risk factors and fundamental causes will change when new knowledge and ways of avoiding risks emerge. Thus the fundamental-cause approach, but not the mechanism-based approach, predicts that the association between fundamental causes and risk factors will become stronger as information about risk factors and ways of avoiding them grow. As already mentioned, this seems to have happened with respect to smoking, but the fundamental-cause approach can be further tested by examining such factors as hypertension, cholesterol, diet, and exercise.

Another area of change concerns changes in health care systems and health care delivery. In this century, a major change took place in this factor-- presumed to be a critical intervening mechanism between SES and health, when most of the developed countries adopted health care plans that provided care to all citizens regardless of their ability to pay. As a result, the use of health care services in these countries has increased and become more equally distributed across levels of SES. Yet in general, SES differences in health in these countries have not decreased. These facts are puzzling from the perspective of a mechanism-based approach that focuses on identifying and modifying intervening mechanisms. If medical care were an important factor mediating the association between SES and health, then surely providing treatment of all citizens should have reduced the association. This paradoxical set of facts has led others (e.g., Evans et al., 1994) to conclude that the essential determinants of health must lie primarily outside the health care system. However, by focusing on the importance of resources in influencing the distribution of health care, the fundamental-cause concept may allow for an explanation of these findings that is consistent with the intuitive and empirically supported (e.g., Lee et al., 1995) idea that health care is indeed beneficial for those who receive it.

Consider an example regarding changes in the health care system in the United States. There is a longstanding association between SES and both low birth weight (LBW) and infant mortality. Between 1966 and 1979, a system of neonatal intensive care units (NICUs) was designed to improve the chances of survival for LBW infants. Numerous evaluation studies have produced widespread agreement concerning the effectiveness of NICUs in reducing mortality (Lee et al., 1995). Before NICUs, the decline in the infant mortality rate had stalled in the United States during the period from 1950 to 1965. After their implementation, dramatic improvements in infant mortality emerged and have continued through the early 1990s (Singh & Yu 1995). Moreover, most of the decline in infant mortality is attributable to the survival of LBW infants rather than to a decline in the incidence of low-weight births (Lee et al., 1995). Despite the widespread implementation of this powerfully effective intervention to address a risk factor that disproportionately afflicted people of low SES, trends in infant mortality show that the association between SES and infant mortality has not declined (Singh and Yu 1995). Although this result is surprising from the perspective of a conventional mechanism-based approach, it is exactly the result that would be predicted from the perspective of fundamental causes. The NICUs represent a beneficial change that anyone giving birth to a LBW infant would want. From a fundamental-cause perspective, one would hypothesize that people with more resources are better able to access and benefit from NICUs than people with fewer resources-- an hypothesis that could be tested as a further step in assessing the validity of the fundamental-cause approach.

Finally, we address a major change in disease-- the AIDS epidemic. According to our reasoning about fundamental social causes, when new diseases emerge in a population, people with greater resources are better positioned to avoid exposure to risks for the disease. In some cases, the risks (e.g., crowded living conditions) may already be higher among people with fewer resources. In other cases, this may not be so initially; rather, an association between fundamental causes and the new disease develops over time as advantaged people use their resources to escape the risks once they become known. Is this reasoning supported in the case of AIDS?

In this country, the two largest risk groups for this disease are gay or bisexual men and injection drug users (Centers for Disease Control, 1994). In examining the resources of these two groups, it is notable that both groups are stigmatized, limiting their prestige-related resources. However, gay men are advantaged over injection drug users in terms of two important resources. Injection drug users, especially those who share needles, tend to have very poor socioeconomic resources, while gay men do not. Moreover, the gay community has abundant social capital in the form of informal social networks and formal social and political organizations. When the AIDS epidemic emerged, these social resources were used to educate gay men about risks, establish norms of safe sex, and lobby for research and public health interventions. Thus, our ideas about fundamental causes lead us to predict that over time, as the risks became understood, gay men would become more successful at avoiding infection than injection drug users. This appears to be precisely what has happened. Data regularly collected by the Centers for Disease Control (e.g., HIV/AIDS Surveillance Reports) show that the proportion of incident cases attributed to sex between men has dropped steadily, while the proportion attributed to injection drug use continues to rise.

Several further tests of the fundamental-cause idea can be conducted in relation to the AIDS epidemic by using existing literature. For example, the importance of SES and social capital will be supported to the extent that: (1) greater research and public health efforts have been directed at understanding and modifying risky sexual behavior between men than risky drug-related behavior; (2) knowledge and modification of relevant risk-related behaviors are greater among gay men than among injection drug users, and among those of higher SES within each risk group; and (3) among gay men, greater embeddedness in the gay community is associated with more knowledge and behavior change.

The Implication of Changing Fundamental Causes Themselves

The situations we just described-- changes in risk factors, healthcare systems, and diseases-- enhance the plausibility of the fundamental-cause idea and give us possible ways to test its validity further. But suppose we were able to change fundamental causes and reduce inequality-- what would the health consequences be?

One possibility is that addressing fundamental causes simply redistributes benefit such that health improvements for initially disadvantaged people are mirrored by losses for people who were initially advantaged. This might occur if the main reason for the SES gradient is that there is something about hierarchy per se that is beneficial for those with high rank and harmful to those with low rank. Although narrowing the degree of inequality might come closer to achieving equality in health, there would be no overall health gain for the population.

A second possibility is that reductions in inequality with regard to fundamental causes would lead to overall improvements in health. Wilkinson (1992) obtained support for this hypothesis in an ecological study comparing life expectancy in developed countries. Countries in which a greater proportion of income goes to the bottom 70% of the distribution have significantly longer life expectancies even when per capita income is controlled. Moreover, time trends suggest that changes toward more equal income distribution are associated with more rapid gains in life expectancy. These findings cohere with our ideas about how fundamental causes shape societal response to disease. When inequality is great, medical intervention and prevention strategies tend to “follow the resources,” and the ones that are developed and implemented tend to be ones that benefit the advantaged. When there is less inequality, the prevention and intervention approaches that are developed and implemented are ones that are more broadly applicable and therefore help more people.

Policy Implications

In introducing Healthy People 2000, former Surgeon General Louis Sullivan included the following statement: “The correlation between poor health and lower socioeconomic status has been well documented, but that does not make it right or inevitable. Good health should not be seen, or, for that matter, be permitted to exist in fact, as a benefit for only those who can afford it; it should be available to every citizen.” The policy relevance of the fundamental-cause idea resides in what it tells us about how this important ideal might be achieved.

Theoretically, effects of fundamental causes can be altered only by addressing the fundamental causes. If the fundamental-cause concept is supported in the future, two sorts of policy questions will arise. First, mechanism-based interventions that claim to address the effects of fundamental causes will have to be viewed with extreme caution. Will the intervention truly address inequities in health or will it simply shift the playing field upon which social advantage is translated into health advantage?

Second, the fundamental-cause concept, with its emphasis on social inequality, points to the relevance of policies not explicitly designated as health policies for addressing inequities in health. Although they are rarely discussed with reference to their health implications, many policies that form a major part of the national agenda,-- such as the minimum wage, housing for homeless people, capital-gains taxes, parenting leave, and Head Start programs-- have a direct bearing on the extent of inequality in our society and thus on the extent to which people from different social circumstances have access to health-related resources. While such policy initiatives often lie outside the realm of influence and expertise of health-policy analysts, a fundamental-cause perspective would induce us to explore ways in which health-policy experts might become involved in and contribute to policy debate about these larger issues.

Conclusion

At the outset of this paper we quoted both Virchow and Susser, Watson, and Hopper regarding the fundamental importance of socio-cultural factors in shaping disease patterns. We also pointed out that the conventional approach to understanding why social conditions are related to disease-- an approach we have come to call a mechanism-based approach-- shifts primary attention away from the social condition to the intervening mechanisms that are thought to explain it. From such a vantage point, the broad statements of Virchow and of Susser and colleagues are overstatements, because once the mechanisms are addressed, the effects of the social condition are accounted for; there is no reason to make broad claims about socio-cultural factors. But from the vantage point of the fundamental-cause concept, there is nothing overdrawn about the idea that societies shape the experience of disease-- across cultures and through time. From a fundamental-cause perspective the statements of Virchow and of Susser and his colleagues seem not only plausible but also extremely compelling.

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