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Topic 1. Structural Patterns of Health Inequalities

# Controlling Disease and Creating Disparities: A Fundamental Cause Perspective

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The United States and other developed countries experienced enormous improvements in population health during the 20th century. In the context of this dramatic positive change, health disparities by race and socioeconomic status emerged for several potent killers. Any explanation for current health disparities must take these changing patterns into account. Any explanation that ignores large improvements in population health and fails to account for the emergence of disparities for specific diseases is an inadequate explanation of current disparities. We argue that genetic explanations and some prominent social causation explanations are incompatible with these facts. We propose that the theory of "fundamental causes" can account for both vast improvements in population health and the creation of large socioeconomic and racial disparities in mortality for specific causes of death over time. Specifically, we argue that it is our enormously expanded capacity to control disease and death in combination with existing social and economic inequalities that create health disparities by race and socioeconomic status: When we develop the ability to control disease and death, the benefits of this new-found ability are distributed according to resources of knowledge, money, power, prestige, and beneficial social connections. We present data on changing mortality patterns by race and socioeconomic status for two types of diseases: those for which our capacity to prevent death has increased significantly and those for which we remain largely unable to prevent death. Time trends in mortality patterns are consistent with the fundamental cause explanation.

IN the last century, human beings greatly expanded their capacity to control disease and death. Any explanation of current health disparities by factors such as socioeconomic status (SES) or race must take account of this fact. We will argue that the capacity to control disease and death creates disparities—that when we make gains in our ability to control disease, people with more knowledge, money, power, prestige, and beneficial social connections are better able to harness the benefits of the control we have developed. If this line of thinking is correct, it has important implications for explaining disparities and for the kind of research and action that are likely to help us reduce those disparities. In this article, we explore these ideas and provide evidence concerning their validity. We begin by calling attention to the nature of the disparities we confront.

## Socioeconomic and Racial Disparities in Health and Mortality

There is a strong, well-established, and very robust association linking both morbidity and mortality to educational attainment, occupational standing, and income (Antonovsky, 1967; Kunst, Feikje, Mackenbach, & EU Working Group, 1998; Sorlie, Backlund, & Keller, 1995). Figure 1 provides a recent example of this association: It shows adjusted death rates per 100,000 by educational status, for people between the ages of 25 and 64 years in the United States in 2001. For both men and women, adjusted mortality rates are much higher for those with less than 12 years of education compared with those with 13 or more years. Similar patterns exist for Black–White mortality differences. Figure 2 presents data from 1998 showing all-cause age-adjusted mortality rates for Blacks to be nearly twice the rates for Whites; again, this is true for both women and men.

These strong mortality gradients based on SES and race are not new. For SES, the association was observed in Mulhouse, France, in the early 19th century, in Rhode Island in 1865, in Chicago in the 1930s (see Antonovsky, 1967), and in the United States and Europe today (Kunst et al., 1998; Lantz, House, Lepkowski, Williams, Mero, & Chen, 1998; Sorlie et al., 1995). Similarly for racial differences, life expectancy has been much lower for African Americans than for White Americans as far back as such data have been available. How can we explain the persistence of these disparities? Why should SES and race have such an enduring and widespread association with mortality?

Two broad types of explanation have been offered and debated. Social causation explanations find the answer in the stress or adversity that is associated with lower SES or minority racial status. Genetic vulnerability explanations, by contrast, argue that genetic vulnerability leads to illness, which in turn impairs a person's ability to attain or maintain socioeconomic resources. A slightly different genetic explanation views genes as strong influences on factors like intelligence or on personality dimensions such as conscientiousness that in turn influence both health and SES.

Each approach offers a different explanation of the processes causing socioeconomic or racial gradients and carries different implications for how we might address them. Because of the extremely critical nature of the facts at issue, it is important to know which explanation or explanations are correct. One possibility is a "happy eclecticism" in which each hypothesized process provides a partial explanation. But this is an

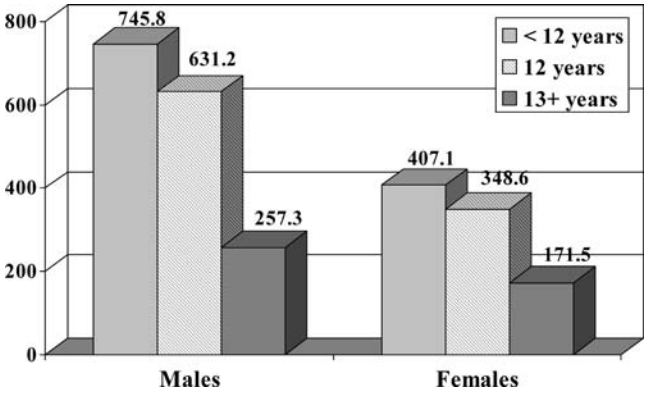


Figure 1. All-cause age-adjusted death rates per 100,000 people aged 25–64 years by education, 2001. [*Health United States 2003*. U.S. Department of Health and Human Services, National Center for Health Statistics. Health United States 2003.]

empirical question that can only be answered by confronting each explanation with the facts with which they must be consistent in order to claim validity. Here we bring current explanations of socioeconomic and racial disparities into contact with a powerful and robust set of facts that, we believe, the explanations have not yet been asked to come to terms with. The facts to which we refer are the dramatic improvements in health over the last half century.

## Improvements in Population Health

Figure 3 shows an increase in life expectancy at birth from only 47 years in 1900 to 77 years in 2000. For some of us, this enormous change occurred roughly within the relatively short time span between the birth of our parents and the birth of our children. Although much of this improvement is due to dramatic declines in infant mortality, life expectancy has also increased for people who are much older. For example, the average man turning 65 years old in 2000 can expect to live almost a year longer than the average man turning 65 years old in 1990. This is a remarkable change in a very short period of time. These

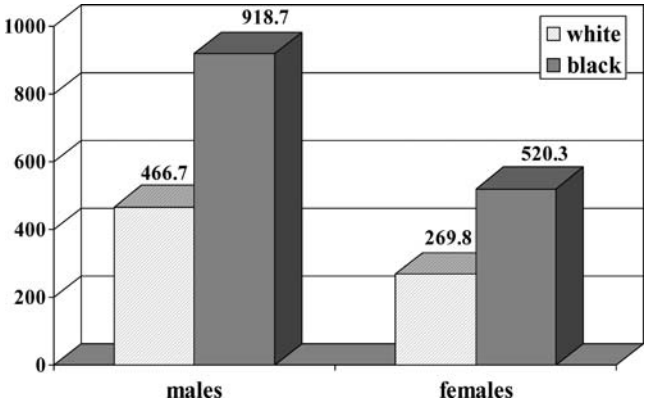


Figure 2. All-cause age-adjusted death rates per 100,000 people aged 25–64 years by race, 1998. [*Health United States 2003*. U.S. Department of Health and Human Services, National Center for Health Statistics.]

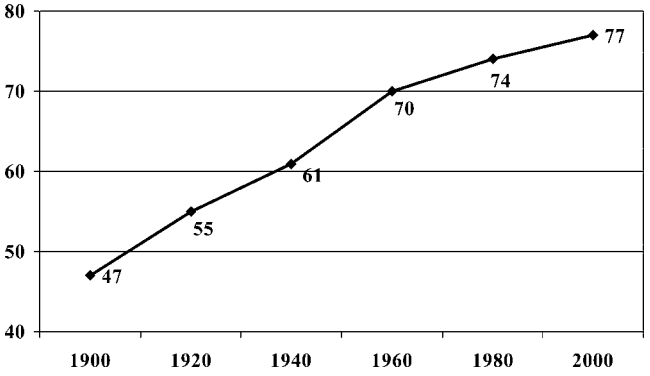


Figure 3. United States life expectancy at birth, 1900–2000. [*Health United States 2003*. U.S. Department of Health and Human Services, National Center for Health Statistics.]

improvements apply to many diverse causes of death, including some of the major killers of our time. Age-adjusted mortality rates per 100,000 people due to heart disease plummeted from 587 in 1950 to 258 in 2000. The figures are equally as dramatic for stroke, where rates fell from 181 per 100,000 in 1950 to 61 per 100,000 in 2000. For all cancers combined, age-adjusted mortality rates rose through 1990 but then began to drop significantly. Turning to infectious diseases, age-adjusted death rates from influenza and pneumonia dropped from 48 per 100,000 in 1950 to 24 per 100,000 in 2000.

What is driving these dramatic improvements in health? Clearly, some powerful processes are at work, which are having a remarkably strong impact on population health. Whatever these processes are—let us call them "X"—should not X also have relevance for explaining health disparities by SES and race? Certainly, explanations of health disparities should not ignore this factor. From this vantage point, let us return to the centerpieces of previous explanations for health disparities and ask whether these factors might be the same ones causing the dramatic improvements in population health we have observed. Let us begin with the first social selection explanation, in which genes affect health, which in turn affects SES. Clearly, disease-related genes cannot have changed so rapidly and in such a uniformly positive direction as to have created the enormous improvements in population health documented above. Next consider the second social selection explanation, in which genes influence factors like intelligence or conscientiousness, which influence both health and SES. Again, one would have to posit enormous and implausible gains in these traits over the last 50–100 years to explain the observed improvements in health. Turning to the mainstay of social causation explanations, we encounter similar problems. One would have to posit that the prevalence of stress or adversity has declined dramatically over the last several decades and that this decline had a powerful impact on illnesses as diverse as heart disease, influenza, and, since 1990, all cancers combined. It seems clear that whatever is driving improvements in population health, that factor is not coterminous with the primary factors that have been put forward to explain health disparities by SES and race.

So what is causing the dramatic improvements in population health—what is X? Of course, it is not one single thing but many different things. For example, recent declines in age-adjusted

rates of lung cancer are probably influenced by the lagged effects of declining smoking rates in previous decades. The rapid decline in HIV/AIDS mortality in the United States is probably related to antiretroviral drugs developed in the late 1990s. The precipitous decline in mortality due to Hodgkin disease since the 1960s is probably due to the development of chemotherapy treatments that were able to cure the disease. The list of factors that have contributed to improvements in population health is a long one, including screening for disease, public health efforts to increase the consumption of fruits and vegetables, promote exercise, and eradicate smoking, pollution control, flu shots, seat belts, cholesterol drugs, angioplasty, and so on. It seems clear that improvement in population health is not due to just one thing and that it is likely due to different factors for different diseases. Just as clearly, the confluence of these many factors has had an enormously positive impact on population health. Over the last century, human beings have dramatically increased their capacity to control disease and death.

## The Core Proposition: Controlling Disease and Creating Disparities

We can now state our core proposition: It is our enormously expanded capacity to control disease and death in combination with existing social and economic inequalities that create health disparities by race and SES. It does so because of a very basic principle. When we develop the ability to control disease and death, the benefits of this new-found ability are distributed according to resources of knowledge, money, power, prestige, and beneficial social connections. Those who are advantaged with respect to such resources benefit more from new health-enhancing capabilities and consequently experience lower mortality rates. Disparities are the result. This explanation for health disparities is a core component of the theory of fundamental social causes. Because our proposition derives from that theory, we briefly develop a broader rationale for the theory and then turn to evidence that bears on our core proposition.

## Fundamental Social Causes of Health Disparities

The theory of fundamental social causes begins with the graded relationship regularly observed between indicators of socioeconomic status, on the one hand, and health and mortality, on the other (Link & Phelan 1995, 1996, 2000). Clearly, biological mechanisms must be involved in this 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 statuses into the body. Despite the necessary role played by these mechanisms, the effect of SES on health and mortality cannot be understood by focusing only on the mechanisms that happen to link them at any particular time.

Imagine a causal model with SES as the distal factor that is linked to death by more proximal risk factors. If the proximal risk factors are eliminated, we would expect the SES–mortality association to disappear. On the contrary, there have been several notable instances in which major proximal risk factors were eliminated but SES disparities in mortality persisted. Consider Europe and the United States in the 19th century, when diseases such as cholera, diphtheria, measles, small pox, and tuberculosis were the major causes of death, and poor sanitation, contaminated water, and substandard and crowded living conditions were the principal risk factors. People of lower SES were exposed to these conditions to a greater degree and had much higher mortality rates as a consequence (Chapin, 1924; Villerme, 1840). Subsequently, however, sanitation was greatly improved, water systems were made safe, housing conditions dramatically improved, and effective vaccines were developed, with the result that death from cholera, diphtheria, measles, small pox, or tuberculosis is now rare in the United States and Western Europe. We would have expected the association between SES and mortality to disappear, because the mechanisms linking them were virtually eliminated or blocked. But it did not disappear. Why not? New risk factors (such as chemical pollutants) arose, new knowledge about risk factors (such as smoking) emerged, and new treatment technologies (such as medicines that reduce cholesterol) were developed, and those possessing the most resources were best able to avoid the new risks and take advantage of the new protective factors, resulting in the emergence of a socioeconomic gradient in these factors. The list of circumstances that are shaped by SES-related resources is very long and is not limited to the standard behavioral risk factors (e.g., smoking, exercise, diet) typically measured in risk factor epidemiology. For example, Lutfe and Freese (2005) use an ethnographic approach to study the management of diabetes; they show how the organization of clinics (single provider versus rotating providers), physicians' expectations of patients' capacity to use the newest diabetes control techniques, access to insurance, and many other circumstances result, on average, in an advantaged circumstance for those who are rich in socioeconomic resources. As Lutfe and Freese argue, it is when circumstances like these are reproduced across many situations (e.g., situations related to health behaviors, preventive health care, and the full range of existing diseases) that the robust association between SES-related resources and health emerges. As new discoveries are made, our ability to control disease advances, new items will be added to the list of health-enhancing circumstances, and, our theory says, those who command more resources will, on average, be better able to access and benefit from the new knowledge we gain. In this way, the association between SES and disease is reproduced dynamically through a complex and evolving set of intervening mechanisms that change over time and vary from place to place.

According to the fundamental cause idea, this dynamic reproduction of the association between SES and disease occurs because the flexible nature of resources of knowledge, money, power, prestige, and beneficial social connections allows the association to be reproduced in widely varying circumstances. Flexible resources are important in at least two ways. First, they directly shape individual health behaviors by influencing whether individuals are aware of, 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, social networks, and occupations that vary in their associated profiles of risk and protective factors. For example, white-collar jobs are less dangerous and more likely to include health-care benefits than blue-collar jobs. Low-income housing is more likely to be located in neighborhoods whose limited power and political organization make them

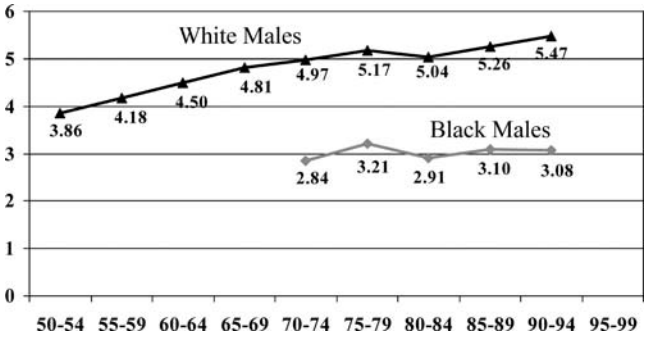


Figure 4. Brain cancer: Age-adjusted death rates per 100,000, 1950–1999 (males). [Cancer Mortality Maps and Graphs Web Site, National Cancer Institute: http://cancer.gov/atlasplus/.]

vulnerable to noise, pollution, and noxious social conditions, including targeted advertising of health-harming products such as tobacco and alcohol. Thus, the processes implied by the fundamental cause perspective operate at both individual and contextual levels.

## Evidence Concerning the Theory of Fundamental Social Causes

We argue that SES disparities in mortality arise because people of higher SES use flexible resources to avoid risks and adopt protective strategies. It follows that SES–mortality gradients should be diminished when people cannot use their resources in this way. Phelan, Link, Diez-Roux, Kawachi, and Levin (2004) constructed a test of the fundamental cause explanation by identifying a situation in which it is difficult to use resources to prolong life—when even the richest or most powerful person on earth cannot use resources to escape death. This is the case when we consider death from diseases that we do not yet know how to prevent or treat. If the utilization of resources is critical in prolonging life, then, when resources associated with higher status are useless, high SES should confer little advantage, and the usually robust SES–mortality association should be reduced. If these expectations were disconfirmed, it would pose a serious challenge to the theory.

### *SES Associations With More and Less Preventable Causes of Death*

Phelan and colleagues (2004) tested this prediction using the National Longitudinal Mortality Study (NLMS) and ratings they developed of the preventability of death from specific causes. The NLMS (Rogot, Sorlie, Johnson, & Schmitt, 1992; Sorlie et al., 1995) 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 9 years. Reliable ratings (intraclass correlation .85) of the preventability of death were made by two physician–epidemiologists. Causes were categorized into high preventability and low preventability groups. Common high preventability causes included cerebrovascular diseases, chronic obstructive pulmonary disease, ischemic heart disease, malignant neoplasm of the trachea, bronchus, and lung, and pneumonia and influenza; common low preventability causes included 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 explanation, Phelan and colleagues found that the SES–mortality association was significantly stronger for highly preventable causes of death than for less preventable causes of death. For example, for individuals between 45 and 64 years of age, the relative risk of death associated with having an eighth grade education as compared with more than a college education is 2.00 for high preventability causes as opposed to 1.21 for low preventability causes. Similarly, in the same age group, the relative risk of death associated with an income less than $5,000 compared with more than $50,000 is 2.81 for high preventability causes and 1.86 for low preventability causes.

As has been found previously (Antonovsky, 1967; Haan, Kaplan, & Camacho, 1987), socioeconomic inequalities in mortality diminished at older ages. In Phelan and colleagues' (2004) results, the mortality advantage of higher SES actually disappeared in the early 80s. Three possible explanations for these patterns are viable. First, socioeconomic variations in exposure to and impact of environmental and psychosocial risk factors are greatest in middle and early old age (House, Kessler, Herzog, Mero, Kinney, & Breslow, 1990). Second, the diminished association between education and mortality with advancing age found in cross-sectional or short-term longitudinal studies may be due to period rather than age effects (Lauderdale, 2001; Mirowsky & Ross, 2003). Third, as when we do not know how to prevent or treat life-threatening illness, old age can be seen as an instance in which socioeconomic resources are of limited use in prolonging life. In the case of low preventability causes of death, available resources are not useful because it is unclear how to direct them. In the case of old age, it is known how resource should be directed, but at some point, the growing frailty of the body places limits on the effectiveness of interventions. Each of these explanations is consistent with the fundamental cause perspective.

### *Time Trends in Socioeconomic and Racial Disparities in Mortality*

If our core proposition is correct, disparities by SES and race should emerge when new health-enhancing information or technology is developed. Diseases for which death has become dramatically more preventable include heart disease, Hodgkin disease, lung cancer, and colon cancer. In contrast, if death remains difficult or impossible to prevent, as it is for brain and ovarian cancer, disparities should not change substantially with time.

Phelan and colleagues' (2004) test did not provide evidence on changes in disparities over time. Here we report evidence bearing on these predictions. First consider mortality trends for diseases from which death remains difficult to prevent. Figure 4 presents age-adjusted brain cancer mortality rates per 100,000 men in the United States between 1950 and 1999. Consistent with the idea that we have not yet learned how to prevent death from this disease, the death rates remain steady or even climb slightly with time. Mortality rates are lower for Blacks than for Whites, and the difference between the groups remains

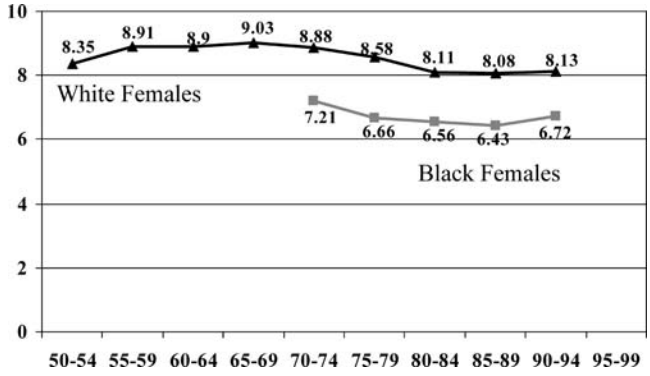


Figure 5. Ovarian cancer: Age-adjusted death rates per 100,000, 1950–1999 (females). [Cancer Mortality Maps and Graphs Web Site, National Cancer Institute: http://cancer.gov/atlasplus/.]

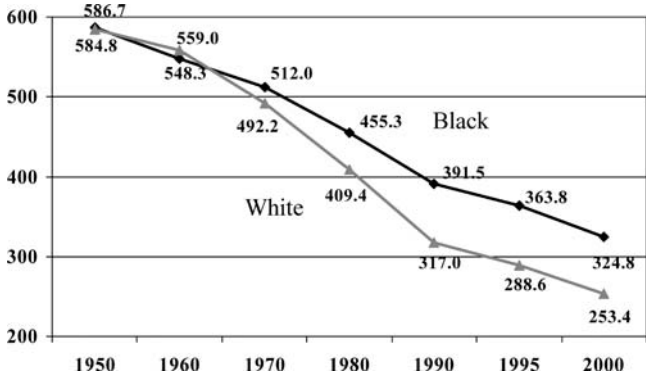


Figure 6. Age-adjusted heart disease mortality rates per 100,000 (1950–2000). [*Health United States 2003*. U.S. Department of Health and Human Services, National Center for Health Statistics.]

relatively constant through time. Age-adjusted mortality rates are lower for females than males, but the differences between Blacks and Whites and their time trends are very similar. Figure 5 shows age-adjusted mortality rates for ovarian cancer. Again, consistent with the idea that we are not yet effective at preventing death from this disease, age-adjusted mortality rates remain relatively constant through time as do the modestly lower rates for Blacks as compared with Whites.

Consider next some diseases for which great strides in prevention and treatment have been made. In order to capture the enormous changes that have taken place in heart disease since the 1950s, it is instructive to imagine oneself reflecting on the evidence available at that time. We can achieve this by returning to the work of one of the most prominent cardiovascular disease epidemiologists of the latter half of the 20th century, who was just beginning his career in the early 1960s. In 1964, Leonard Syme characterized heart disease as occurring "far more frequently among whites than Negroes" and wrote that "there seems to be little evidence of a systematic relationship between social class and coronary disease except perhaps in England where the rate appears to increase with socioeconomic status" (Syme, Hyman, & Enterline, 1964, p. 81). Consistent with the rates in England, Syme's (1964) well executed case–control study in North Dakota revealed a higher risk of heart disease in white collar as opposed to blue collar or farm workers. Figure 6 tells part of the story of what has occurred since Syme's observations in the 1960s. First, consistent with the idea that we have made great strides in our capacity to prevent death from this disease, age-adjusted rates declined rapidly between 1950 and 2000. Second, whereas rates of death for Blacks and Whites were quite similar in1950, disparities favoring Whites over Blacks have emerged since that time. In addition, death rates from heart disease are now much higher among those with lower levels of education. Taken together, this evidence is consistent with the idea that when we develop the wherewithal to prevent or treat a disease, groups that are richer in resources and that are less likely to experience discrimination (such as Whites and people with higher levels of education) benefit more fully from our new-found capacity to control disease.

Monitoring mortality rates by SES is more problematic than by race, because no indicators of SES were identified on death certificates until recently. To achieve some assessment of SES trends over time, Singh, Miller, Hankey, Feuer, and Pickle (2002b) employed principal components analysis to develop socioeconomic scores from 1990 Census data for every county in the United States. Scores were based on 11 variables measuring county-level SES. While all 11 variables loaded on one component, the 3 variables that were most important in defining that component were median family income, family poverty rate, and percentage of the population with more than 12 years of education. Singh, Miller, and Hankey (2002a) examined the stability of this measure using data from the 1970 and 1980 Census and found it to be quite stable.

Using quintiles of the socioeconomic scores, Singh and colleagues (2002b) observed dramatic evidence of changing associations between SES and two major cancer killers: lung cancer and colon cancer. Figure 7 shows age-adjusted lung cancer mortality rates for three quintiles (highest, middle, and lowest) for men aged 25–64 years. Rates were substantially higher in the highest SES counties in 1950. However, once evidence about the harmful effects of smoking emerged in the late 1950s and early 1960s, mortality in the highest SES counties began to flatten out, while rates in the poorest counties continued to rise. By 1998, the association between county-level SES and lung cancer mortality had completely reversed, such

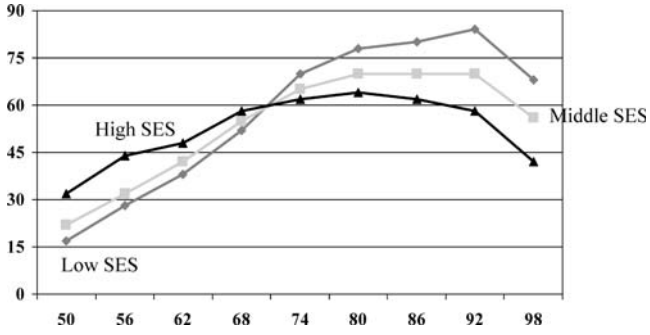


Figure 7. Age-adjusted lung cancer mortality rates per 100,000 (men aged 25–64 years), 1950–1998, by socioeconomic status (SES) of county of residence. [Adapted from Singh et al., 2002b. Changing Area Socioeconomic Patterns in U.S. Cancer Mortality, 1950–1998. *Journal of the National Cancer Institute, 94*, 916–925.]

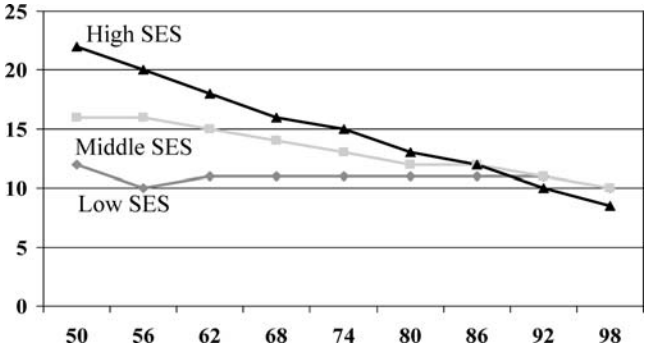


Figure 8. Age-adjusted colon cancer mortality rates per 100,000 (women aged 25–64 years), 1950–1998, by socioeconomic status (SES) of county of residence. [Adapted from Singh et al., 2002b. Changing Area Socioeconomic Patterns in U.S. Cancer Mortality, 1950–1998. *Journal of the National Cancer Institute*, *94*, 916–925.]

that the highest rates are now in the lowest SES counties. The situation for women is not as dramatic, but a more modest version of the same reversal seems to be underway: Until the late 1980s, women from high SES counties had the highest rates of lung cancer mortality, but by 1998, they had the lowest rates.

Figure 8 shows age-adjusted mortality rates from colon cancer in women (aged 25–64 years) in three quintiles of county-level SES (highest, middle, and lowest). As the figure shows, women in low SES counties had dramatically lower rates of colon cancer mortality in 1950. But as our ability to prevent death from this disease increased in the ensuing decades, women in high SES counties experienced a dramatic decline in mortality rates, while women in low SES counties did not. A similar pattern occurred for men such that, currently, overall mortality rates from colon cancer are modestly higher in low SES counties than in high SES counties.

### Potential Limitations

We consider two limitations that apply to the trend data reported in Figures 4–8. The first of these is the possibility that in examining specific diseases we fail to take account of so-called "competing risks." In this alternative explanation, disparities emerge when Blacks or individuals from lower SES areas begin to die at higher rates of diseases like heart disease or colon cancer because they survive long enough to do so. The death rates of these groups from heart disease or colon cancer rise (or fail to fall) because competing risks that may have taken lives in an earlier era (e.g., from infectious diseases) are less likely to take lives in a later one. We focused on relatively early death (before 65 years) to minimize this problem and also note that the effect of such competing risks would have to be very, very large to create the observed disparities. Finally, although the problem of competing risks applies to the time trends we presented, it does not apply to our earlier test using the NLMS, as those data were analyzed using a competing risks model (Phelan et al., 2004).

A second limitation of the time trend data is the possibility of diagnostic change over time by race and SES. It is possible that in an earlier era Blacks and people of low SES were less likely to receive accurate cause of death designations, producing lower rates in these groups for diseases like heart disease and colon cancer. Although this explanation is possible, we do not believe it accounts for the full pattern of results. First, using this reasoning, we might expect to find disparities emerging for diseases like brain cancer and ovarian cancer as well as for heart disease and colon cancer, but we do not. These low preventability diseases show a completely different pattern over time than do the high preventability diseases of colon cancer and heart disease. It is not evident how a diagnostic bias explanation would account for these differences. Second, our previous test of the fundamental cause explanation examines a period of just 9 years and is not nearly as subject to this particular form of bias.

### *Conclusions*

Considered together, these trends over time offer substantial support for fundamental cause theory. For two diseases for which there is no effective prevention or cure (brain cancer and ovarian cancer), we see no indication of emerging disparities over time by race or SES. In sharp contrast, evidence regarding several major killers indicates that disparities arose when new knowledge and technology gave us the capacity to prevent death from those diseases. People from groups that are richer in resources (Whites and people from high SES counties) were more likely to benefit from this new-found capacity, and disparities were created. To the extent that disparities are created in this manner, explanations of mortality must take account of our enormously expanded capacity to control disease and death and they must focus on who benefits from that capacity.

This fundamental cause explanation and the data that support it have implications for other theories that seek to explain disparities in health and mortality. Clearly, genetic factors and stress are enormously important determinants of morbidity and mortality. The point here is that any theory that posits an explanatory factor that has a constant effect over time will have difficulty accounting for disparities that change over time. If, for example, one posited a genetic basis for current racial disparities in coronary artery disease, one would have to explain why the same gene or set of genetic factors failed to produce the same disparities in earlier eras. Similarly, if relative deprivation and the anger, resentment, and envy associated with occupying a lower position in the social hierarchy explain the currently higher rates of heart disease in lower SES persons, why did the same processes not operate in the past? One would imagine that people lower in the hierarchy in 1950 would have experienced the same anger, resentment, and envy with the same pathological consequences. Similarly, if it is stress associated with a low hierarchical position that produces racial and socioeconomic disparities in heart disease and colon cancer now, why did those same stress processes not produce the same disparities in earlier eras? None of these explanations accounts for changes in disparities over time.

In order to account for disparities by race and SES, we need to recognize the great improvements in population health that have occurred over the last several decades, and we need to ask who benefits from that new-found capacity. Thus, the critical question for understanding disparities is who gets what in terms of risk and protective factors and why they get it. The fundamental cause explanation focuses attention on flexible resources of knowledge, money, power, prestige, and beneficial

social connections that can be used to harness advantages and avoid disadvantages in changing circumstances.

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