Is Racism a Fundamental Cause of Inequalities in Health?

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Abstract

We previously proposed that socioeconomic status (SES) is a fundamental cause of health inequalities and, as such, that SES inequalities in health persist over time despite radical changes in the diseases, risks, and interventions that happen to produce them at any given time. Like SES, race in the United States has an enduring connection to health and mortality. Our goals here are to evaluate whether this connection endures because systemic racism is a fundamental cause of health inequalities and, in doing so, to review a wide range of empirical data regarding racial differences in health outcomes, health risks, and health-enhancing resources such as money, knowledge, power, prestige, freedom, and beneficial social connections. We conclude that racial inequalities in health endure primarily because racism is a fundamental cause of racial differences in SES and because SES is a fundamental cause of health inequalities. In addition to these powerful connections, however, there is evidence that racism, largely via inequalities in power, prestige, freedom, neighborhood context, and health care, also has a fundamental association with health independent of SES.

INTRODUCTION

There is growing evidence that socioeconomic status (SES) is a fundamental cause of health inequalities. The hallmark of a fundamental cause is persistent health inequalities despite pronounced changes in diseases, health risks, and treatments. Large racial inequalities in health have also persisted across time despite changes in diseases and health-related factors. In this article we assess the viability of the idea that these racial inequalities have endured because racism is a fundamental cause of health inequalities. If so, we may face the same implication as for SES: Racial inequalities in health cannot be permanently eliminated by addressing proximate risk factors for disease and death, and long-term reduction of racial inequalities in health must address racism as a root cause of those inequalities.

SES AS A FUNDAMENTAL CAUSE OF HEALTH INEQUALITIES

Building on Lieberson (1985) and House et al. (1990), Link & Phelan (1995) developed the theory of fundamental causes to explain why the association between SES and mortality has persisted over centuries despite radical changes in the diseases and risk factors that have been presumed to explain it. Socioeconomic inequalities in health and mortality are large (Kunst et al. 1998, Sorlie et al. 1995) and have persisted at similar levels since the early nineteenth century (Antonovsky 1967) notwithstanding the major diseases and risk factors that appeared to account for the inequalities in earlier periods (e.g., deadly infectious diseases fueled by overcrowding and poor sanitation in low-SES communities) have been virtually eradicated in the developed world. Previous mechanisms have been replaced by newly prominent causes of death, including cancers and cardiovascular illness, fueled by risk factors such as poor diet and inadequate exercise, that are more common in lower-SES groups. Link & Phelan (1995) argued that this replacement occurs because, as risk factors for newly dominant diseases become apparent, higher-SES people are more likely to know about the risks and have the resources—money, knowledge, power, prestige, and beneficial social connections—to engage in prevention or treatment. Key to the importance of these resources is that they can be used in wide-ranging circumstances. If the problem is cholera, a person with greater resources is better able to avoid high-risk areas. If the problem is heart disease, a person with greater resources is better able to maintain a heart-healthy lifestyle and get the best medical treatment.

Link & Phelan (1995) summarized the essential features of a fundamental cause as follows:

> [A] fundamental social cause involves resources that determine the extent to which people are able to avoid risks for morbidity and mortality. Because resources are important determinants of risk factors, fundamental causes are linked to multiple disease outcomes through multiple risk-factor mechanisms. Moreover, because social and economic resources can be used in different ways in different situations, fundamental social causes have effects on disease even when the profile of risk factors changes radically. It follows that the effect of a fundamental cause cannot be explained by the risk factors that happen to link it to disease at any given time. (p. 88)

An important implication of the theory is that health inequalities resulting from a fundamental cause cannot be eradicated by addressing intervening mechanisms, because enduring inequalities in knowledge, money, power, prestige, and beneficial social connections ensure that mechanisms are reliably replaced. The long-term impact of the fundamental cause can only be eliminated by reducing inequalities in the flexible resources.

IS RACISM A FUNDAMENTAL CAUSE OF HEALTH INEQUALITIES?

Race in the United States has also had a large and enduring association with health and mortality. Black Americans have substantially worse health and shorter life expectancies than white Americans (Spalter-Roth et al. 2005). In 2010, life expectancy at birth for black Americans was almost 4 years shorter than for white Americans (75.1 versus 78.9). Even at age 65, life expectancy was about 1.5 years shorter for black Americans (National Center for Health Statistics 2014).

Although data on race and mortality cannot be traced back quite as far as data on SES, inequalities in Philadelphia were described by Du Bois [1967(1899)] just before the turn of the twentieth century and have been documented at a national level from 1900 onward. Life expectancy at age 40 has risen by roughly 10 years for whites and blacks (or nonwhites, depending on the period of data collection) (Carter et al. 2006), and there is some evidence that the absolute mortality gap has diminished somewhat in recent years (Harper et al. 2007, 2012). However, most studies find that the relative mortality gap remains remarkably persistent (Carter et al. 2006, Elo & Drevenstedt 2004, Levine et al. 2001, Satcher et al. 2005, Sloan et al. 2010; but see Hummer & Chinn 2011) across a period characterized by radical changes in the major causes of mortality (Omran 1971), accompanied by changes in risk and protective factors and medical treatments, and across a period characterized by major changes in the life circumstances of black Americans that one might have thought would result in a significant narrowing in the life-expectancy gap.

These persistent racial inequalities in mortality motivate us to investigate whether, as in the case of SES, there are fundamental-cause processes in play. In this article, we evaluate evidence bearing on whether racism is a fundamental cause of racial differences in health and mortality. Specifically, the model we propose and evaluate consists of three sets of fundamental associations: (*a*) Racism is a fundamental cause of racial differences in SES; (*b*) SES is a fundamental cause of inequalities in health and mortality; and (*c*) racism is a fundamental cause of racial differences in health and mortality independent of SES. This model is illustrated in **Figure 1**, which shows systemic racism influencing flexible resources via multiple replaceable mechanisms, which in turn affect racial differences in health via multiple replaceable mechanisms. Some of these resources, such as income, are an inherent part of SES, but others, such as neighborhood conditions, are not. In **Figure 1**, the resources that are coterminous with SES are shown in the smaller circle. Using

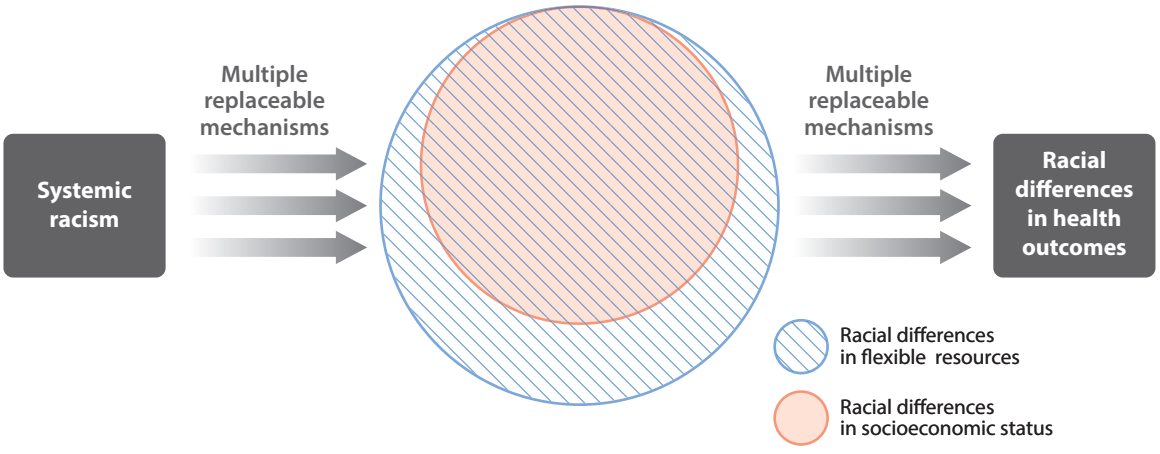

Figure 1

Racism as a fundamental cause of health inequalities: conceptual model.

evidence that a large portion of racial differences in health outcomes are attributable to racial differences in SES (Do et al. 2012; Franks et al. 2006; Hayward et al. 2000; House & Williams 2000; Rogers et al. 2000; Sorlie et al. 1992, 1995), we show the smaller circle taking up a large portion, but not the totality, of the flexible resources that mediate the effect of racism on racial differences in health outcomes. The fundamental association between SES and health outcomes was discussed above. The bulk of this article evaluates whether racism is a fundamental cause of SES and whether racism is a fundamental cause of health outcomes independent of SES.

FUNDAMENTAL CAUSALITY AS A GENERIC CONCEPT

As originally formulated, fundamental-cause theory sought to explain enduring associations between a specific independent variable (SES) and a specific set of dependent variables (health outcomes). In order to analyze racism as a fundamental cause and SES as its consequence, we must broaden this definition. This is possible because, as Freese & Lutfey (2011) articulated, fundamental causality is a particular type of causal relation that need involve neither SES nor health. Here we propose a generic definition of fundamental causes:[1] A fundamental cause embodies a set of flexible resources, and a superior set of resources generates superior results on some outcome. The level of resources varies between social groups, and groups with superior resources are advantaged on the outcome. The flexibility of the resources allows them to influence the outcome under a wide range of circumstances and through multiple pathways. New circumstances may arise that diminish or eliminate particular pathways connecting the fundamental cause to the outcome. However, when new circumstances that affect the outcome emerge or are created, the superior resources of some groups will tend to advantage them in terms of the new circumstance and will tend to create a new pathway connecting the fundamental cause to the outcome. The ebb and flow of specific pathways and the replacement of old pathways with new pathways connecting the fundamental cause to the outcome result in an enduring connection between the fundamental cause and the outcome.

RACISM AS A FUNDAMENTAL CAUSE OF RACIAL DIFFERENCES IN SES

There are profound differences between black and white Americans in every measure of SES. Black median household income is three-fifths that of whites (DeNavas-Walt et al. 2012); black family wealth is less than one-sixth that of whites (McKernan et al. 2013); 31% of whites versus 21.2% of blacks hold a bachelor's degree (US Census Bureau 2013); there are differences in the quality of education received by blacks and whites (Maxwell 1994); occupational prestige is lower for blacks than for whites (Kim & Tamborini 2006, Lemelle 2002); blacks are much more likely than whites to hold service occupations (25.4% versus 16.6%); conversely, roughly twice as many whites as blacks hold managerial occupations (11.6% versus 6.4%) (BLS 2013). Although blacks make up about 12% of the US population, they constitute only 1.2% of Fortune 500 CEOs (Aguilar 2013), 2% of senators (Wikipedia 2015), 2% of governors (Roll Call 2013), and 9.2% of US Congressional Representatives (Roll Call 2013). To consider whether racism may be a fundamental cause of these stark racial inequalities, we employ Feagin's (2000) concept of systemic racism. According to Feagin, systemic racism in the United States was born of the economic advantages of slavery

[1] Freese & Lutfey (2011) provide a broad definition of fundamental causality. However, we constructed our own definition that retains our original emphasis on flexible resources (identified by Freese & Lutfey as one of four meta-mechanisms connecting a fundamental cause to an outcome).

for whites when the nation was founded, and since that time our major institutions have been pervaded by racial stereotypes, ideas, emotions, and practices, reproducing over time the socioeconomic conditions that undergird systemic racism. Like a fundamental cause, systemic racism embodies a set of flexible resources that advantage whites, including (*a*) structural factors, such as the domination by whites of major government and commercial institutions, including elected and appointed government offices at national, state, and local levels, educational institutions, the penal and legal systems, mass media, real estate, banking, medicine, entertainment, and other commercial enterprises; (*b*) individual resources of money, knowledge, power, prestige, and beneficial social connection, which are held disproportionately by whites; and (*c*) social psychological advantages, such as expectations that whites should dominate, beliefs that whites are superior, and desires of whites for social distance from blacks. The broad range and flexibility of the resources that racism provides whites facilitate the reproduction of SES inequalities by race.

CHANGING MECHANISMS CONNECTING RACISM TO SES

Fundamental causality entails the replacement of causal mechanisms over time, and Hatzenbuehler et al. (2013) provide evidence that this has been true of the connection between racism and SES in the United States. From the nation's founding to the Civil War, slavery was an extremely potent mechanism connecting systemic racism to racial inequalities in SES. When slavery was abolished in 1865 and black American men gained the right to vote in 1870, two important mechanisms linking systemic racism to racial differences in SES were eliminated. Subsequently, however, Ku Klux Klan terror; Jim Crow laws maintaining segregation; and legal, overt discrimination in housing, employment, and schooling became prominent mechanisms for maintaining white privilege (Feagin 2000, Van Woodward 1955).

The Civil Rights Act of 1964 might be presumed to have eliminated segregation and discrimination as mechanisms connecting systemic racism to racial differences in SES, but this is not the case. Overt forms of segregation and discrimination, such as separate public facilities for blacks and whites, job or real estate ads that specify whites-only, or explicit rejection of black students on the basis of their race, have been largely eliminated. However, the Civil Rights Act has not been consistently enforced (Feagin 2000), and legal decisions have not consistently upheld it. For example, as of this writing, racial discrimination in higher education is gaining legal ground. In the areas of housing and the workplace, experimental audit studies clearly demonstrate that covert discrimination, illegal but difficult to detect, remains significant (Pager 2003, Turner & Ross 2005). For example, Pager (2003) found that white men identified as having a criminal record received more callbacks for job interviews than black men with no criminal record. In public accommodations, refusal to serve black Americans is illegal, but disrespect communicated by poor service is commonly experienced by black Americans (Feagin 1991). In 1954, the US Supreme Court made racial segregation in public schools unconstitutional. Yet owing to a series of court decisions and policies, schools remain highly segregated by race, and schools that vary in the proportion of white and black students they educate receive highly unequal resources (Orfield et al. 1996).

Throughout this history, a sequence of social psychological processes advantaging whites have also been in play. The ideology of racism specifies that whites are superior and better suited to roles of power and responsibility than blacks (Feagin 2000). The open expression of such beliefs has declined substantially (Schuman et al. 1997), but racist attitudes and beliefs persist in more covert forms. Feagin (2010) analyzed diary data from 626 white college students from several regions of the United States and, on the basis of their experiences in all-white interactions, found frequent observations of blatantly racist commentary, often expressed in a jocular manner. One diarist reflected, "It is this sort of 'joking' that helps to keep racism alive today. People know

the places they have to be politically correct and most people will be. However, until this sort of 'behind-the-scenes' racism comes to an end, people will always harbor those stereotypical views that are so prevalent in our country" (p. 127).

For the covert and subtle forms of discrimination and prejudice we have mentioned so far (e.g., covert job discrimination, racial "joking"), it is likely that individuals are aware of and avow their negative attitudes and behaviors in some way. However, the ideology of racism also operates at an implicit, automatic level (Greenwald et al. 2009). Although these implicit beliefs, expectations, and emotions may operate outside conscious awareness, they have important effects on the behavior of both blacks and whites. For example, aversive racism (Dovidio & Gaertner 1998), an implicit or unconscious bias against blacks on the part of whites who verbally eschew racial prejudice, affects discriminatory behavior toward blacks and hampers interpersonal interactions and cooperation between blacks and whites (Penner et al. 2010). In addition, through a process known as stereotype threat (Steele 1997), high-achieving young black Americans have been shown to experience anxiety that they will confirm negative stereotypes about their group. Such awareness leads to decrements in performance on achievement tests, which in turn hamper academic achievement and confirm stereotypes that blacks are intellectually inferior to whites.

In sum, we observe racial differences in a multitude of resources connected to multiple dimensions of SES via a succession of multiple mechanisms linking systemic racism to racial differences in SES. Finally, the theory predicts that, under these conditions, the fundamental cause will maintain an association with the outcome over time despite radically changing conditions. The data are consistent with this expectation. As educational attainment, income, and wealth have increased over time for both black and white Americans, blacks have gained proportionately to whites on all these outcomes, but the racial gaps in median income (since 1948), wealth (since 1983), and percent with at least four years of college (since 1940) have remained relatively steady or grown (McKernan et al. 2013; US Census Bureau 2014, 2015).

More specific empirical data may be necessary to firmly establish systemic racism as a fundamental cause of racial differences in SES, but we find the extant data to be strongly, broadly, and cohesively consistent with the theory. We conclude that much of the enduring association between race and health inequalities in the United States results from two fundamental associations: one between racism and racial inequalities in SES and a second between SES and inequalities in health outcomes.

RACISM AS A FUNDAMENTAL CAUSE OF RACIAL HEALTH INEQUALITIES INDEPENDENT OF SES

However, this is not the whole story of race and health. Although most studies find that SES explains much of the race difference in health outcomes, they also find that significant health inequalities between black and white Americans remain when SES is controlled (Franks et al. 2006; Hayward et al. 2000; House & Williams 2000; Hummer 1996; Rogers et al. 2000; Sorlie et al. 1992, 1995; see Do et al. 2012 for an exception). In a 10-year prospective study, Sorlie et al. (1995) found that, for middle-aged participants, the age-adjusted risk of death was 1.68 for black men relative to white men and 1.72 for black women relative to white women. After controlling for employment status, income, education, marital status, and household size, these relative risks were reduced to 1.28 for men and 1.35 for women, both statistically significant at the 0.01 level (Sorlie et al. 1995).

Others (notably Hummer 1996, Williams & Collins 1995, Williams & Sternthal 2010) have argued that racism needs to be directly addressed to understand racial inequalities in health that are not attributable to SES. Fundamental-cause theory can help us understand the "residual"

association between race and health by directing our attention to the theory's core construct of flexible resources—resources that can be used under a broad variety of conditions to avoid risk or minimize consequences of disease. Specifically, we focus on flexible resources that differ by race but that overflow the bounds of SES. These resources are shown in **Figure 1** as the blue-striped area of the central circle. We refer to these as race-related resources. We see these resources operating in exactly the same way as SES resources do; that is, racism is a fundamental cause of inequalities in race-related resources, and those resources, as in the case of SES, fundamentally cause inequalities in health and mortality.

To explore whether racism, via flexible resources outside the bounds of SES, serves as a fundamental cause of racial differences in health, we review each of Link & Phelan's fundamental-cause criteria and use existing data to evaluate whether they apply to race. Do black and white Americans have unequal access to flexible resources? Are black and white Americans unequal in terms of multiple disease outcomes? Do racial inequalities in health outcomes result from multiple risk factors? Are racial inequalities in health reproduced over time via the replacement of intervening mechanisms? Because we are trying to establish whether racism is a fundamental cause of health inequalities independent of SES, we also require that each criterion hold when controlling for SES. We apologize to our readers for the complexity and perhaps tedium introduced by these conditions that we imposed on our review. However, we feel these conditions are necessary to seriously evaluate whether racism is a fundamental cause of health inequalities. Space limitations require that this be a selective, rather than comprehensive, review of the relevant literature, and we limit our attention to physical health and mortality.

Is Race Associated with Flexible Resources Independent of SES?

We begin by elaborating a set of flexible race-related resources. Specifically, we focus on nonoccupational prestige and power, beneficial social connections related to neighborhood segregation, and a newly conceptualized flexible resource—freedom.

Prestige. Prestige, or status, as defined by Weber (Gerth & Mills 1946), is honor or deference attached to a person or social status and distributed unequally in a social group. Much of the prestige hierarchy is embodied in occupational structures and thus falls within the domain of SES. Black and white Americans are unequal in terms of occupational prestige, as we documented above. In addition, however, the ideological components of systemic racism (stereotypes and devaluation of black Americans) produce inequalities in the resource of prestige that are specific to race. Here we review evidence on three examples: racial stereotypes, implicit evaluations of "good" and "bad," and disrespectful behavior.

Despite declines in openly avowed racial stereotypes (Schuman et al. 1997), the 2000 General Social Survey found rather striking levels of racial stereotypes: Among white respondents, 20.5% believed that blacks were unintelligent, compared with 8.5% who thought whites were unintelligent; 36.5% thought blacks, compared with 10.4% who thought whites, were lazy; 58.9% thought blacks, compared with 4% who thought whites, preferred to live off welfare; and 50.5% thought blacks, compared with 17.4% who thought whites, were prone to violence (GSS 2007).

A large body of research (Nosek et al. 2007) finds that whites reveal a significant implicit or unconscious preference for whites over blacks, most often measured as a tendency to associate photographs of blacks with the concept "bad" and photographs of whites with the concept "good." Using such measures, Project Implicit reports that 76% of whites have a prowhite bias (Blow 2014). In the same vein, in an experimental study in which identical pairs of baseball cards were offered for sale on the online auction site eBay, one card shown was held by a black hand and one by a white hand. Cards held by black hands sold for 20% less (Ayres et al. 2011).

In the research above, participants respond to whites and blacks without any specification of their SES. Although it seems that stereotypes and implicit bias are attached more likely to race than to SES, the possibility cannot be ruled out that, to some unknown extent, blacks are presumed to have lower SES than whites and that presumed SES levels drive negative responses to blacks. However, some research explicitly controlling for SES demonstrates racial differences in prestige. For example, in a survey about postangiogram patient encounters controlling for patient SES, physicians described black patients as less intelligent, less likely to adhere to medical advice, more likely to engage in high-risk behavior, and felt less affiliation toward black patients (van Ryn & Burke 2000).

Qualitative research by Feagin (1991) also circumvents SES as a possible explanation for negative attitudes toward blacks by focusing on middle- and upper-middle-class blacks, who consistently reported numerous instances of being treated disrespectfully by whites. To illustrate, "A successful black business owner recounted: 'we had a new car. . . . And we stopped at 7-11. . . . We pulled up, and my husband was inside at the time. And this person, this Anglo couple, drove up, and they hit our car. It was a brand new car. So my husband came out. And the first thing they told us was that we got our car on welfare'" (Feagin 1991, p. 112). Thus, there is evidence that prestige is a resource where we observe racial inequalities outside the bounds of SES.

Power. Power can be defined as the intended and successful control of others (Wrong 1979). Much power is associated with occupations and wielded in the context of work and thus is an aspect of SES. Black and white Americans are unequal in terms of occupational power, as we documented above. However, power can also be measured outside the context of work in ways that are independent of SES. Research in the status characteristics tradition examines the impact of status labels on influence (i.e., power) in goal-oriented cooperative groups. Two such studies in which SES was partially controlled found lower influence for blacks than for whites (Cohen & Roper 1972, Webster & Driskell 1978). In the first study, partners were fellow classmates in a junior high school class, and in the second study, they were classmates at the University of Southern California.[2]

Beneficial social connections. Are there racial differences in beneficial social connections that operate independently of SES? We conclude the answer is yes and that racial segregation is a major source of these inequalities. Neighborhoods powerfully structure social connections. Not only do neighborhoods influence exposure to other individuals with whom a social connection may be more or less beneficial, but the individual resources of one's neighbors also strongly affect collective resources (stores, schools, gyms, parks, street lights, fire and police protection, health care) that are more or less beneficial to health. In Freese & Lutfey's (2011) terms, neighborhood effects are in large part spillover effects, in which individuals benefit from collective resources without their active participation. How do neighborhoods bear on racial differences in beneficial social connections? First, black and white Americans tend to live in different neighborhoods (Massey & Denton 1993). In metropolitan areas, in 2010, the average white person lived in a neighborhood that was 75% white and 8% black, whereas the average black person lived in a neighborhood that was 45% black and 35% white (Logan & Stults 2011). Second, black Americans, regardless of their own income, live among neighbors with poorer socioeconomic resources who are less likely to provide beneficial connections for things such as job procurement, admission to college, or access to political power holders. Between 2005 and 2009, the average affluent black household (income

[2]Influence as assessed in these experiments does not meet all the criteria for power, because the influence is not necessarily intended. Nevertheless, the same patterns would presumably hold if the influence were intentional.

of $75,000 or more) lived in a poorer neighborhood than the average lower-income (less than $40,000) white household (Logan 2011). Third, collective resources that may affect health, such as stores and parks, are also inferior in neighborhoods with more black residents. We detail below some of the specific neighborhood resources that have been empirically linked to health outcomes.

Freedom. Considering fundamental-cause theory from the perspective of race has led us to consider an addition to our list of flexible resources—freedom. Whereas power refers to the ability to control others, freedom entails the ability to control one's own life circumstances and actions (Sen 1999). It is a flexible resource in that it allows one to protect one's health in a variety of circumstances. An enslaved person does not have the freedom to avoid harm from his or her owner or overseer, to avoid harsh working conditions, to relocate to escape infectious disease, or to control his or her access to health care. An imprisoned person does not have the freedom to avoid violent victimization, to benefit from family and friends' social support, and, possibly, to exercise and eat healthful foods.

Do black Americans have less freedom than white Americans? Before the US Civil War, slavery was a severe and very prevalent form of unfreedom, and slavery was extremely maldistributed by race. In 1790, 92.1% of black Americans were slaves; in 1860, 89% were slaves (Bailey 1994). No whites were slaves. Slavery was abolished in 1865, ending that form of unfreedom for black Americans.

Currently in the United States, imprisonment is an extreme and prevalent form of unfreedom. In 2001, the imprisonment rate, at 472 per 100,000, approached five times its historic average (Pettit & Western 2004). Imprisonment is greatly maldistributed by race. In 1999, non-Hispanic white men born between 1965 and 1969 had a cumulative risk of imprisonment of 2.9, compared with a risk of 20.5 for non-Hispanic black men. Risk of imprisonment is strongly related to educational attainment as well as race, but very large racial inequalities are found at every educational level. For those with some college, the risk for white men was 0.7 and 4.9 for black men. For those with a high school diploma or GED, the risk was 3.6 for white men and 18.4 for black men. For those who did not complete high school or a GED, the risk was 11.2 for white men and 58.9 for black men (Pettit & Western 2004). This extraordinarily high risk of imprisonment for poorly educated black men has led researchers to describe imprisonment as a normative part of early adulthood for black men in poor urban neighborhoods (Freeman 1996).

Slavery and imprisonment are extreme examples of unfreedom. Less extreme but very real examples include official and unofficial harassment, discrimination and threat of harm that thwart black Americans' freedom of movement and behavior, and placing limitations on how one dresses and behaves and where one walks, shops, eats, or drives (Feagin 1991, Harris 1999). Another highly consequential unfreedom is discrimination in housing and employment, which thwarts black Americans' ability to live and work where they want (Pager 2003, Turner & Ross 2005). All these sources of unfreedom hold controlling for SES.

In sum, we have shown evidence for racial differences in every flexible resource proposed by Link & Phelan (1995). Many of the important differences, i.e., large racial differences in income, wealth, formal education, and occupational prestige and power, cannot be said to hold controlling for SES, because those resources are components of SES. However, we also documented significant differences in resources controlling for SES. These included prestige as measured by negative stereotypes, devaluation, and disrespectful treatment; power as measured by influence in groups; freedom as measured in terms of slavery and incarceration; and beneficial social connections as operationalized by neighborhood segregation and inequality.

Returning to **Figure 1**, we now ask whether racism leads to differences in race-related resources via replaceable mechanisms and whether race-related resources lead to racial differences in health

outcomes via replaceable mechanisms. Regarding the former question (**Figure 1**, *left*), we refer back to our section on racism as a fundamental cause of racial differences in SES, where we pointed to the enduring effect of systemic racism on racial differences in SES via a sequence of mechanisms at multiple levels, including slavery and disenfranchisement, racial violence, legal and covert segregation and discrimination, overtly and covertly expressed and implicit stereotypes, and differential valuation of whites and blacks. These sequences of replaceable mechanisms not only affect SES outcomes, but equally influence our race-related resources. Indeed, perhaps the most striking example of mechanism replacement bears on a race-related resource—the replacement of slavery by incarceration in the denial of freedom of African American men.

Is Race Related to Multiple Disease Outcomes via Multiple Mechanisms Independent of SES?

According to fundamental-cause theory, differences in access to flexible resources should result in inequalities in not one or two, but many disease outcomes. This is because the flexibility of resources allows them to be applied to a variety of situations relevant to many different health outcomes (**Figure 1**, *right*). Because of racial differences in flexible resources that result from racism, we expect this to be the case for race.

In fact, race is related to virtually all major disease outcomes. In 2000, black Americans had elevated death rates for 8 of the 10 leading causes of death (National Center for Health Statistics 2004). Reporting on the prevalence and incidence of six major fatal conditions (hypertension, diabetes, cancer, chronic obstructive pulmonary disease, heart disease, and stroke), nine nonfatal diseases and physical impairments (arthritis, asthma, back, foot and leg, kidney, stomach, vision and hearing problems, and pain), and five types of disability for men and women, Hayward et al. (2000) found that, of the 80 racial comparisons, blacks fared worse on 80% of the outcomes. Rogers et al. (2000) reported significant elevations in mortality for each of the categories of circulatory, cancer, social pathologies, and other.

This broad association between race and health also holds when SES is controlled. As with overall health, controlling SES reduces but does not eliminate racial differences in multiple health outcomes. Hayward et al. (2000) found that education accounted for much of the inequalities in chronic diseases and disabilities but did not eliminate them. Similarly, Rogers et al. (2000) found that blacks' elevated rates of mortality were diminished but remained significant when SES and other demographic factors were controlled, except for social pathologies, for which blacks had lower mortality rates once these controls were included.

On the basis of fundamental-cause theory, we also expect race to be related to disease outcomes via multiple mechanisms independent of SES. Following Hummer (1996), Williams & Collins (2001), and others, we review a variety of situations and processes that connect race to health outcomes.

Stress. Social stress is a response to threatening or burdensome situations that induces physiological responses that can harm health. Both acute and chronic stress have been linked to mortality and to numerous health outcomes (House 2002, Lantz et al. 2005).

Experiences of discrimination. Discrimination has been conceptualized as a social stressor (Clark et al. 1999, Meyer 2003) that may be particularly harmful to health because it is uncontrollable and unpredictable (Williams & Mohammed 2009), spans the life course, is encountered in multiple contexts, and induces a psychically painful state of vigilant anticipation (Pearlin et al. 2005). Studies of the health consequences of discrimination take two general forms. In the first, participants

report on experiences of discrimination in their lives, and these are related to outcomes such as hypertension, self-reported poor health, breast cancer, obesity, and hypertension.[3] In the second, participants are exposed to analogues of racist events (such as watching a racist film clip) in a laboratory setting, and cardiovascular reactivity or psychological stress responses (such as anger) are measured. Experiences of discrimination are associated with poorer health-related outcomes in both types of studies (Paradies 2006, Pascoe & Smart Richman 2009, Williams & Mohammed 2009). The magnitude of these associations was summarized in a meta-analysis of 134 samples relating experienced discrimination to health-related outcomes. Pascoe & Smart Richman (2009) found significant correlations between experienced discrimination and each of the three health-related outcomes examined: physical health (mean $r = -0.13, p < 0.00$), stress response (mean $r = 0.11, p < 0.00$), and health behaviors (mean $r = -0.21, p < 0.00$). Although some of these studies included discrimination based on characteristics other than race, 65% of the studies examined racial discrimination.

These associations between discrimination and health cannot easily be attributed to SES. In the first set of studies, although results are not consistent (Paradies 2006), higher-SES participants are generally more likely than lower-SES participants to report discrimination. Hence, it is unlikely that experiences of discrimination are actually due to low SES rather than to race itself. The stress-response studies do not directly control for SES; however, these studies are experimental, for example, randomly exposing participants to a racist or a neutral film clip. Because SES cannot be a confounder in such experimental studies, they effectively control for SES.

Other stressors. In addition to direct experiences of discrimination as a stressor, other forms of stress, reflecting more general life circumstances, are much more common among black Americans. For example, in a random sample of 1,200 members of a large health maintenance organization, Breslau et al. (1995) found that, prospectively over a period of three years, black Americans were 2.26 times as likely as white Americans ($p < 0.0001$) to be exposed to traumatic events, as defined by DSM-III-R, controlling for education as well as gender, extroversion, neuroticism, early misconduct, and family history of psychiatric disorder.

Weathering. Geronimus (1992) proposed the weathering hypothesis, according to which blacks experience early physiological and health deterioration as a consequence of the cumulative stress of living in a society that stigmatizes and disadvantages them. McEwen and colleagues (e.g., McEwen 1998) developed the concept of allostatic load, that is, the cumulative wear and tear on the body's systems owing to repeated adaptation stressors. Consistent with the weathering hypothesis, Geronimus et al. (2006) found that at all ages black Americans had higher mean allostatic load scores than whites. Importantly for the questions addressed in this article, blacks' elevated allostatic load scores were not explained by poverty. High allostatic load is significantly associated with racial identity among African Americans, controlling for SES and reported experiences of discrimination (Brown & Turner 2014), suggesting that black Americans with a strong racial identity may experience the distress of the social stigmatization of and discrimination against this group more acutely, above and beyond their own personal experiences.

Medical care. There is abundant evidence that black Americans receive lower-quality health services than white Americans do, and these inequalities are remarkably consistent across a range of illnesses and health care services (National Center for Health Statistics 2012, Smedley et al.

[3]Much of the research on the effects of perceived discrimination has focused on mental health. However, our focus in this article is physical health.

2003, Spalter-Roth et al. 2005). Black Americans are less likely to undergo coronary artery bypass surgery (Ayanian et al. 1993, Petersen et al. 2002), are less likely to receive peritoneal dialysis and kidney transplantation (Epstein et al. 2000), are more likely to receive a lower quality of basic clinical services such as intensive care (Ayanian et al. 1999), and experience greater delays in transfer from hospitals to nursing homes (Weissert & Cready 1988) even when variations in such factors as insurance status, income, age, comorbid conditions, and symptom expression are taken into account. Critical for their importance as a mechanism linking race to health outcomes, inequalities in medical care are associated with greater mortality among black patients (Bach et al. 1999, Peterson et al. 1997).

Neighborhood effects. The availability of flexible resources is influenced by the neighborhoods in which one lives and works. Neighborhoods bundle together beneficial or less–than-beneficial sets of circumstances, such as the availability of healthful foods and good medical care. The importance of neighborhoods in understanding racial differences in health is hard to overstate (Morenoff 2003, Spalter-Roth et al. 2005, Williams & Collins 2001). Williams & Collins (2001) in fact identified racial residential segregation as a fundamental cause of racial disparities in health. We documented above that blacks tend to live in different and poorer neighborhoods than whites. In turn, residing in poor (Kawachi & Berkman 2003, Robert 1999) and segregated (Collins & Williams 1999, King et al. 2011) neighborhoods has been linked to poor health and mortality, independent of individual-level SES. Below we examine racial differences in specific neighborhood conditions that have been linked to health outcomes. Each of these individual conditions (toxic exposures, nutrition) can be considered a mechanism connecting race and health outcomes, but they are strongly shaped by neighborhood and are considered here under the umbrella of neighborhood effects.

Recreation. Segregated black neighborhoods contain fewer recreational outlets than white neighborhoods (Moore et al. 2008). There is a strong relationship between neighborhood recreational resources and physical activity (French et al. 2001), and physical inactivity is associated with obesity, diabetes, hypertension, cardiovascular disease, and cancers (Auchincloss et al. 2008, CDC 2004).

Nutrition. Segregated black neighborhoods contain two to three times as many fast-food outlets as white neighborhoods of comparable SES (Powell et al. 2007). Fast-food outlets contribute to blacks' consuming more fast food, which contributes to racial disparities in obesity and diabetes (French et al. 2001, Pereira et al. 2005). Predominantly black neighborhoods have two to three times fewer supermarkets than white neighborhoods of comparable SES (Moore & Diez Roux 2006, Powell et al. 2007). The availability of supermarkets is associated with more fruit and vegetable consumption and lower rates of obesity, diabetes and other conditions (Auchincloss et al. 2008; Morland et al. 2002, 2006).

Harmful substances. Research shows that the tobacco and alcohol industries target minority neighborhoods for advertising and sales (Hackbarth et al. 2014, LaVeist & Wallace 2000). In turn, controlling for individual SES and family history of alcohol problems, Kwate & Meyer (2009) found an association between exposure to alcohol advertising and problem drinking in a predominantly black community.

Protection and crime. Segregated black neighborhoods have poorer fire and police protection (Evans & Saegert 2000), and crime rates are higher (Massey 2004). Nationally, the age-adjusted

death rate from homicide for African Americans is more than five times as high as that for white Americans (National Center for Health Statistics 2012). In addition, perception of neighborhood safety is associated with level of exercise (MMWR 1999).

Toxic environmental exposures. Environmental exposures in minority neighborhoods are 5 to 20 times as high as exposures in white neighborhoods, after controlling for neighborhood SES (Hofrichter 2000, Morello-Frosch & Jesdale 2006, Morello-Frosch & Lopez 2006). In turn, toxic environmental exposures contribute to disease and poor birth outcomes (Basil et al. 2007, Diez Roux et al. 2008, Kouznetsova et al. 2007, Parker et al. 2005).

Medical care. We have already considered racial differences in the quality of medical care. These inequalities apply not only to individuals but to neighborhoods. Hospitals in neighborhoods with more black residents have fewer technological resources, specialists, and board-certified physicians, and higher rates of negligent adverse events and mortality for both black and white patients (Smedley et al. 2003).

In sum, consistent with expectations based on our conceptual model of racism as a fundamental cause of health inequalities (**Figure 1**), a "multiplicity of mechanisms" (Lutfey & Freese 2005), many of which are channeled through highly disparate neighborhoods in which black and white Americans live, link race to health outcomes independently of SES.

Are Racial Inequalities in Health Reproduced Over Time via the Replacement of Intervening Mechanisms Independent of SES?

If racism is a fundamental cause of health inequalities between black and white Americans, these inequalities should endure despite changes over time in diseases, risk, and protective factors and medical interventions, because mechanisms that have been reduced or eliminated are replaced by new mechanisms. In **Figure 1**, we proposed that, in the case of racism, replaceable mechanisms, independent of SES, come into play in the link between racism and racial differences in SES, the link between SES and health outcomes, and the link between racism and health outcomes. In previous publications (Phelan et al. 2004, 2010) and sections of this article, we have provided evidence for the replacement of mechanisms in the first two linkages. Here we ask whether there is evidence of the replacement of mechanisms linking racism to health outcomes, controlling for SES.

We have suggested (Link 2008, Link & Phelan 1995, Phelan et al. 2010) that new mechanisms connecting a fundamental cause to health outcomes get created when knowledge emerges about how an illness can be prevented or treated. As death from particular diseases becomes more avoidable, mortality inequalities between advantaged and disadvantaged groups should increase. This occurs because greater resources cannot be brought to bear when it is not known how to prevent death, but when they can be brought to bear, those with superior resources can use them to benefit their health and will gain a relative health advantage. According to our conceptual model (**Figure 1**), we would predict, because of the racial differences in flexible resources caused by racism, that emerging knowledge about treatment or prevention should increase racial differences in mortality. Further, owing to racial inequalities in the portion of flexible resources that is not coterminous with SES, this increase should hold controlling for SES. Two studies provide support for this hypothesis. Rubin et al. (2010) examined AIDS mortality rate ratios for black versus white Americans in the periods before, during, and after the introduction of highly active antiretroviral therapy (HAART). Because of HAART's effectiveness, fundamental-cause theory predicts increasing racial inequality in mortality across these three periods. Rubin et al. found this to be the case in analyses adjusting for age, sex, and SES and urbanicity of county of residence.

The rate ratio for black mortality compared with white mortality rose from 3.6 pre-HAART to 5.6 peri-HAART to 7.8 post-HAART. A limitation of this study is that it examined a single cause of death. Tehranifar et al. (2009) avoided this limitation. Prior to hypothesis testing, they identified cancers that are more or less amenable to treatment and examined whether racial differences in cancer-specific mortality varied according to the degree to which that cancer is amenable to available medical intervention. This study used five-year survival rates for 53 different cancer sites as a measure of effectiveness of treatment and/or early detection. Consistent with our prediction, survival disparities comparing blacks to whites were substantially greater for cancers that were more amenable to treatment (i.e., with five-year relative survival rates of 70% of more, such as bladder, breast, and prostate cancers) than for cancers that were less so (such as liver, pancreatic, and esophageal cancers), adjusting for gender, age, disease stage, and county-level poverty concentration.

In sum, consistent with the idea that new mechanisms emerge to connect a fundamental cause to health outcomes to replace old ones that have diminished or disappeared, we have provided evidence that, when knowledge emerges about prevention or treatment of a disease, a mortality advantage emerges for white Americans over black Americans and that this pattern holds controlling for SES.

CONCLUSION

Large and persistent racial inequalities in mortality across the twentieth century and into the twenty-first century give cause to expect that racism, like SES, may be a fundamental cause of health inequalities. We have attempted to evaluate this question.

We developed and provided evidence consistent with a conceptual model (**Figure 1**) that begins with systemic racism (Feagin 2000), a fundamental cause that advantages white Americans in terms of flexible resources including control of governmental and commercial institutions, disproportionate possession of socioeconomic resources at the individual level, and an ideology of white superiority (Feagin 2013) that benefits whites socially and psychologically and that justifies the more material and structural aspects of systemic racism. Systemic racism generates multiple mechanisms that produce and maintain white advantage. Consistent with fundamental causality, mechanisms have been replaced over time. At the level of structural and material discrimination, slavery and disenfranchisement were replaced by racial violence, Jim Crow segregation laws, and more recently by covert, illegal discrimination. Note that racial violence and legal discrimination and segregation persist but at more muted levels than in the past. At the ideological level, overtly expressed stereotypes and differential appraisals of the value and worth of whites and blacks have been substantially reduced but covertly expressed, and implicit stereotypes and differential appraisals of worth remain quite strong. As shown in **Figure 1**, these mechanisms result in the maintenance over time (Merton 1968) of racial differences in flexible resources of money, knowledge, power, prestige, beneficial social connections, and freedom, which in turn are connected by multiple replaceable mechanisms to racial differences in health and mortality. A subset of these resources (money, occupational power and prestige, and knowledge as embodied by formal education) strongly overlap with SES, which we have evidenced is a fundamental cause of health inequalities. Thus, we see much of the enduring association between race and health in the United States as resulting from two fundamental associations: one between systemic racism and racial inequalities in SES and a second between SES and inequalities in health outcomes.

Nevertheless, not all flexible resources associated with race overlap with SES. We found several important resources beyond the bounds of SES to differ between black and white Americans, especially nonoccupational prestige and power, freedom (control of one's own behavior and life

circumstances), and beneficial social connections associated with residential segregation. These were connected, independent of SES, to multiple health outcomes by multiple mechanisms. Notable among these mechanisms are racial discrimination as a stressor; inequalities in health care; and myriad neighborhood conditions, involving nutrition, protection and crime, exposure to harmful substances, recreational opportunities, toxic environmental exposures, and medical care, that affect blacks and whites very differently because of the extreme degree of racial residential segregation and neighborhood inequality in the United States. We conclude that the connection between race and health outcomes endures largely because racism is a fundamental cause of racial differences in SES and because SES is a fundamental cause of health inequalities, but that racism also has a fundamental association with health outcomes independent of SES.

If, as we suggest, the bulk of the effect of racism as a fundamental cause works though SES, why do we care whether racism is a fundamental cause of health inequalities independent of SES? The social and policy importance of a fundamental cause of health inequalities lie in the fact that inequalities based on a fundamental cause cannot be eliminated by addressing the mechanisms that currently link the fundamental cause to health. The fundamental cause must be addressed directly. If the distinctive features of a fundamental cause that reproduce inequalities over time characterize SES but not racism, then persistent racial differences in health should be addressed by breaking the link between race and SES. If, however, racism has the distinctive features of a fundamental cause independent of SES, then persistent racial differences in health cannot be remedied solely by reducing socioeconomic differences between black and white Americans. Even if such SES differences were eliminated, racial differences in health would persist. In this case, racial differences in health would have to be addressed like a fundamental cause, reducing racism itself. Our review suggests that such is the case.

Our findings suggest many avenues for addressing racial health inequalities in the short term: reduce racial differences in SES, in neighborhoods, in freedom, in power and prestige, in health care. Each of these is extremely important and should be vigorously pursued. At the same time, by nature, a fundamental cause is expected to replace any of these mechanisms that might be effectively blocked. We have previously identified two approaches to reducing the impact of SES as a fundamental cause (Phelan et al. 2010). One is to prioritize the development of health interventions that minimize the relevance of an individual's level of resources, such as providing health screenings in schools and other community settings rather than only through private physicians, and the other is to develop interventions, such as drugs, that are relatively affordable and easy to disseminate and use (beta-blockers) rather than those that are expensive and difficult to implement (HAART treatment for HIV) (Chang & Lauderdale 2009, Goldman & Lakdawalla 2005). Such approaches should be pursued for race-related resources as well. However, we have argued (Link & Phelan 1995) that to permanently reduce SES inequalities in health requires reducing SES inequalities themselves. By the same token, because of the reliable replacement of mechanisms linking racism to health outcomes, we conclude that racism must be the ultimate target in attempts to effectively and permanently reduce racial inequalities in health and mortality.

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