

U.S. Disparities in Health: Descriptions, Causes, and Mechanisms

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Abstract

Eliminating health disparities is a fundamental, though not always explicit, goal of public health research and practice. There is a burgeoning literature in this area, but a number of unresolved issues remain. These include the definition of what constitutes a disparity, the relationship of different bases of disadvantage, the ability to attribute cause from association, and the establishment of the mechanisms by which social disadvantage affects biological processes that get into the body, resulting in disease. We examine current definitions and empirical research on health disparities, particularly disparities associated with race/ethnicity and socioeconomic status, and discuss data structures and analytic strategies that allow causal inference about the health impacts of these and associated factors. We show that although health is consistently worse for individuals with few resources and for blacks as compared with whites, the extent of health disparities varies by outcome, time, and geographic location within the United States. Empirical work also demonstrates the importance of a joint consideration of race/ethnicity and social class. Finally, we discuss potential pathways, including exposure to chronic stress and resulting psychosocial and physiological responses to stress, that serve as mechanisms by which social disadvantage results in health disparities.

Social class: social groups that arise from interdependent economic relationships among people on the basis of their structural location within the economy (107)

INTRODUCTION

Few terms have had such a meteoric rise into common usage in the health literature as has “health disparities.” In the 1980s this was a key word in only one article, and in the 1990s there were fewer than 30 such articles. In contrast, during the five years from 2000 through 2004, more than 400 such articles appeared (3). An equivalent increase occurred in the number of articles containing the key term of “health inequalities.” Prior to this time, there was substantial work on the problem of health disparities, but it was usually framed in terms of specific factors such as race or poverty (60).

One of the first uses of the term inequality with respect to health differences was in the title of the Working Group on Inequalities in Health, which issued the Black Report in Great Britain in 1980. In advance, it seemed likely that the working group would find reductions in social class differences in mortality following the provision of universal health care through the National Health Service. However, they found that the gap between the health of low and high social class individuals had actually widened. Around the same time, the Whitehall Study of British Civil Servants (68) revealed significant differences in cardiovascular disease and mortality (69) by occupational level within a population of office-based workers. Notably, differences were not just between those at the top and bottom. Rather, disease prevalence and mortality increased at each step down in occupational grade. Spurred by these and other data, another commission, the Independent Inquiry into Inequalities in Health, made recommendations for policies in Great Britain to reduce health inequalities (2).

During this period, research on socioeconomic and racial/ethnic differences in health was also being conducted in the United States. Beginning in the 1970s, investigators linked death records to socioeconomic data from the Current Population Study, to the U.S. Census, and to Social Security Administration records. The findings documented at a nationwide level substantially higher age-

adjusted mortality rates for nonwhites, individuals with less education, individuals with low incomes, and for some occupational categories (16, 58, 59). These data and the British findings provided an impetus to determine the extent and nature of health disparities in the United States and identify ways to reduce them. Efforts have included a report from the National Center for Health Statistics on differences in mortality and morbidity by socioeconomic status (80), *Healthy People 2010* (100), which established the goal of eliminating health disparities in addition to the goal of improving health, and the passage of the Minority Health and Health Disparities Research and Education Act of 2000. This legislation established the National Center on Minority Health and Health Disparities to coordinate activities among the NIH institutes. The Institute of Medicine recently reviewed the NIH plan and made a number of recommendations to improve its effectiveness (99).

As reflected in the dual goals of *Healthy People 2010*, public health research and practice aim both to improve health and to eliminate disparities. Previous papers in the *Annual Review of Public Health* have examined substantive and methodological aspects of specific types of disparities. Some reviews concerned measurement issues and health effects of poverty, class, and/or socioeconomic status (e.g., 1, 36, 61), of race and ethnicity (e.g., 64, 70, 105), and of rural residence (88). None has considered disparities per se. Eliminating disparities requires a clear definition to allow measurement and monitoring of progress toward that goal and to understand their causes. Here we examine the definition of health disparities and empirical findings on disparities associated with race/ethnicity and socioeconomic status. We then consider methodological challenges and solutions to understanding the causes of health disparities.

DEFINITION OF HEALTH DISPARITIES

The literature lacks a consensually agreed on definition of health disparities. *Healthy*

People 2010 referenced “differences that occur by gender, race or ethnicity, education or income, disability, geographic location, or sexual orientation” (100, p. 14). Carter-Pokras & Baquet (17) identified 11 different definitions of health disparities. Some were inclusive, some limited disparities to those associated with race and ethnicity, and still others defined it only in terms of disparities in health care.

The various definitions imply and sometimes explicitly suggest the relevant comparison group for establishing a disparity. Definitions of racial/ethnic disparities suggest that a group’s health status be compared with the majority, the population average, or the healthiest group. Thus, one might compare African American mortality rates to national rates, to European Americans who are the majority group in the United States, or to Asian Americans, who have in aggregate the lowest mortality rates. Depending on the relative size and the relative health of the majority group and the healthiest group, one could reach different conclusions about the extent of a disparity.

With the exception of Murray and colleagues (78), who examined a range of sociodemographic characteristics of groups with markedly different life expectancies, most approaches to disparities start with bases of social disadvantage, which result in differences that are unjust and avoidable (13, 15). *Healthy People 2010* distinguishes between a health difference, which results from inherent biological differences (e.g., only women are subject to ovarian cancer and men to prostate cancer), and a disparity, which results from social factors. What constitutes a difference versus a disparity may sometimes be unclear, however. In the example of ovarian and prostate cancer, differential investment in research on treatment and prevention of one disease versus the other could reflect the relative advantage of males versus females. If men have more power to allocate resources for research and health care and differentially provide funding for prostate versus ovarian cancer, the resulting death rates from these diseases could con-

stitute a disparity. This suggests that simple comparisons of mortality rates are not an adequate basis on which to evaluate health disparities. One also needs to know the biological potential of each group. Although women outlive men (a fact pointed to by some who advocate for more attention to men’s health as a disparity issue), the gap between current life expectancy and life expectancy under optimal conditions could potentially be greater for women than for men.

Differences in biological potential have been raised in relation to racial/ethnic health disparities, suggesting these are differences rather than disparities. However, the contribution of unavoidable biological differences to overall disparities by race/ethnicity is relatively small. A few diseases (e.g., sickle cell anemia) have a clear primary genetic basis, but these are of a limited number and there is little evidence for a differential genetic basis for the many diseases for which disparities occur (75). For example, African Americans have higher rates of hypertension than do European Americans, which some attribute to differential genetic vulnerability. However, prevalence of hypertension among blacks is lower in Caribbean countries than in the United States and lower still among blacks in Africa. Hypertension rates in Africa are, in fact, equivalent to or lower than rates among whites in the United States (26). These findings suggest that higher rates of hypertension for blacks in the United States compared with other racial/ethnic groups are more likely to be due to social factors than to underlying biological vulnerability.

Health disparities result from both biological differences and social disparities. We focus on the latter not just because the effect is greater, but also because they are avoidable and inherently unjust.

EMPIRICAL WORK ON DISPARITIES

The bulk of research has focused either on disparities due to race/ethnicity or disparities due

FOREIGN-BORN POPULATIONS

Place of birth is a critical and frequently ignored component of socioeconomic and racial/ethnic disparities. To the extent that first-generation immigrants make up a substantial proportion of a given group's population in the United States, immigrants' health advantage may contribute to differences between groups. For most health outcomes (notable exceptions are stomach cancer and liver disease), foreign-born individuals in the United States have lower rates of disease than do their native-born peers. Controlling for demographic and socioeconomic factors, immigrant men and women 25 years of age and older had mortality rates 18% and 13% lower, respectively, than did nonimmigrants (95). Immigrants as a group lived 3.4 years longer on average than did those born in the United States in 1999–2001, an increase over a gap of 2.3 years two decades earlier (94). The gap was largest for native-born vs. immigrant blacks and Hispanics.

Most analyses of health disparities do not include birth place and do not account for the generally lower rates of disease among foreign-born individuals (79). U.S. Hispanics as a group have lower all-cause mortality rates than do non-Hispanic blacks or non-Hispanic whites; a difference that becomes even greater after controlling for household income. The relatively lower rates of all-cause mortality among Hispanics as compared with non-Hispanics in the United States have been well documented, and a large literature investigating the substantive and potentially artifactual reasons for this has emerged (although no clear consensus has been reached yet) (79). Asian Americans, too, show favorable health profiles, with the lowest prevalence of a number of diseases and the lowest all-cause mortality rate of any major racial/ethnic group, and the role of migration processes in these disparities is also an area of active research.

Socioeconomic status (SES): an individual or group's position within a hierarchical social structure, measured by variables including education, occupation, income, wealth and place of residence

to social class and socioeconomic resources. Disparities by gender and geography have also been investigated, often in terms of how these factors modify racial/ethnic or social class disparities. Most research has not invoked an explicit model of disparities and studies are shaped and constrained by the availability of relevant data. For example, British studies emphasize social class as determined by occupational status using the Registrar General's measure of social class. This measure

has been in use for many years and provides a fine-grained hierarchical ordering of occupations. Nothing comparable exists in the United States, where national data are more likely to include race/ethnicity than measures of socioeconomic position. For example, it was not until 1989 that education was added to the U.S. standard certificate of death, and health records of large population groups such as those enrolled in Kaiser Permanente often include only race/ethnicity but not socioeconomic status (SES). Thus, it has been easier to characterize racial/ethnic disparities in the United States than those linked to social class. The data show that African Americans have higher mortality and poorer health status than does any other group, as do Native Americans. Overall mortality rates are surprisingly higher for non-Hispanic whites than for Hispanics or Asian Americans; relative mortality varies for specific causes of death (3, 94). For 1999–2001, male life expectancy for U.S.-born blacks and whites was 67.5 and 74.8 years, respectively. Life expectancy for U.S.-born Hispanic males (75.2 years) was greater than for non-Hispanic whites and was greater still for U.S.-born Asian/Pacific Islanders (78.9 years). The same pattern is shown for women. For both men and women, the health advantage of Hispanic and Asians compared to U.S.-born whites is even greater for recent immigrants in these groups (see sidebar on Foreign-Born Populations).

Intersection of SES and Race/Ethnicity

Some definitions limit disparities to those associated with race/ethnicity. This focus has been fostered both by relative availability of data as described above and by social equity concerns based on current and historical racism and discrimination. Such a limitation can be problematic, however, given marked differences in the distribution of racial/ethnic groups across levels of education, income, occupation, and wealth (29, 56). Examining race/ethnicity without simultaneously

considering socioeconomic position can attribute too much influence to race/ethnicity per se, and may inadvertently foster an emphasis on biological differences. This point is forcefully made by Isaacs & Schroeder (52) who argue that social class is the “ignored determinant” of health in the United States

Researchers are increasingly looking at how SES and race/ethnicity function jointly and independently to affect health. Socioeconomic measures often account for a large part of racial/ethnic differences, although independent effects of race/ethnicity on health outcomes also exist, depending on what outcome is examined. Adequate control for SES across racial/ethnic groups may be difficult to achieve (54). SES indicators may have different meanings for different groups. For example, at the same income level, the amount of wealth and debt differ substantially by racial/ethnic group; Hispanics and African Americans have lower wealth than non-Hispanic whites and Asians at a given income level (14, 24). Similarly, at any given educational level, these groups have lower incomes than do whites (14). Although some studies “control” for SES by adjusting for an indicator such as education or income, this adjustment is insufficient given evidence for independent effects of the different domains of SES. Controlling for a single measure is unlikely to capture the effects of social class per se, and residual confounding may be erroneously interpreted as racial/ethnic differences (14, 54).

Descriptive Findings

A descriptive understanding of socioeconomic and racial/ethnic disparities is important for (a) understanding both long- and short-term trends in health disparities, (b) informing causal investigations of health disparities, (c) targeting resources for prevention and treatments to reduce disparities in specific diseases, and (d) increasing public awareness of the existence and characteristics of health disparities. Below we briefly consider descriptive data

regarding mortality disparities, cause-specific disparities, geographic variation in disparities, and time trends in these disparities.

All-cause mortality. The first U.S. study with a sample size sufficient to allow the examination of socioeconomic disparities within race/ethnicity based on individual-level data was done by Kitigawa & Hauser (58), although data constraints limited comparisons to whites and nonwhites. Using data from the 1960 matched records of persons age 25 and over, they documented that compared with whites, age-adjusted all-cause mortality rates for nonwhites were 34% higher for females and 20% higher for males, correcting for net census undercount. They also examined mortality by education, occupation, income, and geographical location. For white men and women ages 25–64 mortality was respectively 64% and 105% higher for the least compared with the most educated. For nonwhite men and women the comparable difference in mortality by education was 31% and 70%, respectively. Pappas et al. (81) revisited this work, with data from 1986, showing a relatively sharper decrease in mortality over this time period for higher-income and more-educated individuals, thus creating greater relative disparities by income and education overall and within racial/ethnic groups over time. This and other work also highlights the importance of disparities based on social class for both women and men, despite some earlier work that suggested smaller social class disparities among women (72).

In addition to dichotomizing race into white and nonwhite, earlier U.S. research generally dichotomized income into below versus above the poverty line. Publication of the Whitehall study inspired researchers to see if SES formed a graded association with health in the United States, as it did in England. Multiple studies have now demonstrated SES gradients by income and by education for a range of health outcomes including mortality, incidence of cardiovascular disease, arthritis, diabetes, asthma, cervical cancer,

NLMS: National Longitudinal Mortality Study

SMR: standardized mortality ratio

COPD: chronic obstructive pulmonary disease

depression, and disability in children, adolescents, and both younger and older adults (4, 22, 43, 76). Although these associations occur across the distribution, they are generally stronger at the lowest levels of income and education (8, 33, 91).

Cause-specific mortality. Studies uniformly find higher all-cause mortality for blacks than for whites under age 65, but within this overall trend there is heterogeneity by cause of death. For example, data from the National Longitudinal Mortality Study (NLMS) of 1.3 million persons (89) reveal a racial/ethnic difference for mortality from many but not all diseases. Black and white men under age 65 had approximately the same standardized mortality ratio (SMR) for ischemic heart disease, whereas (in order of magnitude of difference) black men had substantially higher SMRs than did whites for homicide, hypertensive heart disease, esophageal cancer, and pulmonary circulation but had relatively lower SMRs for aortic aneurysm, suicide, leukemia, and chronic obstructive pulmonary disease (COPD). Black women had substantially higher rates of homicide, hypertensive heart disease, diseases of pulmonary circulation, nephritis, and stomach cancer than did white women, with comparatively lower levels of suicide, COPD, and leukemia.

Howard et al. (51) also used data from the NLMS and found that SES accounted for different amounts of black-white mortality differences depending on the cause of death. For men, SES accounted for 30%–55% of the black-white mortality differences for accidents, lung cancer, stomach cancer, stroke, and homicide, but less than 17% of the differences for prostate cancer, pulmonary disease, and hypertension. For women, SES accounted for 37%–67% of differences for accidents, ischemic heart disease, diabetes, and homicide, but less than 17% for hypertension, infections, and stomach cancers. However, only income and education were used as SES controls, which could underestimate the

contribution of SES to black-white mortality differences. Kington & Smith (57) found that with more complete demographic controls including wealth, racial/ethnic differences in functional limitation in health of older individuals were eliminated, although differences remain for other chronic diseases.

Wong et al. (106) also studied the contribution of education and race/ethnicity to different causes of death. Whereas many causes of death contributed in a similar way to both racial/ethnic and educational disparities in mortality (e.g., cardiovascular disease, liver disease), other causes were responsible for greater educational differentials (e.g., cancer, lung disease) or greater black-white differences (e.g., hypertension, lung disease, homicide). The data from these studies show that although the direction of disparities is fairly consistent, the extent of socioeconomic and racial/ethnic disparities and their interactions differ substantially by cause.

Geographic variation. Although marked differences in mortality rates across the United States have been noted, the extent to which socioeconomic factors and race/ethnicity explain these variations had not been adequately studied. However, data from within metropolitan areas reveal a geographic variation that can be substantially explained by considering these factors. These data also suggest that differences in local socioeconomic conditions have a greater impact on African American mortality than white mortality, resulting in an interaction between socioeconomic factors and race/ethnicity with respect to geography (23, 98). This is consistent with data from the NLMS (89) showing that the locations with the lowest mortality rates for whites and for blacks were at an equivalent level, even as overall rates were higher for blacks. These studies of geographic differences show the importance of area context for disparities and note that relationships among race/ethnicity, class, and health are not fixed, even within the United States during a given time period.

Changes in disparities over time. The magnitude of disparities in mortality by race/ethnicity and by SES have changed over time, providing further evidence that these disparities are changeable and preventable. Preston & Ilo (86) confirmed Pappas's finding of increasing education gradients for all-cause mortality for men since 1960 but also found that education differentials in mortality declined for women 25–64 and remained stationary for women 65–74. Ward et al. (104) examined disparities in cancer mortality by race/ethnicity 1975–2000. Prior to 1980 investigators saw no black/white disparities in breast cancer mortality among women and saw slightly higher rates of colorectal cancer mortality among white as compared with black men. But this changed, and by 2000, black women had higher breast cancer mortality than did white women and black men had higher colorectal cancer mortality than did white men. The black-white gap in overall life expectancy decreased from 1975 to 1984, increased from 1984 to 1992–1994, then decreased again through 2004 (48). Most of these changes stemmed from relative improvements for blacks in specific causes of death (e.g., relatively greater decreases from 1994 to 2004 in homicide and unintentional injuries for both sexes and for HIV for men and heart disease for women).

Disparities in risk factors for disease have also changed over time. For example, Zhang & Wang (108) examined obesity rates among U.S. women 20–60 years old from 1971 to 2000 using data from the National Health and Nutrition Examination Survey (NHANES). Owing to rapid increases in obesity prevalence among all educational groups, education disparities actually decreased, although all groups were worse off. These results highlight the importance of overall population trends for assessing progress in reducing health disparities.

Changes in disparities over the life course.

The extent and nature of health disparities changes over the life course. Substantial dis-

parities begin at birth; babies born to mothers who are poor, have lower education, and/or are African American are smaller at birth and are more likely to die within the first year of life. Disparities are smallest during childhood, adolescence, and early adulthood and greatest in middle age, becoming weaker again in older populations (5). The primary explanation for diminished disparities in older populations is that the least healthy individuals are no longer in the population, and mortality will eventually be experienced by all regardless of socioeconomic status and race/ethnicity. Although selection over time can produce artifactual population patterns (102), the proportion of the narrowing of disparities explained by selection is unclear. There may also be etiologic reasons, including the provision of safety net supports such as Social Security and Medicare, which are available to older adults and may reduce and/or buffer the effects of disadvantage.

Variation by measure of SES. Occupation, income, and education have different associations with health outcomes (58, 89). As currently operationalized, education and income are generally more strongly associated with health in U.S. data than are measures of occupation other than employed versus nonemployed. However, weaker associations with occupation may be due to the use of standard U.S. occupational measures (14). Using a classification based on the new U.K. national statistics social class measure—which categorizes individuals as managers/professionals, intermediate, small employers and self-employed, lower supervisory and technology, and semiroutine/routine or not in labor force—Barbeau et al. (9) found occupational associations with current smoking status as strong as those with education or income. Variations by SES measure used speak to the frequent recommendation of using discrete measures of SES such as education or income rather than a composite (32). In addition to empirical reasons, use of specific SES measures clarifies intervention possibilities.

UNDERSTANDING THE NATURE AND CAUSES OF DISPARITIES

General patterns of disparities over the late twentieth century in the United States are similar: Those with fewer resources have worse health outcomes for a number of different causes. But variations by health outcome, place, time, and age point to the fact that these associations are not fixed or immutable, and that this heterogeneity should be used to better understand the causes of disparities. Kunitz (63) places links between distribution of resources and health within particular historical, socioeconomic, and cultural contexts. Given these variations, a deeper understanding of off-diagonals may be informative about the nature of disparities. This analysis would include diseases that do not show disparities or are more prevalent in more advantaged groups (e.g., black-white differences in kidney function and socioeconomic differences in breast cancer). It would also include those who do not show expected patterns such as immigrants (see sidebar), low-SES individuals in good health, and high-SES individuals in poor health. Finally, international comparisons of socioeconomic disparities highlight the importance of national contexts for understanding the nature of health disparities.

Establishing Causality

There are clearly documented associations of SES and health outcomes, but the causal link is still debated. Some questions are methodological, dealing with alternative explanations for the associations. Others are concerned with the nature of the mechanisms by which these upstream factors influence health. SES is unlikely to affect health directly (e.g., having more dollars in one's pocket is not health protective). Rather, it shapes life conditions that, in turn, influence health. In this section we first consider the methodological challenges to understanding causes of health disparities and then consider potential mecha-

nisms by which SES may affect morbidity and mortality.

Methodological challenges—alternative explanations. When asserting that a measure of SES leads to sub-optimal health and premature mortality, researchers must address possible alternative explanations for the associations that are found (42, 45). The first possibility is that associations result from random chance; this possibility can be assessed by specifying confidence intervals around the effect estimate or p-values. Second, associations may be due to conditioning on an effect of the exposure and outcome occurring either through the selection of the sample (i.e., selection bias) or through use of inappropriate control variables (42, 50). Avoiding this possibility requires using a causal understanding of the process that created the data to inform sample selection and an appropriate choice of control covariates.

A third challenge is that the presumed health outcome may cause the exposure (reverse causation or health selection bias) (58, 96). For example, illness may prompt individuals to decrease work hours, change to less demanding and lucrative jobs, or leave the labor force entirely. Using data from the Health and Retirement Study of individuals over the age of 50, Smith (96) found that wealth decreased by \$17,000, and earnings by \$2600 per year with the onset of major disease. Collecting measures of income that predate the health assessment through longitudinal designs, data linkage or retrospective earnings recall can decrease reverse causation potential between income and health. Using a lagged approach with longitudinal data, McDonough et al. (71) found little difference in predicting all-cause mortality between a one-year lag and a five-year lag, thus questioning the importance of reverse causation for explaining the mortality associations. Using another approach to account for health selection, Benzeval & Judge (10) controlled for initial health status in addition to using measures of income prior to

disease onset, and the associations between income and health outcomes remained.

There is less reason for concern about reverse causation between education and health. Generally the temporal lag between education exposure and adult health outcomes argues against adult health impacting education (58). However, childhood illnesses and low birthweight may contribute to lower educational attainment (18, 25). These factors are themselves a function of SES. Haas (46) demonstrated that disadvantaged social background led to sub-optimal health in childhood, which made a subsequent impact on adult social class.

Overall, although health can affect SES, SES significantly affects health. The extent of reciprocal influence for specific outcomes is generally not understood. Longitudinal data with health, education, income, labor force participation, and wealth measures over time can more accurately model the process of social stratification and the extent to which causation and selection impact specific health outcomes at different points in the life course.

A fourth concern is whether associations result from the joint association of SES and health with a common underlying cause such as genetic factors, time preferences/delayed gratification (39), or cognitive ability (44). As with reverse causation, these confounders may themselves reflect SES. Early family environments affected by parents' education and income may shape all three of these potential confounders, including the extent to which genetic potential is realized through epigenetic processes. As evidence of the importance of SES and child environments for adult health increases, rather than viewing these factors as undermining evidence for the importance of socioeconomic factors on health, they should be viewed as part of the dynamic process between SES and health over the life course.

Data structure and methods. In addition to collecting appropriate data to control for potential alternative explanations in regression

models, several types of data structures can also facilitate better determination of causal relationships and help rule out alternative explanations for observed correlations. True experiments are rare because individuals cannot easily be randomly assigned to levels of education, income, or occupation. However, experimental trials of interventions that modify some aspect of SES or factors associated with it are informative. Researchers have also taken advantage of natural experiments to assess the effects of economic or policy changes that affect an individual's SES but are not due to his or her own characteristics or behaviors. These reduce confounding and allow for a more easily conceptualized counterfactual (45). Relevant examples include using German reunification to estimate the effects of income on health (38), changes in the Earned Income Tax Credit to estimate the effects of household income on children's test scores (28), enactment of schooling laws to estimate the effects of education on mortality (66), and changes in legislation affecting Social Security benefits to estimate the effects of income on mortality in an older population (97). With the exception of the Social Security payments, these studies confirm the effects from observational studies of socioeconomic factors to health.

Data with repeated measures on individuals over time also provide some strength for making causal claims (87). Repeated measures allow observation of the temporal sequence of cause and effect. Birth cohorts provide particularly rich data for modeling early life confounders and exposures of interest. Three British studies of representative samples of children born in 1946, 1958, and 1970 have provided critical data about the causes of health disparities and have shown the impact on adult health and behaviors of early life exposures and socioeconomic position at different points in life (84, 103). Using data from the 1958 cohort, Power et al. (85) found a number of causes of health inequalities at age 33, including class at birth, socioemotional adjustment, educational level, and psychosocial job strain. In the absence of a birth cohort,

Structural equation

models: techniques to test associations between variables, typically relations between variables in the model based on prior subject knowledge

Directed acyclic graph (DAG):

graphical representation of the single direction causal relationships between variables

follow-up of members of completed studies of children and adolescents can provide some of the same advantages (41).

Analytic approaches. In addition to the design approaches described above, new analytic methods are facilitating a better understanding of the causes of health disparities. Five methods that may be particularly useful are propensity score matching, instrumental variables, time-series analysis, causal structural equation modeling, and marginal structural models.

Propensity scores provide an analytical method for balancing factors associated with being in either of the analytical comparison groups of interest in a particular study (e.g., high versus low education). If assumptions are met it allows for unbiased causal estimates of the exposure under study (27, 90). They have been used to identify the effects of gun violence exposure on subsequent violent activity (11), neighborhood characteristics on dropping out of high school (47), and neighborhood socioeconomic environment on cardiovascular mortality (30). This approach is based on the same principle as adjusting for confounders in a regression model and similarly requires all confounders be measured. However, they facilitate assessment of whether overlap of confounding variables actually allows one to compare the analytic groups of interest appropriately, and they also provide power to control for a larger number of confounding covariates.

Instrumental variables (IV) offer advantages when analyzing data from natural experiments or similar designs. The crucial assumption is the availability of a variable (the instrument) that does not directly affect the outcome but is only associated with the predictor of interest, and where the exposure (instrument) is not itself influenced by known confounders (7). This approach has been used to show causal effects of income on health outcomes (34) and to demonstrate the effect of years of schooling on all-cause mortality (66).

Time-series analyses are particularly helpful for evaluating policy changes or other population exposures by analyzing the variation in health outcomes over time, while allowing investigators to identify and remove temporal autocorrelation and also account for lag effects between exposure and outcome. Particularly useful are data from multiple locations with different temporal ordering of the exposure to remove more general temporal trends. This approach has been used to demonstrate the effects of unemployment on alcohol abuse (19) and on very low birthweight (20) and to examine trends in black-white disparities over time (65).

Structural equation models have been used extensively in the social sciences to understand complex relations between variables and to test relationships among hypothesized causes, mediators, and outcomes. Despite controversy, work over the past two decades by Pearl and others (82, 83) has clarified the conditions under which the models may be used to represent cause. A significant innovation for gaining this understanding is the use of directed acyclic graphs (DAGs), a graphical language for describing causal relations. These form a framework for representing assumptions about elements of the causal pathways from social exposures to outcomes and information about possible confounders. Explicit delineation of the proposed causal structures through DAGs allows other researchers to evaluate the assumptions made and to build on the proposed structures. These models facilitate identification of valid empirical tests of proposed causal models (31). This is helpful in testing proposed mediators between social class and health (55). A causal structural modeling approach using DAGs is also mathematically equivalent to marginal structural models (82), which allow (when assumptions are met) a determination of the overall causal effect of an exposure within a framework based on treating unobserved counterfactuals as missing data (101).

Chandola et al. (21) used this approach with data from the 1958 British Birth Cohort

to examine the relative contributions of six different pathways connecting education and health. The structural model included factors at age 7 (cognitive ability, father's social class), age 16 (adolescent health), age 23 (education), age 33 (adult social class, sense of control, healthy behaviors), and age 42 (adult health). It showed no direct effect of education on adult health but showed significant effects through adult social class, control, and behaviors, with differences by gender in the strength of pathways (21). A similar approach was taken by Mulatu & Schooler (77) in examining the relative strength of behavioral and psychosocial pathways between SES and health.

Pathways and Mechanisms

Much recent research has attempted to explicate the pathways and the mechanisms by which SES influences health. Although few studies have explicitly tested these through structural equation models, the studies provide many candidates. Physical and social environments, including a person's home, school, work, neighborhood, and community, vary by SES and affect the likelihood of individuals' exposure to both health-damaging conditions and health-protecting resources. Health-damaging exposures within these pathways include early life conditions, inadequate nutrition, poor housing, exposure to lead and other toxins, inadequate health care, unsafe working conditions, uncontrollable stressors, social exclusion, and discrimination (5, 6, 105).

Some of the exposures listed above have direct effects on health, whereas others may influence psychological dispositions and behaviors that have health consequences. A vast literature demonstrates the contribution of psychosocial and behavioral factors to morbidity and mortality. These factors include cognition and emotion (e.g., depression, hopelessness, hostility, and lack of control) and behavior (e.g., use of cigarettes, alcohol, and other substances). Gallo & Matthews (40) observed that substantial evidence links neg-

ative emotions with many health outcomes and links SES with negative emotions, but few studies have analyzed these pathways together. For example, hostility and hopelessness are strongly predicted by childhood socioeconomic position (49) and are linked, in turn, to poorer health (12, 37, 40). However, the extent to which the links between childhood SES and adult health are accounted for by hostility and hopelessness has not been determined.

The few studies that have considered mediation by psychosocial factors provide supportive findings, but these have used regression rather than structural equation models. For example, Marmot et al. (67) examined the role of sense of control over one's work in explaining health disparities within the Whitehall sample. The higher the grade of the civil servants, the more control they experienced in relation to their work conditions. Consistent with hypothesized mediation, the association of occupational grade with health was substantially reduced when adjusted for sense of control.

A common element in many of the proposed mechanisms linking SES to health is differential exposure to stress. Disadvantaged environments expose individuals to greater uncertainty, conflict, and threats for which there are often inadequate resources to respond effectively. These experiences cumulate to create chronic stress. Until recently, stress research focused primarily on acute stress, which is more easily modeled in the lab, and was based on a model of homeostasis. The development of the model of "allostatic load" (AL) (73) provided a major conceptual advancement to understand health disparities. This model posits that the body does not simply reestablish homeostasis after experiencing a perturbation associated with a stressor. Rather, with repeated exposures, set points for various systems involved in the stress response, including the endocrine, metabolic, cardiovascular, and immune systems, may shift. Although the body may be in balance, the systems become burdened and

Stress: psychological and physiological response to threat under circumstances where individuals do not have adequate capabilities to deal with it effectively

AL: allostatic load

dysregulated by the costs of the repeated adaptation cycles (74). Precise ways to assess AL are still being developed, but early findings suggest that it is a useful approach. Seeman et al. (92, 93) assessed AL in terms of 10 dysregulation indicators in a sample of older adults who had no major diseases at baseline. AL scores were higher in those with less education and predicted subsequent decline in physical and cognitive functioning, new cardiovascular disease, and seven-year mortality. Using data from the Normative Aging Study, Kubzansky et al. (62) also found higher AL among those with less education and further found evidence that the effect was partially mediated by hostility.

Although the effects of chronic stress cumulate over time, the biological manifestations may be seen relatively early in life. Evans (35) found that children from disadvantaged environments had higher AL than did children from more affluent backgrounds, and one indicator of AL was found in structural equation models to mediate the impact of poorer housing conditions on illness-related school absences (53).

These examples are a few of thousands of studies on a variety of potential mechanisms and pathways. Most of these have not been linked specifically with health disparities but provide detailed information on dif-

ferent levels of cause that could result in disparities. Data sets with adequate measures of socioeconomic factors and race/ethnicity, potential psychosocial and biological mechanisms, and health outcomes are necessary to best understand pathways. These then can be analyzed using techniques such as causal structural models that allow modeling and testing of multiple direct and indirect pathways to health outcomes that are the bases of disparities.

CONCLUSION

Substantial health disparities exist in the United States by social class and race/ethnicity. It would, of course, be preferable to eliminate disparities by addressing the root causes, changing the inequitable resource distribution that now accompanies SES and race/ethnicity as well as other bases of disparity. For effective policy development and interventions, we need persuasive data on the causes of disparities. This entails moving beyond associations to establish causal relationships. In addition, understanding the pathways and mechanisms that mediate these effects provides more information about the multiple causes of health disparities and offers possible interventions to alleviate their occurrence.

SUMMARY POINTS

1. In the United States, health disparities associated with race/ethnicity and socioeconomic status (SES) are widespread.
2. Variation in disparities by cause of death, geographic region, and time suggest that disparities are modifiable and avoidable.
3. Differences in distribution across levels of SES for blacks and whites may account for many racial/ethnic health disparities; socioeconomic causes of racial/ethnic disparities cannot be ruled out without comprehensive measures of SES.
4. A variety of strategies can be used to provide stronger evidence of causal influences of SES on health, including use of data structures, such as natural experiments, and analytic methods, such as structural equation modeling.

5. Identifying pathways and mechanisms by which SES and race/ethnicity affect health provides better evidence of causation and more options for intervention to eliminate disparities.
6. Evidence shows multiple pathways from SES and race/ethnicity to health; one pathway is through differential exposure to chronic stress and its resulting biological toll.

DISCLOSURE STATEMENT

The authors are not aware of any biases that might be perceived as affecting the objectivity of this review.

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