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Authors
Pantell, Matthew
Adler, Nancy
Bush, Nicole

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Rigor, vigor, and the study of health disparities

Nancy Adler[a,b,1], Nicole R. Bush[a], and Matthew S. Pantell[b,c]

[a]Department of Psychiatry, and [b]Department of Pediatrics University of California, San Francisco, CA 94143-0848; and [c]University of California San Francisco–University of California Berkeley Joint Medical Program, University of California, Berkeley, CA 94720

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Health disparities research spans multiple fields and methods and documents strong links between social disadvantage and poor health. Associations between socioeconomic status (SES) and health are often taken as evidence for the causal impact of SES on health, but alternative explanations, including the impact of health on SES, are plausible. Studies showing the influence of parents' SES on their children's health provide evidence for a causal pathway from SES to health, but have limitations. Health disparities researchers face tradeoffs between "rigor" and "vigor" in designing studies that demonstrate how social disadvantage becomes biologically embedded and results in poorer health. Rigorous designs aim to maximize precision in the measurement of SES and health outcomes through methods that provide the greatest control over temporal ordering and causal direction. To achieve precision, many studies use a single SES predictor and single disease. However, doing so oversimplifies the multifaceted, entwined nature of social disadvantage and may overestimate the impact of that one variable and underestimate the true impact of social disadvantage on health. In addition, SES effects on overall health and functioning are likely to be greater than effects on any one disease. Vigorous designs aim to capture this complexity and maximize ecological validity through more complete assessment of social disadvantage and health status, but may provide less-compelling evidence of causality. Newer approaches to both measurement and analysis may enable enhanced vigor as well as rigor. Incorporating both rigor and vigor into studies will provide a fuller understanding of the causes of health disparities.

methodology | social determinants of health | cumulative risk

Good health and longevity are unequally distributed in populations. Although some unavoidable variations in health emanate from individual differences in vulnerability to disease, others are linked to membership in socially disadvantaged groups. The latter are unjust and have been the focus of considerable research and policy (1). Research on these health disparities has demonstrated consistent graded associations between various components of socioeconomic status (SES) and a wide range of health indicators; at each step up the social ladder, whether indexed by higher income, education, and/or occupational level, rates of morbidity and mortality decrease (2). The prevailing understanding of this relationship is that SES is a "fundamental cause" of health status (3), which operates through physical and/or psychosocial resources that are more readily available as SES increases, and hardships and adverse exposures that are more intense and frequent as SES decreases.

Evidence for the SES–health gradient comes primarily from cross-sectional data linking contemporaneous measures of SES in adulthood with prevalence of disease, poor health, or mortality. Smaller literatures have established associations between socioeconomic conditions in childhood and childhood health, and between childhood SES and health in later life, independent of adult SES (4–8). The strength of the associations varies in relation to the degree and chronicity of exposure to adverse conditions and is most robust for adult cardiovascular disease and mortality (reviewed in refs. 9–11).

Despite the large and growing literature linking SES and health, controversy remains over the strength of the evidence that SES *causes* disease or shortens life. Below, we discuss several challenges to interpreting the SES–health gradient as evidence that lower SES causes poorer health. These emerge from the difficulty of establishing causality between complex and changing variables and the inevitable tradeoffs between precision and control vs. ecological validity and generalizability.

Individual researchers, guided by the norms of their discipline have to balance what Herbert Kelman in 1968 termed "rigor" vs. "vigor" (12). Kelman drew on economist Kenneth Boulding's humorous depiction of a debate between proponents of each perspective, which contrasted not only methodological approaches but philosophical and social values. A chasm still exists between the rigor of reductionist research designs that emphasize maximizing precision of measures and control over causal direction vs. the vigor of both qualitative and quantitative approaches that emphasize maximizing the full depiction of both predictor and outcome variables and their actual operation in real-world settings. As we discuss below in reviewing the evidence regarding SES and health, a full understanding of the relationship of SES to health requires both rigor and vigor.

Causality and Its Discontents

Studies reporting a significant association between SES and health support the assertion that socioeconomic conditions contribute to poor health. However, there are multiple challenges to making this inference from cross-sectional findings. It could be that poor health leads to lower SES or that the association between SES and health is spurious and emerges from their joint association with a third, underlying variable. Although there have been suggestions that individual qualities, such as intelligence, time preference, or self-efficacy, could serve as confounding underlying determinants (13, 14), there is relatively little evidence for these as major explanatory variables (15).

Reverse Causation. The possibility of reverse causation, from health to SES, is a more serious concern, particularly in relation to income. Income is volatile and reflects people's capacity to work. Impaired health and functioning may reduce opportunities for income generation. For example, using longitudinal data, Smith (16) showed that declines in health status among older adults predicted subsequent drops in income and household wealth through expenditures for health care and early retirement.

Education as a marker for SES has less potential for reverse causation than does income. Education is arguably the most fundamental aspect of SES; in addition to the specific resources education provides, it indirectly affects health through increasing occupational opportunities and earning capacity. Importantly, education is relatively stable. Educational attainment is generally

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[1]To whom correspondence should be addressed. E-mail: nancy.adler@ucsf.edu.

established in early adulthood, and it is implausible that adult health could affect prior earlier educational qualifications. However, this does not rule out the possibility of health effects on education because poor health in childhood may limit educational attainment. For example, using data from a British birth cohort, Case et al. (17) found that children with more chronic conditions passed fewer of the advanced tests needed for university admission, and subsequently had lower SES and poorer health in adulthood.

Early Life Determinants. The finding that health in childhood can affect educational attainment begs the question of what determines health in childhood. Although some health problems emanate from genetic vulnerabilities or random exposures or events, childhood health is also a function of parental SES (18). Rates of many childhood illnesses (e.g., asthma, ear infections, injury, respiratory infections, conduct problems) increase as family SES decreases (4, 19), and effects of parental SES can be seen early in life. Children of low-SES parents are more likely to be born prematurely and to have low birth weight, even after adjusting for covariates (20, 21). Low-SES children are also at heightened risk for morbidity and mortality (22–24) and of cognitive and developmental delays (25–27).

Mechanisms for Transmission of Risk. Studies of "prenatal programming" point to plausible mechanisms by which parental SES can influence newborn health status as well as health in childhood and adulthood (28–30). Fetal adaptations to the intrauterine environment seem to bring about permanent changes in metabolism and physiology that affect disease risk across the lifespan. Risk factors for adverse fetal programming (discussed below) are closely linked to SES. These confer increased risk for a variety of health problems from infancy through adulthood, including overweight and obesity (31, 32); illness and antibiotic use (33); cardiovascular disorders, diabetes, and obesity (34); and mental health and behavioral problems (35, 36).

Physical Environment. Low-SES mothers live in more adverse environments, with greater exposure to such hazards as peeling lead-based paint, diesel exhaust, industrial emissions, and secondhand smoke (37, 38), coupled with poorer access to health-promoting resources, such as recreational facilities, safe environments for exercise, full-service supermarkets, and produce markets (39). Such environments may foster overweight and obesity among low-SES women entering pregnancy and excessive or inadequate weight gain during pregnancy (40, 41). These weight difficulties are associated with more adverse neonatal outcomes (42).

Stress. Lower-SES environments also engender more frequent stress responses. Although all people are exposed to some degree of stress, as one moves lower on the SES ladder, exposure to chronic, toxic stress increases (43). Both animal and human studies link greater stress experienced by the mother during pregnancy with slower offspring growth, impaired immune functioning, damage to brain structure and functioning, and a range of pediatric illnesses (31, 33, 44–46).

In addition to direct psychophysiological effects of stress on a pregnant woman and her developing fetus, behavioral responses to stress also have health implications. Many individuals cope with stress and adversity through use of cigarettes and other substances that harm fetal development (47, 48), and women with fewer socioeconomic resources are more likely to smoke both before and during pregnancy (48, 49).

Childhood Environments. After birth, conditions linked to low SES can affect a child's health and development, and the longer a child's family is poor, the more he or she is exposed to these conditions and the greater the impact on health (50, 51). Adverse conditions include residential mobility, parenting style, neighborhood crime, and the child's own exposure to toxins (reviewed in refs. 37 and 52). Although SES can affect health throughout childhood, there is evidence for sensitive periods during specific developmental stages of childhood during which children are most vulnerable to the effects of SES, depending on the exposure and outcome of interest (4).

Rigor and Vigor: Design Issues

Designs used in the study of health disparities vary in their capacity to establish causal direction.

Randomized Studies. The gold standard for determining causality is the randomized experiment. Social factors are not easily amenable to experimental manipulation, but randomization has been accomplished in a handful of trials. These have, for example, randomly provided supplemental income (sometimes tied to incentives for health-related behaviors) (e.g., ref. 53), enhanced early childcare and preschool (54, 55), and provided opportunities to move to more advantaged neighborhoods (56). Although representing the strongest methods for determining causality, most of these were designed to assess outcomes other than health, were funded by government agencies outside of the traditional health research realm (e.g., the Departments of Education or Housing and Urban Development), and have relatively sparse health data.

Randomization is easier to accomplish in laboratory studies that simulate experiences of low status. Studies have manipulated relative status within experimentally created groups and found adverse effects of low status on both psychological and physiological responses (57, 58). Others have induced relative status by manipulating whether participants compare themselves with someone of higher or of lower status. For example, Zink et al. (59) found brain patterns that reflected heightened stress responses when participants were randomized to make upward vs. downward comparisons in the context of an economic game.

Natural Experiments. Natural experiments take advantage of changes in SES that occur for one group of people owing to events tied to neither their health nor their SES. Such events include lower Social Security payments for individuals born after 1917 (50), increased income for some Native American families after establishment of a casino (61), and receipt of higher earned income tax credits in some states (62). Unfortunately, because the changes were not planned, relevant health data that can be linked to these random events are limited, and findings have been mixed.

Mediators. Other studies, although not manipulating SES, gain control over causal effects by manipulating presumed mediators of SES and health or taking advantage of natural variation in these mediators. For example, poor maternal nutrition is a presumed mechanism by which low SES affects children's health, but it may be confounded with other determinants of fetal development. Using a natural experiment—the Dutch famine during World War II—Ravelli et al. (63) provided a strong test of the role of diet and demonstrated that babies born during the 2 years of the famine had poorer glucose tolerance as adults than did those born in the years preceding or following the famine. In a study of compromised immune function associated with low SES, Cohen et al. (64) obtained data from healthy adult volunteers on both childhood and adult SES before experimentally exposing them to a rhinovirus and examining whether they developed a clinical cold. Employment in adulthood and parental home ownership in childhood predicted a lower likelihood of developing a cold after the viral exposure.

Cohort Studies. Longitudinal cohort studies can establish temporal ordering and provide important tests of prospective associations

between childhood SES and adult health. The British cohort studies, which obtained samples of children in England and Wales born in specified years and followed them through adulthood, provide some of the strongest evidence of long-term health effects of childhood conditions. Both the 1946 and the 1958 cohorts demonstrated significant associations between low childhood SES and poor health in adulthood. Significant effects persisted even after adjusting for SES in adulthood (65, 66).

Rigor and Vigor: Measurement Issues

Maximizing rigor encourages precise measurement of a limited set of variables that lend themselves to statistical testing to isolate mechanisms. Maximizing vigor encourages in-depth assessment, including use of multiple measures and/or multifaceted constructs to more fully capture key phenomena. Proponents of rigor may view the latter approaches as messy and imprecise. Proponents of vigor may argue that using a single measure cannot capture the true essence and power of a variable such as social disadvantage, which is not a singular event but an entwined set of conditions whose impact may depend on the combinations of exposures that a person experiences.

Operational definitions of SES include components of income, wealth, education, and occupation, yet the majority of studies use only one indicator. SES also intersects other bases of social stratification, such as race/ethnicity. Using a single variable as a proxy for overall social disadvantage or even as a proxy for SES makes it difficult to determine its true impact. Although poverty is often used as a single indicator, Duncan and Magnuson (67) point out, "poverty is associated with other experiences of disadvantage (such as poor schooling or being raised by a single parent), making it difficult to be certain whether it is poverty per se that really matters or related experience" (page 25).

When a single indicator is used, the health effects generated by related aspects of disadvantage may be erroneously attributed to that factor, and its role may be overestimated. Even when other variables are used as covariates, adjustment is likely to be incomplete. At the same time, because no one indicator captures the multiple risks associated with social disadvantage, using estimates from a single association as a proxy for the effects of social disadvantage will underestimate the true effect of what it means to be low on the social hierarchy.

Entwined Risks: Case Example. Data from the National Survey of Children's Health demonstrates the entwining of poverty with other risk factors. This nationally weighted sample of more than 90,000 parents/caretakers of children up to age 18 y (20,000 of whom had children under 5 y of age) was surveyed by phone by the National Center for Health Statistics (http://www.cdc.gov/nchs/slaits/nsch.htm). Parents reported on eight established correlates of poorer child health (low maternal education, low paternal education, poor maternal mental health, poor paternal mental health, nonwhite ethnicity, more frequent moves, resource-poor neighborhood, and low family income). These risk factors were not evenly spread in the population. Just under half of children up to age 5 y had no risks at all, and another third had only one risk, whereas more than one-tenth of children had two risks, 6% had three risks, and another 6% had four or more risks. We divided families into four income categories—below the federal poverty line (FPL), 100–200%, 200–400%, and >400% above the FPL—and found that the distribution of the seven remaining risks was skewed. The likelihood of having multiple risks was substantially higher for children from low-income families and was especially great for children whose families were in poverty (Fig. 1). More than 60% of children in high-income families had no risks at all, and 90% had one risk or less. In contrast, only approximately one-tenth of the children in poverty had only the single risk of low income. Similarly, whereas virtually no children in middle- and high-income groups had more than two risks, more than one-fifth of those in poverty did so. Whereas a small but observable number of children in poverty and in low-income families had more than four risks, virtually no children in middle- and high-income families did so.

Race and Ethnicity. Among risk factors, income and race/ethnicity show the greatest overlap. Given the long history of discrimination in the United States, nonwhites are overrepresented among those in poverty and with low income (68, 69). Surprisingly, most studies of social disadvantage do not account for this confounding. Largely separate literatures address racial and ethnic disparities and socioeconomic disparities. Studies that have data on both variables often use one as the control for the other and rarely consider how their joint functioning affects health or how each is entwined with other threats to good health (70).

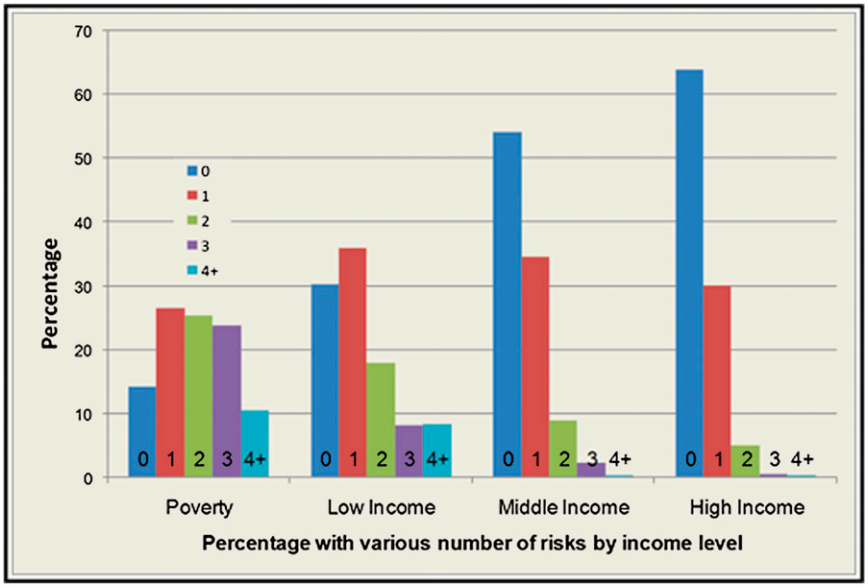

Fig. 1. Distribution of risks by income.

Health. The above discussion addresses the measurement of SES and its complexity. Although beyond the scope of this article, we note that health, too, is a complex phenomenon. Although most studies look at a single indicator or risk factor for a specific disease, social disadvantage increases risk for multiple diseases. Not surprisingly, most measures of disadvantage have stronger associations with indicators of overall health (e.g., mortality, self-reported global health, functional status) than with incidence of specific diseases.

Increasing Complexity

Theories of social disadvantage and how it affects health increasingly point to the need for complex designs and measures. Although early studies of health disparities simply demonstrated that given health outcomes were more common in some groups than others, theories of social determinants of health, psychological development, and biological embedding of disadvantage are fostering increasingly nuanced and complex analytic and measurement approaches (2) and weakening the polarity of rigor vs. vigor. In addition to conducting more longitudinal research, many researchers have moved beyond testing a single main effect to test interactions. For example, studies of biological sensitivity to environments have demonstrated the importance of examining both children's reactivity and the environments to which they are exposed to understand behavioral and physical health outcomes (71, 72). Even more complex relationships are being captured by the applications of systems theory, allowing simulations of complex interactions and feedback loops (73).

Conceptual and empirical advances concerning the impact of stress on the body are enabling more complex measurement of biological processes by which social disadvantage can diminish health. The concept of allostatic load, which encompasses multiple biological systems, has generated new measures of the impact of chronic stress exposure (74). Although the measurement of allostatic load is not settled, the concept has been of great heuristic value and generated numerous studies, including those showing that allostatic load associated with social disadvantage can be detected even among children (51, 75, 76).

Assessing Cumulative Risk. Social disadvantage is not a singular experience. It reaches across time and domains of life. Cumulative risk measures better represent these complexities than do single indicators. The impact of cumulative risk on children's health was first demonstrated in the Isle of Wight study (77), in which Rutter assessed six risks: low parental SES, large family size, discord between parents, parental criminality, maternal mental illness, and foster placement. Although no single risk significantly raised the rates of childhood mental disorder, the incidence increased geometrically as the number of risks increased. Compared with children with no risk factors, those with two risk factors had a fourfold increase in mental disorders, and those with four risk factors had a 10-fold increase. The Rochester Longitudinal Study assessed 10 risks and found that children with eight or more risks had rates of poor academic performance that were seven times greater than for children with zero to three risks (78). Subsequent studies support the view that the number of adversities in children's environment affects their healthy development independent of the occurrence of any individual adverse exposure (79–81).

Although cumulative risk measures capture the multiple effects of social disadvantage, they may not capture the real impact of each risk factor, because some may have a more potent impact than others. If so, weighted scores could be more predictive. More research is also needed regarding the nature of the association between cumulative risk and health. Whereas studies cited above find a geometric increase in health problems at higher levels of cumulative risk, others find a linear association.

Independent Impact of Cumulative Risk. Theories of cumulative risk posit that the experience of multiple risks affects health independent of risk conferred by the various adversities that contribute to the cumulative count. However, this assumption has not, until recently, been directly tested, because most studies test the association of cumulative risk with health outcomes in a separate analysis from tests of association between individual risk factors and the outcomes (e.g., ref. 82). Green et al. (83) tested whether cumulative risk moderated the impact of specific adversities and found that cumulative adversity in childhood added to the prediction of adult psychiatric illness associated with the set of individual adversities, such that the impact of the adversities differed depending on the overall number of adversities experienced in childhood.

Alternative Approaches to Capturing Vigor. Traditional regression approaches have limited tolerance for inclusion of colinear variables, which poses challenges to using them to assess entwined risks. Green et al. (83) dealt with this issue by creating a set of dummy variables for the cumulative risk scores to test the moderation of specific risks by overall cumulative risk. Other techniques, some of which are described below, can also provide information on the patterning of the associations among sets of entwined risk factors.

Structural equation modeling provides a test of both direct and indirect effects. Using structural equation modeling with longitudinal data from one of the British birth cohorts, Chandola et al. (84) demonstrated links between education and health that differed by sex. Using structural equation modeling of longitudinal data, Quesnel-Vallée and Taylor (85) discovered a chain of mediators between parental education in childhood and depressive symptoms in adulthood. They found that parental education influenced one's own educational attainment, which in turn influenced one's income; income in turn affected the likelihood of depressive symptoms in adulthood.

The use of "directed acyclic graphs," known as DAGS, inform such analyses and help make explicit researchers' theories and assumptions. DAGS represent hypothesized causal links among variables in graphical format and clarify the choice of statistical tests. This approach is equivalent in its computational strategy to marginal structural modeling and is particularly helpful for depicting and testing possible mediating pathways (86).

In contrast, recursive partitioning is not theoretically grounded but provides an empirical basis for determining the combination of risk factors that best predict a given outcome. It incorporates multiple predictors and allows for nonlinear relationships and higher-order interactions (87). The variable that best predicts who will or will not experience the outcome of interest is used for an initial division of the sample. This process is then repeated using the same variables within each of the two subpopulations created at this "node" and so on until the best predictive patterns are identified. Using recursive partitioning, Keegan et al. (88) analyzed 49 variables spanning individual traits, specific neighborhood characteristics, and composite measures of positive and negative neighborhood environments to predict women's physical activity and obesity. Obesity rates were lower for women younger than 45 y or older than 56 y than for those in between, and no additional variables predicted obesity risk for the younger and older women. However, among middle-aged women, those living in a lower SES neighborhood were more likely to be obese than those in a higher SES area.

Other techniques such as grade-of-membership analysis and canonical correlation can also help advance modeling of the complex associations among and between socioeconomic variables and other aspects of social disadvantage or between socioeconomic variables and health outcomes. However, their application needs to be informed by well-articulated theories of how health and social disadvantages interact over the life course.

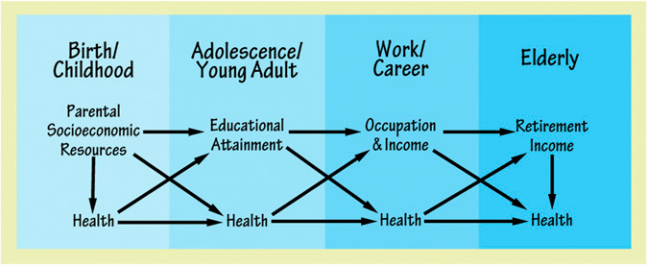

Fig. 2. Dynamic relationship between SES and health. Reproduced from Adler et al. (90).

Theories provide expected patterns of findings, identify the key tests of whether the underlying assumptions are true, and inform the selection of appropriate analytic techniques. Theoretical assumptions about the nature of disadvantage and how it becomes biologically embedded are often implicit in researchers' choices of samples and measures. We need more explicit consideration of the combinations of theory and methods that will bridge the divide between "rigor" and "vigor."

Conclusion

There is little doubt that low SES and its accompanying social disadvantages affect health, and there is also evidence that health can affect SES. Fig. 2 shows a simplified schematic of how SES and health reverberate over the life course. It depicts a dynamic relationship in which the two domains interact over time. Studies generally test one or two links in this overall model, and it is important to situate their findings in the larger context and time frame. This dynamic relationship suggests that policies that work to increase SES will result in better health, but also that policies that work to improve health will increase attainment of higher SES.

This article has examined designs and measures used to study the association of SES with health. These methods are constrained and informed by researchers' theories of causation. The research designs needed to determine causality must include reductionist and mechanistic studies linking precise causes to specific outcomes to clarify the ways in which social disadvantage gets biologically embedded. These may span animal models through human research, and incorporate experimental and quasi-experimental techniques. At the same time, studies that model the complex nature of social disadvantage and the ways in which the accompanying adversities increase vulnerability to a range of health problems are necessary to capture the real-world experience and operation of social disadvantage; such studies will likely involve data on cumulative adversity and summative measures of health, and the use of new analytic techniques.

Other areas of science are also struggling with the need to incorporate vigor along with rigor. For example, there is growing concern about the disconnect between positive findings from rigorous efficacy trials of drugs and interventions and their poor effectiveness in actual practice. Glasgow et al. (89) note that the former use homogeneous samples, specialized interventions, and randomized designs, whereas the latter engage a wider range of participants studied over a variety of conditions using different analytic methods. In brief, efficacy trials stress internal validity and maximize rigor, whereas effectiveness studies stress external validity and incorporate more vigor in the assessment. In calling for greater attention to effectiveness, Glasgow et al. (89) argue that "we need to embrace and study the complexity of the world rather than attempt to ignore or reduce it. . .."

In studying health disparities, the precision afforded by rigorous mechanistic research increases our understanding of how social factors impinge on human biology, and can inform policies to reduce disparities by identifying the "active ingredients" in social disadvantage. The insights into the realities of the lives of people at different socioeconomic levels afforded by vigorous research can engage policy makers in understanding the true costs of social disadvantage and motivate action. Ideally, we will develop sufficiently high-level theories that encompass both approaches to assessing social disparities in health. Eliminating these disparities will require both rigor and vigor on the part of theoreticians and researchers to maximize each of these important aspects of science.

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