

PART II



*Promoting Population
Health and Reducing
Disparities*

Fundamental Sources of Health Inequalities



BRUCE G. LINK AND JO C. PHELAN

The primacy of social conditions as determinants of health has been observed for centuries. The idea was forcefully articulated by nineteenth-century proponents of “social medicine,” who noted strong relationships between health and the dire housing circumstances, poor sanitation, inadequate nutrition, and horrendous work conditions that poor people encountered at that time. This social patterning of ill health led to Virchow’s famous declaration that “medicine is a social science” and “politics nothing but medicine on a grand scale” (1848). The idea is also prominent in the work of McKeown, who focused attention on dramatic secular trends toward improved population health (1976). The McKeown thesis, as it has come to be called, states that the enormous improvements in health experienced over the past two centuries owe more to changes in broad economic and social conditions than to specific medical advances.

Nevertheless, this perspective has not always been prominent. In the late twentieth century, the rise and influence of “risk-factor epidemiology” focused attention on individually based biological and behavioral risks for ill health. While this perspective has been enormously successful in providing information that has helped reduce individual risk, and thereby improve population health, its dominance has also helped downplay social conditions as important causes of ill health. Social factors came to be seen not as causes but as clues—starting points in the search for “true” causes that were seen to reside in individual health behaviors and the biological mechanisms that produce pathogenesis. Reflecting this trend, Rothman’s influential text on modern epidemiological methods indicated that social class is “causally related to few if any diseases but is a correlate of many causes of disease” (1986, 90).

But risk-factor epidemiology has recently experienced its own crisis, criticized from within for its rote “black box” approach, and for having run out of large risk factors to uncover (Susser and Susser 1996). While numerous factors have contributed to this crisis, two major problems have been the approach’s inattention to multiple levels of influence and its inability to understand empirical associations between population characteristics and the health profiles of populations.

For example, both the behavioral and biological risk factors identified by this approach have generally failed to account for or explain gradients in morbidity and mortality associated with socioeconomic status (Lantz et al. 1998; Marmot et al. 1991).

In the context of these problems with risk-factor epidemiology, a revitalization of interest in social and economic factors in health has occurred within social epidemiology and medical sociology. For example, investigators have turned intense attention to macro-level influences on health such as income inequality, social cohesion, and racial segregation and discrimination (Lynch et al. 2000; Kaplan et al. 1996; Kennedy, Kawachi, and Prothrow-Stith 1996; Kawachi et al. 1997; Williams and Collins 2001; Williams, Neighbors, and Jackson 2003). Researchers have also sought to assess multiple levels of social and economic influence, particularly the effects of neighborhood and community-level factors (Diez-Roux et al. 2001; Robert and Li 2001). Finally, analysts have directed intense effort to understanding how these distal causes affect more proximal influences on health to produce pathogenesis, disease, and death (Adler et al. 1994).

Like many of these lines of investigation, the theory of fundamental causes arose in response to the dominant risk-factor approach (House 2002). Instead of social conditions as mere correlates or clues pointing the way to true causes, we claim that social conditions are fundamental causes of health inequalities. We go beyond prior statements regarding the prominence of social factors to indicate why social conditions deserve to be called fundamental and why risk-factor approaches are unsuccessful in accounting for the persistence and pervasiveness of associations between social conditions and health.

Our approach claims that some types of policy interventions will be far more effective in reducing health disparities than others. First, according to the fundamental-cause explanation, social inequality produces health inequality, and thus policies that reduce social and economic inequality will reduce health inequality. Second, policies that benefit people irrespective of individual resources or initiative (for example, fluoridating water versus brushing with fluoride toothpaste) will be more effective in reducing health disparities than policies that require individuals to marshal resources to obtain health benefits. Third, we hold that policies that attend to the social distribution of knowledge about risk and protective factors—and the ability to act on that knowledge—are essential.

Social Conditions as Fundamental Causes

We use socioeconomic status (SES) to exemplify the theory of fundamental causes, although the idea may also pertain to circumstances such as social capital, social stigma, and racism. We begin with the well-established and robust association between mortality and educational attainment, occupational standing, and income (Antonovsky 1967; Sorlie, Backlund, and Keller 1995; Kunst et al. 1998). Biological mechanisms are clearly involved in the SES-disease association. Just as clearly, other mechanisms involving behaviors and environmental exposures must also be present: disease does not flow directly from income, educational, or occupational status into the body. Nevertheless, we cannot understand the effect of SES

on mortality by focusing solely on the mechanisms that happen to link the two at any particular time.

To show why, we turn to one of the most striking features of the SES-health association: its persistence across time and place. The association was present in Mulhouse, France, in the early nineteenth century, in Rhode Island in 1865, in Chicago in the 1930s, and occurs in Europe and the United States today (Antonovsky 1967; Sorlie, Backlund, and Keller 1995; Lantz et al. 1998; Kunst et al. 1998). Given the vast differences in life expectancy, risk factors, diseases, and health care systems characterizing these different places and times, the persistence of the SES-mortality association is remarkable. Indeed, it is this persistence that suggests the irreducible nature of SES as a fundamental cause.

Imagine a causal model with SES as the distal factor linked to death by more proximal risk factors. If the proximal risk factors are eliminated, we would expect the SES-mortality association to disappear. However, in several important instances SES disparities in mortality persisted even though major proximal risk factors were eliminated. As a first example consider circumstances in the nineteenth century in which overcrowding, poor sanitation, and widespread infectious diseases such as diphtheria, measles, typhoid fever, tuberculosis, and syphilis appeared to explain higher mortality rates among less-advantaged persons. But the virtual elimination of those conditions and diseases in developed countries in the late twentieth century did not diminish SES inequalities in mortality (Rosen 1979).

As a second example consider that in the twentieth century, countries created national health programs providing free medical care to all with the express purpose of radically altering an important link between SES and health—differential access to care. While such programs addressed important mechanisms and may have kept disparities from growing even larger than they have, SES disparities in mortality nevertheless remained undiminished decades after these programs were implemented (Black et al. 1982). In both examples mechanisms explaining the SES mortality association were dramatically modified. The causal-model approach would predict a substantial reduction in the association between SES and mortality, but that did not happen.

A ready answer to this puzzle is that other mediating risk factors, such as health behaviors and psychosocial stress, have replaced earlier ones. This situation calls to mind Lieberman's description of "basic causes," which have enduring effects on a dependent variable because when the effect of one mechanism declines, others emerge or become more prominent (1985). House and colleagues first suggested that such a process might explain the enduring SES-mortality association (House et al. 1990). Still, although Lieberman's notion of basic causes is critical for understanding the tenacity of the SES-mortality association, it does not tell us what about SES allows it to reproduce its effects even as intervening mechanisms are eliminated.

We have argued that new mechanisms arise because persons higher in socioeconomic status enjoy a wide range of resources—including money, knowledge, prestige, power, and beneficial social connections—that they can utilize to their health advantage (Link and Phelan 1995). Such resources are important in at least

two ways. First, they directly shape individual health behaviors by influencing whether people know about, have access to, can afford, and are supported in their efforts to engage in health-enhancing behaviors. Second, resources shape access to broad contexts such as neighborhoods, occupations, and social networks that vary dramatically in associated risk profiles and protective factors. Housing that poor people can afford is more likely to be located near noise, pollution, and other noxious conditions; blue-collar occupations tend to be more dangerous than white-collar occupations; and high-status jobs are more likely to include health care benefits. Thus the processes implied by the fundamental-cause perspective operate at both individual and contextual levels.

As a result, socioeconomic resources shape access to a broad range of circumstances that affect health. Examples include gaining access to the best doctors; knowing about and asking for beneficial health procedures; having friends and family who support healthy lifestyles; quitting smoking; getting flu shots; wearing seat belts; and eating fruits and vegetables. Other examples include exercising regularly; living in neighborhoods where garbage is picked up often, interiors are lead-free, and streets are safe; having children who bring home useful health information from good schools; working in safe occupational circumstances; and taking restful vacations.

This reasoning introduces four essential components of the theory of fundamental causes of morbidity and mortality. First, such causes influence multiple disease outcomes. For example, SES was related to cholera, tuberculosis, and diphtheria in the nineteenth century and is now related to heart disease, stroke, and many types of cancer. Second, such causes operate through multiple risk factors, including but not limited to the items listed above. Third, new intervening mechanisms reproduce the association between fundamental causes and mortality over time. Finally, the “essential feature of fundamental social causes is that they involve access to resources that can be used to avoid risks or to minimize the consequences of disease once it occurs” (Link and Phelan 1995, 87).

Because these resources are general in nature, people can adapt them to changing health-related conditions and use them to protect health no matter what the risks, treatments, and diseases are in a given situation. Thus, for example, socioeconomic resources were equally as useful in avoiding the worst sanitation, housing, and industrial conditions of the nineteenth century as they are in shaping access to health-promoting conditions today. As new discoveries expand our ability to control disease processes, the list of health-enhancing circumstances will only grow. According to our theory, people who command more resources will, on average, hold an advantage in gaining access to and benefiting from this new knowledge.

Evidence for the Fundamental-Cause Theory

An Empirical Test

Phelan and colleagues constructed a test of the theory of fundamental causes by identifying situations where even the richest and most powerful people on earth cannot use resources to escape death. One such situation occurs in the case of po-

tentially fatal diseases that we do not know how to prevent or treat. If the utilization of resources is critical in prolonging life, then in circumstances when resources associated with higher status are useless, high SES should confer little advantage, and the usually robust SES-mortality association should be reduced.

Phelan and colleagues tested this prediction using the National Longitudinal Mortality Study and ratings they developed of the preventability of death from specific causes (1999). The National Longitudinal Mortality Study (Sorlie, Backlund, and Keller 1995; Rogot et al. 1988, 1992) is a large prospective study that uses combined samples of selected Current Population Surveys that are then linked to the National Death Index to determine occurrences and causes of death in a follow-up period of approximately nine years. Reliable ratings (intra-class correlation .85) of the preventability of death were made by two physician-epidemiologists. Causes were categorized into high-preventability and low-preventability groups with common high-preventability causes being cerebrovascular diseases, chronic obstructive pulmonary disease, ischemic heart disease, malignant neoplasm of the trachea, bronchus, and lung, and pneumonia and influenza; and common low-preventability causes being arrhythmias and malignant neoplasms of the pancreas, female breast, and prostate. Gradients according to SES indicators of education and income were then examined separately for high—and low-preventability causes. Consistent with predictions derived from the fundamental-cause theory, Phelan et al. found that the SES-mortality association is much stronger for highly preventable causes of death than for less preventable causes (1999).

Resources versus the Stress of Hierarchical Position

Unlike the fundamental-cause theory, which strongly emphasizes the role of resources, British sociologist Richard Wilkinson argues that the “psychosocial effects of social position” influence the SES gradient more than material conditions such as “bad housing, poor diets, inadequate heating” (1997, 591). Wilkinson believes that the anger, resentment, and envy associated with where one stands in relation to others—along with maladaptive behaviors such as smoking, drinking, and overeating that follow from these emotions—are the most important determinants of SES gradients in health. Support for this view is drawn from ecological studies in industrialized nations, animal studies, and the finely graded nature of status differences in health investigations like the Whitehall study.

Even though SES gradients are apparent within countries, differences in median per capita income between countries are a relatively weak predictor of life expectancy (Wilkinson 1992; Lynch et al. 2000). For Wilkinson, this suggests that relative deprivation is more important than absolute differences in deprivation. Whether conducted in the wild or in captivity, studies of monkeys generally find that a lower position in a dominance hierarchy is associated with worse health, as indicated by measures of atherosclerosis, hyper-secreted cortisol, blood pressure, and immune function (Sapolsky 1990; Shively and Clarkson 1994). Finally, even within the relatively stable employment of the British civil service, and given extensive controls on behavioral and biological risk factors, the Whitehall study found finely graded differences in mortality by occupational level. Because hierarchical

position remained a prominent predictor of mortality, and because material deprivation was largely absent in this group of men, the study seems to support the centrality of hierarchy for understanding SES gradients in health.

But the hierarchy-stress explanation cannot account for important changes in the association between SES and specific diseases. According to such an explanation, the stress of low positional location should remain relatively constant across cohorts and therefore produce relatively stable associations between positional location and disease outcomes in different eras. However, evidence concerning two major killers—coronary artery disease and lung cancer—runs counter to this prediction. The link between SES and coronary heart disease shifted dramatically over the last century, changing from a direct to an inverse association (Beaglehole 1990; Marmot, Kogevinas, and Elston 1987). And whereas lung cancer mortality was not related to SES as late as 1931, a large inverse association emerged in the 1950s and 1960s (Logan 1982).

If positional location is the prime determinant of SES gradients in health, one would not expect such dramatic changes; the stress of low position should produce the same inverse gradient across time. On the other hand, from a fundamental-cause perspective, these shifts represent important instances in which SES gradients for specific disease outcomes changed after knowledge about health risk and protective factors emerged. High-SES groups use this new knowledge and its benefits disproportionately, resulting in shifts in the SES-disease/mortality gradient that benefit higher-SES groups.

Intelligence: A Competing Flexible Resource?

Once we have the idea that broadly serviceable resources are required to understand the persistence and the ubiquity of the association between SES and health, we see that a resource other than the social resources identified in the fundamental-cause approach is possible. Intelligence or cognitive ability can also be conceptualized as a broadly serviceable resource that enhances people's abilities to deal with life situations, including situations that have health implications. In seeking to maximize one's chances for a healthy life, one must be able to gain access to information, identify the most salient aspects of one's health situation, and craft an effective approach to addressing it. A case can be made that someone who is more gifted with respect to cognitive ability will fare much better than someone who is not. Moreover, as with other fundamental social causes, people can use intelligence to gain a health advantage no matter the health circumstances of a particular place or time. Because it can be conceptualized in this way, many of the theoretical predictions one might make from a fundamental-social-cause perspective could just as easily be made from a perspective that emphasizes intelligence. Indeed, this is precisely what Linda Gottfredson does in a paper published in the *Journal of Personality and Social Psychology* (2004). By collecting evidence from disparate sources, Gottfredson makes the argument that "general intelligence" may be the fundamental cause of health inequalities. While the evidence she gathers is consistent with this possibility, none of it involves direct measures of cognitive ability, SES, and health.

The research issue concerning the role of cognitive ability is relatively straightforward. In our formulation, social and economic resources of knowledge, money, power, prestige, and beneficial social connections are critical, whereas for Gottfredson the psychological resource of intelligence is the source of both the socioeconomic-related resources and health. Critical facts that separate these two interpretations hinge on the importance of cognitive ability for health with SES controlled and the role of SES for health with cognitive ability controlled. To investigate these relationships, Link, Phelan, and Meich located two large public-access data sets—the Wisconsin Longitudinal Study and the Health and Retirement Survey—that provide the requisite measures of SES and IQ and allow us to examine relationships prospectively (2003).

Link and colleagues found no evidence to support the idea that cognitive ability might supplant socioeconomic-related resources of knowledge, money, power, prestige, and social connections in the fundamental-cause framework (Link, Phelan, and Meich 2003). Specifically, in examining mortality and life-threatening illnesses, the investigators found that the effects of education and household income remained significant and were changed only slightly by controls for cognitive ability. In sharp contrast, measures of cognitive ability—though related to health at the bivariate level—declined dramatically, generally to nonsignificant levels, when educational attainment was controlled.

Thus while cognitive ability plays an important role in determining SES resources, it cannot account for the connection between those resources and health. Instead, the findings tell us that within levels of educational attainment, differences in cognitive ability have little consequence for health outcomes. On average, two people with comparable IQ scores, one of whom receives more education, will have different health, but two people with different IQs but the same education will have similar health.

Policy Considerations

The fundamental-social-causes approach leads to very different policies for addressing health disparities than does an individually oriented risk-factor approach. The latter promotes strategies that ask us to locate modifiable risk factors that lie between distal causes (such as SES) and disease, and to intervene in those risk factors to break the link between the distal factors and disease. By addressing intervening factors, the logic goes, we will eliminate health disparities.

But our approach points to the pitfalls of this logic and leads us to recommend policies that take a distinctly population-health perspective in addressing health disparities. Specifically, our approach points to policies that eliminate or reduce the ability to use socioeconomic advantage to gain a health advantage—either by reducing disparities in socioeconomic resources themselves, or by developing interventions that, by their nature, are more equally distributed across SES groups.

We make three general policy recommendations. First, create contextually based health interventions that automatically benefit individuals irrespective of their

own resources or behaviors. Second, prioritize interventions that are potentially available and beneficial to people at all socioeconomic levels and target the special needs of resource-poor groups who may face barriers in implementing those interventions. Third, promote policies that increase the SES-related resources available to resource-poor groups.

We assume that social inequalities in health according to SES, race/ethnicity, and other social circumstances are undesirable: that every person, whether rich or poor, black or white, top executive or manual laborer, should have an opportunity to live a healthy life. We hold that something is wrong when social positions of power and privilege determine who lives and who dies, and that efforts to ameliorate such circumstances are desirable. We emphasize this latter point because the goals of improving overall population health and decreasing health disparities may require different kinds of policy initiatives (Marchand, Wikler, and Landesman 1998). Progressive public-health-oriented interventions (such as life-saving screens for colorectal cancer and flu shots) may improve population health without addressing social inequalities in health, and may even contribute to such inequalities if life-enhancing interventions are mal-distributed by SES and other social variables (Mechanic 2002).

***Interventions That Benefit Individuals
Regardless of Their Own Resources and Actions***

In the United States we tend to emphasize both the ability of individuals to control their personal fate and the importance of doing so (Becker 1993), and thus we carry a strong orientation toward individually based solutions to health problems. But individuals frequently encounter barriers that block their capacity to maximize health. For people at lower socioeconomic levels, lack of money, awareness, understanding, time, social support for health-enhancing behavior, and optimism that adopting certain behaviors will result in a long and satisfying life can be obstacles. When we construct individually based interventions focused on diet, exercise, dental care, illness screening, and the like, we create the possibility that people with greater resources will benefit more from these interventions. This contributes to SES gradients in health. A fundamental-cause approach calls for population-based interventions that influence everyone. When we observe a health problem, we should ask how we can change the context to eliminate the problem or minimize its consequences.

Consider rising asthma rates, particularly in low-income urban areas of developed countries such as the United States (Claudio et al. 1999). Individuals can address known (or strongly suspected) modifiable risk factors and employ medical responses to lessen symptoms and keep asthma from worsening (National Institutes of Health 1997). Medical practitioners can tell individuals that asthma is less severe if homes and apartments are well ventilated and kept spotlessly clean to avoid dust mites, animal dander, and other environmental toxins. Parents can also be informed that cockroach infestation may be problematic for children with asthma and that they should try to eliminate this risk factor from their children's living space. Parents can also be exhorted to get their children away from the tele-

vision and into the open air. Finally, parents can be told to use preventive medications even when no symptoms are present to reduce the risk of hospitalizations and (rarely) death.

However, while these individually based responses would certainly be helpful to those who implemented them, such a strategy is bound to create SES disparities in the prevalence of severe symptoms of asthma. People with fewer resources are less likely to receive information regarding risk factors and to be able to pursue strategies to counteract them. Resource-poor persons are more likely to live in areas where rodents are common and are unlikely to have household help or equipment for effective cleaning. Roaches are difficult to eliminate from an individual apartment when they infest the entire building, and getting kids away from the television is difficult when open spaces are not available or safe. Finally, uninsured persons are less likely to obtain effective medical interventions to deal with chronic symptoms, and Medicaid reimbursement levels make it hard for doctors to spend enough time to provide parents with needed skills and children with quality care.

Fortunately, pressing individuals to address risk factors is not the only option. Contextually based interventions would seek to encourage city-sponsored rodent and roach reduction efforts that target entire buildings or areas, to provide sponsored activities for children in open areas such as parks, and to locate health screening and medical interventions in schools, where all children can receive them free of charge. Like individually based approaches, contextually based approaches would reduce the overall incidence of asthma symptoms and hospitalizations; but, unlike individual approaches, they would have the added benefit of reducing SES disparities.

Another contextual intervention is to employ air bags rather than seatbelts to reduce road fatalities. Seatbelt use requires each person to secure his or her own belt, and ample evidence shows that people with higher educational attainment are more likely to do so. To the extent that seatbelts are effective, highly educated people benefit more than less-educated people, thereby contributing to a gradient in a health-related outcome. Another contextual intervention concerns the decades-long buildup of lead paint in the homes of many U.S. residents. Will we move from a strategy of warning parents about the dangers of paint chips and paint dust to actually removing the hazard from the environment?

Similar examples include providing vaccinations and health screening in schools, workplaces, and other community settings rather than through private physicians, requiring window guards in all high-rise apartments versus advising parents to watch their children carefully, and adding warning labels to health-hazardous products versus relying on individuals' knowledge of product risks. Other choices include banning smoking in public buildings versus advising people to avoid secondhand smoke, thoroughly inspecting meat instead of advising consumers to wash cutting boards and cook meat thoroughly, and fluoridating water rather than exhorting people to brush often with fluoridated toothpaste. To the extent that interventions influence everyone regardless of the resources they possess or the health behaviors they manifest, we can block the creation of SES gradients: everyone

benefits equally. Creative interventions that influence entire contexts rather than individuals could go a long way toward narrowing health disparities.

Monitoring the Dissemination of Health-Enhancing Information and Interventions

Even if we become far more creative in developing population-based interventions focused on contexts, addressing many health problems will still require individual resources and action. The fundamental-cause idea tells us that resource-rich persons will be far more effective in gaining access to and employing health-enhancing initiatives focused on individuals than people who are resource poor. This means that policy approaches will need to address the consequences of individually targeted information and interventions for health disparities. Two issues are critical.

The first concerns whether we promote initiatives that people with fewer resources may not be able to access. As we seek to create interventions to respond to disease, we need to ask if an intervention is something anyone can potentially adopt, or whether the benefit is available only to people with the requisite resources. The point becomes clear if we take a global view of the medical response to the AIDS epidemic. Research, policy, and investment in the West spawned drugs that have been enormously effective in enhancing the survival and quality of life of people with HIV/AIDS in the United States and Europe. But, because of their cost, these drugs have been unavailable in poor areas of Africa and Asia, creating an enormous resource-related health disparity.

Similar circumstances exist within the United States regarding access to optimal procedures following heart attacks, control of diabetes, rapid response to strokes, and many other interventions. When we create interventions that are expensive and difficult to distribute broadly, we create health disparities. While we might proceed with such interventions because they help some people, and because expensive initiatives sometimes become less expensive with time, we must also weigh the fact that they will create disparities. If our goal is to reduce disparities, we must ask whether we could redirect our intellectual, social, and economic resources to produce a more broadly distributable health benefit.

A second issue in disseminating health-enhancing information and interventions entails understanding why people with fewer resources do not always act on information or adopt health-enhancing ways of life. We call this contextualizing risk and protective factors: that is, understanding what puts people at “risk of risk” and what blocks them from adopting protective strategies (Link and Phelan 1995). The idea is to use such understanding to construct interventions that simultaneously address a risk or protective factor and any barriers to broadly implementing it. For example, interventions that tackle diet and exercise to reduce obesity and encourage a heart-healthy lifestyle would begin with careful attention to all the life circumstances that might create barriers to behavioral change. Examples of such contextual factors include the cost, availability, and convenience of healthful versus unhealthful foods; targeted advertising of convenience and fast foods; access to safe and accessible settings in which to exercise; time for exercise; and support

for healthful habits from family, friends, and employers. Absent such efforts, the life circumstances of people with fewer resources will tend to block the adoption of beneficial information, and we will create a health disparity.

A good example of an initiative that has mandated the kind of scrutiny we endorse is the mapping of the human genome. Concerned about the impact of new genetic knowledge, including the possibility that expected benefits might be maldistributed, Congress reserved 5 percent of funding for the Human Genome Project to consider social, ethical, and legal issues (Collins et al. 2003). Whether the initiative will be successful or not remains to be seen; but in general, to the extent that we can anticipate and address factors that produce the unequal distribution of health benefits, the better able we will be to minimize health disparities.

Policies That Distribute Resources to Resource-Poor Populations

The fundamental-cause idea stipulates that people use their knowledge, money, power, prestige, and social connections to gain a health advantage, and thereby reproduce the SES gradient in health over and over again. It follows that if we increase the resources available to resource-poor populations, the relative health of those populations will likely improve. We provide several examples consistent with this prediction.

First, consider an analysis by Peter Arno and James House of the impact of Social Security in the late 1930s. Before Social Security, elders were often extremely resource poor, and death rates were very high. After the advent of Social Security, poverty declined sharply among people sixty-five and older, and death rates also dropped faster than among people under sixty-five. While these data do not prove that Social Security had a causal effect on mortality, they are certainly consistent with that interpretation. If Social Security had an impact on health, it is an indication that providing resources to a group with relatively few resources has health benefits for that group and moves its health profile toward that of groups with more resources.

Another example is the work of Costello and colleagues, who capitalized on a natural experiment where an influx of money from a casino dramatically altered the monetary resources of American Indians living in eleven rural counties in North Carolina (2003). The casino netted each man, woman, and child a monetary stipend that reached six thousand dollars a year in 2001. Costello and colleagues' study of children found substantial improvements in the mental health status (externalizing behaviors) of the children living in these communities from a baseline starting point before the influx of resources to a follow-up point about four years after the monetary stipends began.

A final example is the U.S. Department of Housing and Urban Development's Moving to Opportunity study, which randomly assigned families eligible for housing assistance to one of three groups—those that were given Section 8 vouchers, which help cover housing costs, plus special assistance in moving to low-poverty neighborhoods; Section 8 vouchers alone; or no vouchers at all. Even though only 40 percent of the families receiving assistance actually moved to new housing, the group assigned to Section 8 vouchers plus assistance in relocating to low-poverty

areas recorded lower levels of parental anxiety as well as less anxiety and depression among male offspring. Families that actually moved to better neighborhoods showed the greatest improvements (Leventhal and Brooks-Gunn 2003).

Together these studies suggest that providing resources to populations that are resource poor, such as elderly people, American Indians, and people living in areas of concentrated disadvantage, may improve their health profiles. The further up the SES gradient the infusion of resources enables people to move, the more we can expect them to benefit. While the reasons for boosting groups that are relatively resource poor go beyond health benefits, these studies suggest that providing resources to populations at risk of negative health consequences is one important way to reduce health disparities.

Conclusion

Perhaps the strongest policy conclusion we can offer is that standard risk-factor-oriented thinking about social disparities in health will fail to produce policies that can narrow such disparities. We will not be able to eliminate disparities if we focus solely on the individually based risk factors that happen to link SES and health in a given place or time. While the fundamental-cause approach points us away from policy based on a standard risk-factor approach, it points us toward other policies. We endorse three broad types: (1) policies that benefit all people in a context irrespective of their behaviors and resources, (2) policies that minimize resource-related barriers to avoiding risks or implementing beneficial interventions, and (3) policies that distribute resources to resource-poor groups. Pursuing policies like these provides the best opportunity for reducing health disparities in the time ahead.

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