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11 The Science and Epidemiology of Racism and Health: Racial/Ethnic Categories, Biological Expressions of Racism, and the Embodiment of Inequality—an Ecosocial Perspective

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What's the use of race? The question posed by the title of this book, and the conference that gave rise to it, is deceptively simple. Its use of the singular for both *use* and *race* implies that there is only one use of—and only one entity captured by—the notion of race.

The reality, however, is far more complex, as made amply clear by the other chapters in this volume. The framing of and answer to this question—what's the use of race?—has everything to do with who is asking it, who is answering, and why.

In this chapter, I approach the question, what's the use of race? from the standpoint of a social epidemiologist who is concerned with the impact of racism on health and whose conceptual and empirical research focuses on the societal determinants of the population distribution of health, disease, and well-being (Krieger et al. 1993; Krieger 1994, 2000a, 2001, 2003, 2005a, 2008). The answer I offer is straightforward: to determine how racism harms health, it is necessary to employ the socially created categories of race/ethnicity, to distinguish between and compare the health status of the populations that are likely to be harmed by—or benefit from—racial injustice.

Three arguments that support the use of racial/ethnic categories in epidemiologic studies of racism and health are as follows:

1. Racism harms health.
2. Racism creates the very categories of race, which, in turn, demarcates groups differentially harmed and benefited by inequitable race relations.
3. Racial/ethnic health inequities are a biological expression of racism; their origins lie in injustice, not biology.

Although there may well be other questions that health researchers seek to answer about the relationship of what they define to be race to the health outcomes they find to be of interest, my twofold purpose in this chapter is to focus attention on racism and its health and to provide evidence that these health inequities are not immutable and can be changed. When categories of race/ethnicity are no longer linked to inequity, we will see it in the epidemiological data. Until then, we cannot afford to be blind to the realities and impact of racism on health.

Bean Counting, Gene Counting, and Health Inequities: On Race, Scientific Racism, and the Countervailing Claims of Antiracist Science

The legacy of scientific racism runs long and deep and is beyond the purview of this chapter (Chase 1977; Krieger et al. 1993; Gould 1996; Ernst and Harris 1999; Ewen and Ewen 2008). Its essential claims are that (1) race equals genetics; (2) "races" are natural populations, meaning that they are defined by innate genetic differences, rather than socially created demarcations; and (3) observed differences in the social standing and health status of racial groups are due to inherent, inborn factors. A recent prominent example of this orientation was provided in October 2007, when Dr. James Watson, one of the codiscoverers of the double helix structure of DNA and first director of the U.S. National Institute of Health's Human Genome Project, publicly asserted that blacks are inherently less intelligent than whites (see box 11.1; Milmo 2007).

The good news is that Watson was widely excoriated by leading scientific institutions and scientists for what he said (box 11.1; AFP 2007; Zerhouni 2007; Gumbel 2007). The criticisms impaled him for speaking scientific garbage and for attempting to use his scientific laurels as a substitute for scientific evidence. In no short order, Watson was suspended from the Cold Spring Harbor labs, where he served as chancellor, and he resigned in ignominy a few days later (BBC News 2007). As Henry Kelley, the president of the Federation of American Scientists, said, it was one "sad and revolting way to end a remarkable career" (AFP 2007).

The bad news is that the views Watson expressed are all too common, deeply ingrained, and legitimized by centuries of scientific racism (Chase 1977; Krieger et al. 1993; Gould 1996; Ernst and Harris 1999; Ewen and Ewen 2008). Hence, as part of the work required to debunk this heavy legacy, we need to counter with the science of investigating the health consequences of racism.

What are some of the testable claims of this alternative science? The first claim, also discussed in other chapters in this book, is that the long-standing simplistic equation that race equals genetics is wrong (Goodman 2000; Feldman, Lewontin, and King 2003; Krieger 2005a; Braun et al. 2007). This allegedly scientific view, articulated so baldly by Watson, racializes both biology and ancestry. Premised on the view that genes-R-us (i.e., gene counting) and that genetic differences explain why racial/ethnic groups can be differentiated and counted (i.e., bean counting), it proffers genetics as the reason why different racial/ethnic groups have different health statuses. Strong evidence shows, however, that not only can a given phenotypic trait, for example, skin color, be compatible with many different genotypes for other traits (Parra et al. 2003; Parra et al. 2004), but also that genetic variation within groups demarcated by conventional, "racial" categories is far greater than between them (Goodman 2000; Feldman, Lewontin, and King 2003). The alternative is to posit that race/ethnicity, like social class, is a historically contingent social category that imposes biological

Box 11.1

October 2007 comments by James Watson on the innate biological inferiority of black persons and the response of the scientific establishment

October 14, 2007 (not 1607, or 1707, or 1807, or 1907): *Dr. James Watson* (1962 Nobel Prize recipient for codiscovering the DNA double helix and genetic code) "told *The Sunday Times* that he was 'inherently gloomy about the prospect of Africa' because 'all our social policies are based on the fact that their intelligence is the same as ours whereas all the testing says not really.' He said there was a natural desire that all human beings should be equal but 'people who have to deal with black employees find this not true.'" (Milmo 2007)

October 18, 2007: Federation of American Scientists (FAS) president Henry Kelly: "Dr. Watson chose to use his unique stature to promote personal prejudices that are racist, vicious and unsupported by science . . . a sad and revolting way to end a remarkable career." (AFP 2007)

October 19, 2007: Watson is suspended from chancellor position at Cold Spring Harbor. (Gumbel 2007)

October 19, 2007: National Institutes of Health director Elias Zerhouni: "The comments, which were attributed to Dr. James Watson earlier this week in the London Times, are wrong, from every point of view—not the least of which is that they are completely inconsistent with the body of research literature in this area. Scientific prestige is never a substitute for knowledge. As scientists, we are outraged and saddened when science is used to perpetuate prejudice." (Zerhouni 2007)

October 25, 2007: Watson resigns from Cold Spring Harbor. (BBC News 2007)

consequences (Krieger et al. 1993; Krieger 2000a, 2001, 2003). One corollary is that gene expression, and not gene frequency, is key to generating and explaining health inequities (Krieger 2005). Another is that even in those cases where groups differ in gene frequency due to histories of geographical ancestry and migration (both forced and voluntary), the existence of such differences in no way contradicts the more general thesis that racism can harm health—no matter what the genotype at issue. Differences and inequity are not the same (Braveman and Gruskin 2003), and my concern here pertains to the excess burden of disease and death due to racial injustice.

A second claim, turning racist notions of racial categories on their head, is that there can be meaningful social categories of race/ethnicity that can be used thoughtfully to document, monitor, and analyze the impact of racial injustice on people's lives, including their health (Krieger 2000a, 2000b, 2003, 2004a; Kington and Nickens 2001; Mays et al. 2003; Smedley, Stith, and Nelson 2003; Blank, Dabady, and Citro 2004). What this statement recognizes is a profound change in the use of racial/ethnic data: from an initial purpose of discriminating adversely to, instead, providing evidence of discrimination that must be countered. Consider, for example, the case of the U.S. census, the population data of which are used for many reasons, including providing denominators to calculate population rates of disease, essential for quantifying the magnitude of racial/ethnic health disparities (Anderson 1988; Krieger 2000b). In the mid-nineteenth century, scientists lobbied for inclusion of racial categories to confirm their ideas about racial inequality (Anderson 1988; Nobles 2000). The current reason why the U.S. census obtains data on age and race/ethnicity in the 100 percent population count, however, is the recently renewed Voting Rights Act, combined with the 1990 Census Redistricting Act, whereby the collection of racial/ethnic data for the voting-age population is mandated to create fair voting districts and prevent racial gerrymandering that would dilute the vote of racial/ethnic minorities (U.S. Commission on Civil Rights 1981; U.S. Bureau of the Census 2009a). Although there still are problems, at least there are data to document the problems and contest illegitimate boundaries in court.

In other words, we cannot escape history—or pretend that it has not happened. The seemingly same categories of race have been used, at different times, and by different groups, for very different reasons: either (1) to argue for inherent racial difference and inequality, or, conversely, (2) to argue against assumptions of innate racial difference and for racial justice. Making clear that these are not simply academic points is the example of California's recently defeated Proposition 54, which, in 2003, sought to prohibit the state government from collecting or using any racial/ethnic data, including census data, allegedly on the grounds that because race is not real, biologically, data on race should not be collected (see figure 11.1; Racial Privacy Initiative 2003; Krieger 2004a). Funded by conservative groups, the actual intent of Proposition 54 was to end statistical documentation of discrimination, following a time-honored, or perhaps I should say time-disgraced, practice of getting rid of problems by removing them from view, otherwise known as "no data, no problem" (Krieger 1992). To counter, opponents argued that the social realities of race and ongoing racism require collecting the data to monitor racial/ethnic inequalities in health and other outcomes—and, indeed, the public health arguments about the need for data to document and address racial/ethnic health disparities proved decisive in helping defeat the measure (Coalition for an Informed California 2003; Schevitz 2003; Krieger 2004a).

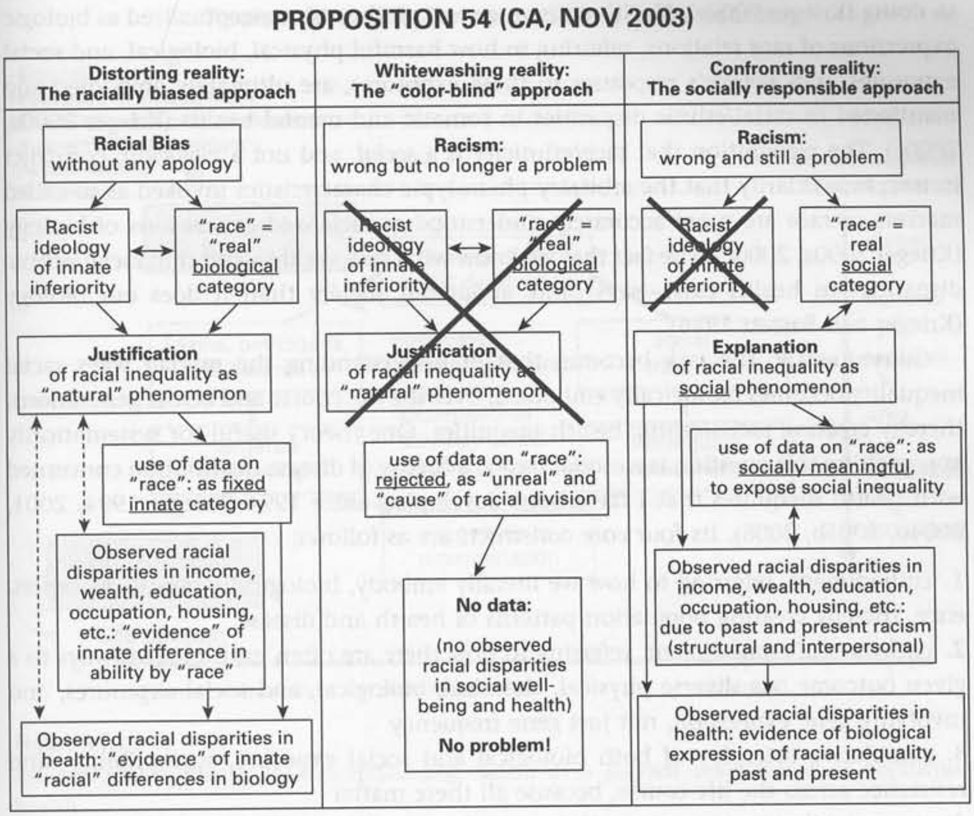

Figure 11.1

Three approaches to conceptualizing and collecting data on race/ethnicity and racial inequality, as revealed by the Proposition 54 campaign in California, 2003.

Source: Krieger (2004).

Analyzing the Impact of Racism on Health: Conceptual Issues

How, then, to conduct scientific research to test the hypothesis that racism harms health? One useful place to start is with definitions of key concepts relevant to formulating research questions and study design on through data collection, analysis, and interpretation.

Briefly stated, racism refers to institutional and individual practices that create and reinforce oppressive systems of *race relations* and their contingent *racial definitions*, whereby dominant groups define themselves and others through the possession of arbitrary physical characteristics, such as skin color, and adversely restrict, by judgment and action, the lives of those against whom they discriminate, and benefit from so doing (Krieger 2000a). Health consequences can thus be conceptualized as biologic expressions of race relations, referring to how harmful physical, biological, and social exposures, plus people's responses to these exposures, are ultimately embodied and manifested in racial/ethnic disparities in somatic and mental health (Krieger 2000a, 2000c). The recognition that race/ethnicity is a *social*, and not a *biological*, construct in turn helps clarify that the arbitrary phenotypic characteristics invoked as so-called markers of race are more accurately understood as racialized expressions of biology (Krieger 2000a, 2000c). The fact that we know what race we are—and that racial/ethnic disparities in health exist—says more about our society than it does our biology (Krieger and Bassett 1986).

Consequently, the task becomes that of understanding the myriad ways racial inequality becomes biologically embodied, over the life course and across generations, thereby creating racial/ethnic health inequities. One theory useful for systematically approaching this question is *ecosocial theory*, a theory of disease distribution concerned with health inequities that I have been developing since 1994 (Krieger 1994, 2001, 2004b, 2005b, 2008). Its four core constructs are as follows:

1. embodiment, referring to how we literally embody, biologically, our lived experience, thereby creating population patterns of health and disease
2. pathways of embodiment, referring to how there are often multiple pathways to a given outcome, via diverse physical, chemical, biological, and social exposures, and involving gene expression, not just gene frequency
3. cumulative interplay of both biological and social exposure, susceptibility, and resistance across the life course, because all these matter
4. accountability and agency, both for social inequalities in health and for ways they are—or are not—monitored, analyzed, and addressed

As emphasized by ecosocial theory, we must pay heed to context; to life course; to historical generation; to levels of analysis; to the interrelationships between diverse forms of social inequality, including racism, class, and gender, among others—and also to people's relationship to the rest of the ecosystem.

Informed by ecosocial theory, figure 11.2 accordingly lists broad categories of inequitable exposures by which racism is posited to shape population distributions of disease, taking into account domains, levels, and spatiotemporal scale (from individual life course to historical generation, and from current geographical location to histories of territorial dispossession and migration). These exposures include economic and social deprivation; toxic substances, pathogens, and hazardous conditions; social trauma; targeted marketing of harmful commodities; and inadequate and degrading medical care (Krieger 2000a). The net implication is that there can be many different ways of studying how racism harms health, depending on the type of exposure and outcome considered. No one study can ever investigate all the pathways, but any given study can at least specify which pathways it is addressing, and why (Krieger 2006).

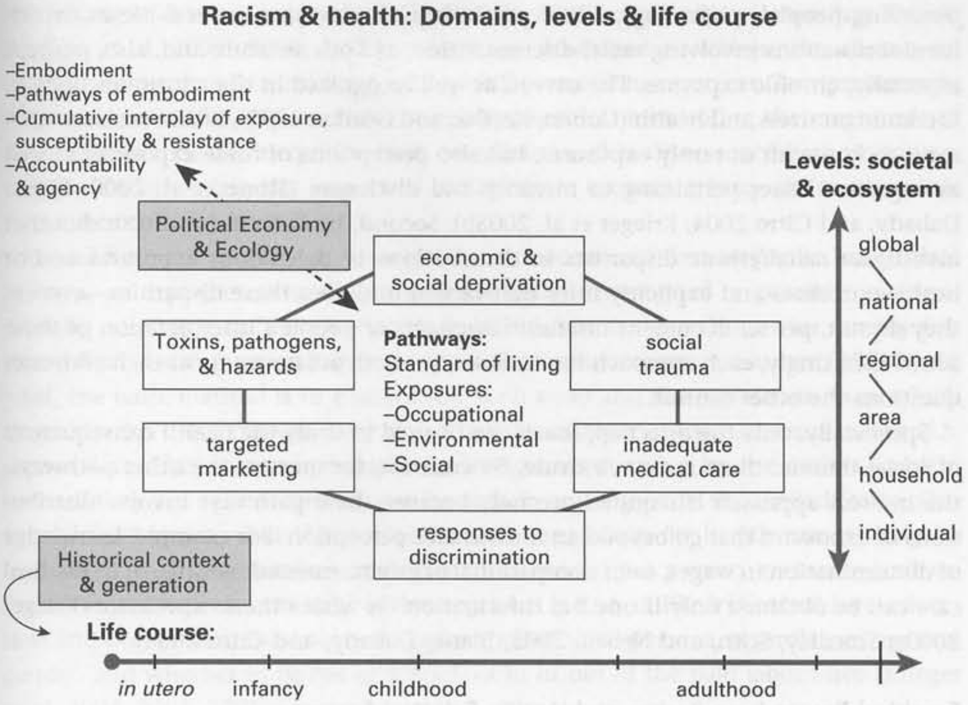

Figure 11.2

An ecosocial approach to conceptualizing racism as a societal determinant of population health.

Source: Krieger (2000a, 2008).

Moreover, once a study opts to investigate the impact of racism on health, it necessarily must use categories of race/ethnicity to demarcate the groups at risk of being harmed by racial injustice. Otherwise, there would be no exposed groups and no comparison groups and, in the case of both research studies and public health surveillance systems and medical records, no ability to document, monitor, or analyze racial/ethnic health inequities. The caveat is that the use of the racial/ethnic categories must be clearly justified and defined, in relation to both (1) how race/ethnicity is being conceptualized (e.g., as a social category reflecting societal race relations) and (2) the specified study hypotheses regarding the impact of racism on health (Krieger 2000a; Kaplan and Bennett 2003; Mays et al. 2003; Braun et al. 2007). Race/ethnicity, consequently, is an obligate, not optional, construct for research on racism and health.

As I have reviewed previously (Krieger 2000a), two kinds of studies are needed to investigate, empirically, the impact of racism on health, *direct* and *indirect*, each of which can employ quantitative or qualitative methods. First, by *direct*, I mean health studies explicitly obtaining information on people's self-reported experiences of—and observing people's physiological and psychological responses to—real-life or experimental situations involving racial discrimination, as both an acute and, also, perhaps especially, chronic exposure. The caveat, as well recognized in the enormous body of literature on stress and health (Cohen, Kessler, and Gordon 1995), is that such research must reckon with not only exposures, but also perceptions of these exposures as well as cognitive issues pertaining to memory and disclosure (Stone et al. 2000; Blank, Dabady, and Citro 2004; Krieger et al. 2005b). Second, by *indirect*, I mean studies that investigate racial/ethnic disparities in distributions of deleterious exposures and/or health outcomes and explicitly infer that racism underlies these disparities—even as they do not, per se, document discriminatory acts or people's interpretation of these acts. Taken singly, each approach has its flaws, yet both are necessary as each addresses questions the other cannot.

Specifically, only the direct approach can be used to study the health consequences of social trauma; there is no substitute. By contrast, for most of the other pathways, the indirect approach is required precisely because these pathways involve distributions of exposures that go beyond an individual's perception. For example, knowledge of discrimination in wages, or in occupational or environmental hazards, or in medical care can be obtained only if one has information on what others experience (Krieger 2000a; Smedley, Stith, and Nelson 2003; Blank, Dabady, and Citro 2004).

Empirical Research on Racism and Health: Selected Examples

The number of studies explicitly testing hypotheses about the impact of racial discrimination on health is burgeoning (Krieger 2000a; Williams, Neighbors, and Jackson 2003; Paradies 2006; Mays, Cochran, and Barnes 2007; Kressin, Raymond, and Manze 2008; Williams and Mohammed 2009). In 1999, when I wrote the first epidemiologic review article on the topic, I could identify only fifteen relevant studies in the broader public health literature (Krieger 2000a). By contrast, an epidemiologic review article published in 2006, which focused on articles published between 2000 and 2004, was able to include 138 articles (Paradies 2006), and a new 2009 review article has identified 115 articles, listed in the PubMed database and published solely between 2005 and 2007, that investigated associations between self-reported experiences of racial discrimination and either health status or health care utilization (Williams and Mohammed 2009). Thus my purpose in this section is not to review this large and growing literature, but instead, to illustrate some of the approaches being used, using examples of my own research.

Indirect Approach: Racism, Class, and Health Inequities

I start with two studies that employed the indirect approach; each was concerned with links between racial/ethnic inequality, socioeconomic deprivation, and health. The first example draws on data from my Public Health Disparities Geocoding Project (Krieger et al. 2003, 2004, 2005a), which we developed to address the absence of socioeconomic data in most U.S. public health surveillance systems. Two problematic consequences of this gap are that it precludes both (1) monitoring the magnitude of socioeconomic health inequities, both overall and within diverse racial/ethnic groups, and (2) documenting the impact of racial/ethnic economic inequalities on racial/ethnic health inequities.

To address the problem of nonexistent socioeconomic data in public health data systems, our project accordingly systematically investigated a possible solution, drawing on an approach used eclectically in U.S. health research for over seventy-five years: that of using geocoding and area-based socioeconomic measures (ABSMs). In brief, the basic method is to characterize both cases and the population from which they arise by the socioeconomic characteristics of their residential areas, using U.S. census data. This, in turn, permits calculating rates stratified by the ABSMs—which, because they are census derived, can be used in any region in the United States. Our central finding was that U.S. socioeconomic inequalities in health can suitably be monitored with the common metric of the census tract poverty measure—and we further note that one advantage of this approach is that, unlike individual-level education and occupation, this measure can be applied to all persons, regardless of age and gender, and whether in or out of school, or in or out of the paid labor force (Krieger et al. 2003, 2004, 2005a).

Table 11.1 presents the impact of adjusting for census tract poverty on black-white and Hispanic-white age-adjusted health disparities for a variety of outcomes, using data from my home state of Massachusetts, centered around the U.S. 1990 census (Krieger et al. 2004, 2005a). The first point to note is that the magnitude of racial/ethnic health inequities varied by race/ethnicity, by outcome, and by gender. African Americans, for example, generally were at excess risk compared to white Americans across the board, with relative risks ranging from around 2 (e.g., premature mortality) to upward of 20 (gonorrhea and tuberculosis). By contrast, for Hispanics, the picture was more variable; Hispanics were often at higher risk than whites for the childhood and infectious disease outcomes, but at lower risk for premature mortality (possibly a reflection of immigrants returning to their home countries to die). In both groups, the racial/ethnic disparity for deaths due to HIV/AIDS was far greater for women compared to men.

Nevertheless, the second major point is that even with this heterogeneity of risk, what table 11.1 reveals is that adjusting for census tract poverty, in virtually every case, reduced the observed racial/ethnic disparities, as shown by the shaded cells. In some cases, the reduction was quite dramatic, as shown by the outcomes in boldface, for example, nearly a halving of the excess risk, if not more, for childhood lead poisoning, gonorrhea, tuberculosis, and for mortality due to HIV/AIDS and to homicide

Table 11.1

Effect of adjusting for socioeconomic position on racial/ethnic health inequities: Results of the Public Health Disparities Geocoding Project for Massachusetts and Rhode Island, circa 1990

		Black-white				Hispanic-white			
		Women		Men		Women		Men	
Outcome	Adjusted for	RR (95% CI)		RR (95% CI)		RR (95% CI)		RR (95% CI)	
% Low birth weight	Age	2.8	(2.6, 3.0)	2.5	(2.3, 2.7)	1.5	(1.4, 1.6)	1.5	(1.4, 1.7)
	Age + census tract poverty	2.3	(2.1, 2.5)	2.0	(1.8, 2.2)	1.2	(1.1, 1.3)	1.2	(1.1, 1.3)
Childhood lead poisoning	Age	**6.5**	**(5.6, 7.6)**	**5.4**	**(4.7, 6.3)**	**4.0**	**(3.5, 4.5)**	**3.4**	**(3.0, 3.8)**
	Age + census tract poverty	**2.4**	**(2.0, 2.9)**	**2.1**	**(1.8, 2.6)**	**1.3**	**(1.1, 1.6)**	**1.2**	**(1.1, 1.4)**
Gonorrhea	Age	**22.0**	**(19.9, 24.2)**	**21.3**	**(19.6, 23.2)**	**10.7**	**(9.5, 12.0)**	**10.5**	**(9.4, 11.6)**
	Age + census tract poverty	**14.0**	**(12.6, 15.7)**	**13.0**	**(11.8, 14.3)**	**6.2**	**(5.4, 7.1)**	**5.9**	**(5.2, 6.6)**
Tuberculosis	Age	**20.4**	**(16.5, 25.3)**	**13.9**	**(11.8, 16.4)**	**19.8**	**(14.6, 26.9)**	**15.1**	**(12.1, 18.7)**
	Age + census tract poverty	**13.8**	**(10.7, 17.8)**	**6.9**	**(5.7, 8.3)**	**15.1**	**(10.5, 21.7)**	**8.0**	**(6.3, 10.3)**
Premature mortality	Age	1.9	(1.8, 2.0)	2.0	(1.9, 2.1)	0.9	(0.9, 1.0)	1.2	(1.1, 1.3)
	Age + census tract poverty	1.5	(1.4, 1.6)	1.4	(1.3, 1.5)	0.7	(0.7, 0.8)	0.9	(0.8, 0.9)
Diabetes mortality	Age	2.0	(1.6, 2.4)	1.5	(1.2, 1.9)	1.0	(0.7, 1.5)	0.9	(0.6, 1.4)
	Age + census tract poverty	1.7	(1.3, 2.0)	1.4	(1.1, 1.8)	0.9	(0.6, 1.3)	0.8	(0.5, 1.3)
HIV/AIDS mortality	Age	**17.3**	**(12.6, 23.6)**	**4.3**	**(3.7, 5.0)**	**8.9**	**(5.9, 13.2)**	**2.9**	**(2.4, 3.5)**
	Age + census tract poverty	**9.3**	**(6.4, 13.5)**	**2.2**	**(1.9, 2.6)**	**4.4**	**(2.7, 7.0)**	**1.5**	**(1.2, 1.8)**
Homicide and legal intervention	Age	**9.3**	**(6.5, 13.2)**	**19.6**	**(16.2, 23.7)**	**3.9**	**(2.3, 6.5)**	**9.4**	**(7.4, 12.0)**
	Age + census tract poverty	**5.6**	**(3.7, 8.4)**	**10.8**	**(8.7, 13.4)**	**2.3**	**(1.3, 4.0)**	**5.1**	**(3.9, 6.7)**

Note: Shaded rows show outcomes for which adjusting for census tract poverty reduced the magnitude of racial/ethnic health inequities; data in boldface are for outcomes for which there was effectively a halving or more of risk. CI = confidence interval. RR = relative risk.

Source: Krieger et al. (2004, 2005a).

and legal intervention. If these are the results we obtained with just this one admittedly crude measure of socioeconomic position, measured at the time of the health outcome, it is highly likely that the magnitude of racial/ethnic health inequities would have been further reduced had we been able to use more comprehensive measures of socioeconomic position, across domains and across the life course (Krieger, Williams, and Moss 1997; Shaw et al. 2007).

A third major point is that our results are complex, which is likewise the message of the extant literature on the impact of adjusting for or stratifying by socioeconomic position when analyzing racial/ethnic disparities in health (Krieger et al. 1993; Krieger 2000a; Kington and Nickens 2001; Williams and Jackson 2005). In other words, there is no one-size-fits-all scenario. That said, it is safe to say that more often than not, racial/ethnic socioeconomic inequities do play a major role in racial/ethnic health inequities, both in the onset of the event and once disease is diagnosed. Moreover, within the U.S. context, the continued salience of poverty for racial/ethnic health inequities remains large. Data from the U.S. Current Population Survey (U.S. Bureau of the Census and U.S. Bureau of Labor Statistics 2007), for example, documented that in 2006, fully 12.3 percent of the U.S. population, including 20.7 percent of children under age five—that is, one in every five children—lived under the notoriously stingy U.S. poverty line (O'Connor 2001). This is a very high level of poverty, and especially childhood poverty, compared to other industrialized nations (UNICEF 2005). Only then consider the magnitude of the racial/ethnic inequities in poverty identified in this survey, which translated to fully four in ten black children and three in ten Hispanic children, versus less than one in ten white children, growing up impoverished. At a time when ever more research shows the profoundly devastating effects of childhood poverty not only on children, but also on their later health status as adults (Krieger 2006; Shaw et al. 2007), it is obvious that research on health inequities must reckon with huge socioeconomic disparities between U.S. racial/ethnic groups. Equally obvious, it is only through the use of racial/ethnic categories that it becomes possible to see the patterning of racial/ethnic socioeconomic inequities and their implications for racial/ethnic health inequities.

The second example concerns long-term trends in U.S. racial/ethnic and socioeconomic health inequities. Motivating our study (Krieger et al. 2008b) were articles in the current literature arguing that racial/ethnic and socioeconomic health disparities are, in effect, inevitable and likely to increase because the more educated and wealthier will always be most able to take advantage of the latest health knowledge and medical innovations (Phelan and Link 2005; Mechanic 2005; Cutler, Deaton, and Lleras-Muney 2006). But is this necessarily the case? Or might the magnitude of these health inequities be historically contingent and reflect, in part, societal priorities—and hence be amenable to social change?

To address this question, we decided to examine, empirically, long-term trends in socioeconomic inequities in U.S. premature mortality and infant death rates, overall and by race/ethnicity (Krieger 2008b). Prior to our study, research on this topic had been hampered by the absence of socioeconomic data in U.S. death and birth certificates until 1989 and 1968, respectively—and also because the public access version of the U.S. Compressed Mortality Files only goes back to 1968 (National Center for Health Statistics 2009a). Arguably, however, from a policy and public health perspective, the period directly preceding 1968 is crucial. Critical changes included the 1964 Civil Rights Act, the 1965 establishment of Medicare and Medicaid and the consequent desegregation of U.S. medical facilities, the accompanying expansion of community health centers and maternal and child health programs, and the many other federal policies comprising what was then called the "War on Poverty" (Davis and Schoen 1978; O'Connor 2001; Fairclough 2001; Quadagno and McDonald 2003; Almond, Chay, and Greenstone 2006; Lefkowitz 2007). These policies, intended to counter structural racism, could plausibly be conceptualized as societal determinants of health, whose embodied consequences would be manifest in trends in U.S. health inequities.

We accordingly sought out data to analyze rates of premature mortality and infant death among U.S. counties, ranked by income level, for the period 1960–2002, for both the total population and also stratified by what W. E. B. Du Bois (1904/2004) famously termed the U.S. "colorline," which divides the racially dominant U.S. white population and U.S. populations of color. We decided that this dichotomy—between whites and people of color—was the best way to handle the limitations of the available racial/ethnic mortality data that extended back to 1960, which used only the categories of white, black, and other. We chose age sixty-five as the cutoff point for premature mortality because this age determines eligibility for Social Security and Medicare and also because an average life expectancy of at least sixty-five years was consistently attained by U.S. black men only in 1995—compared to 1944 for the total U.S. and white population and 1973 for the black population overall (National Center for Health Statistics 2009b).

To create our study database, we extracted the 1968–2002 mortality data from the public-use U.S. Compressed Mortality Files, and then we also obtained U.S. county 1960–1967 mortality data, which additionally required manually locating and identifying the correct county code for each of the 3,073 counties. Denominators consisted of U.S. census decennial counts and intercensal estimates. With regard to socioeconomic data, because of the loss of the computerized 1960 census "100 percent detail" file, the economic measure we employed was county median family income, chosen because we were able to locate this for the 1960 as well as the 1970–2000 census. We assigned counties to quintiles of median family income weighted by county population size and then calculated, for each calendar year, each quintile's aggregated age-standardized premature mortality rate (deaths before age sixty-five, using the year 2000 standard million) and infant death rate (deaths among persons under age one).

We then tested our hypotheses about trends in the socioeconomic inequities in premature mortality and infant death in several ways, using measures of relative risk, absolute difference, changes in slope, and excess fraction of premature deaths. Our a priori hypothesis was that inflection points—that is, changes in the rate of decline (slope) of the premature mortality and infant death rates—would occur in 1965 and 1980, given major federal policy changes during the Kennedy/Johnson and Reagan administrations.

As we reported in our study findings, and as shown in tables 11.2a and 11.2b and figure 11.3, the key results were that between 1960 and 2002, in the United States overall, even as premature mortality (table 11.2a) and infant death (table 11.2b) rates declined in all county income quintiles, the gap between the lowest- and highest-income quintiles persisted and was relatively greatest for premature mortality in 2000, and barely changed for infant deaths. The greatest progress in reducing these income gaps occurred between 1965 and 1980; thereafter the health inequities widened. Lending further support to our a priori hypothesis, additional analyses showed that the average annual percentage change in premature mortality rates dropped to less than half that of the preceding period for all socioeconomic-racial/ethnic strata—except for the white population living in the two highest county income quintiles, whose rate of decline stayed the same or increased. The population impact of these trends is highlighted by our finding that between 1960 and 2000, 18 percent of premature deaths would have been averted had the populations in the bottom four quintiles experienced the same yearly, age-specific, premature mortality rates as the highest quintile. This excess fraction translates to an estimated 4.9 million lives cut short.

The overall picture, however, obscures stark racial/ethnic disparities within and across income quintiles. As additionally revealed by tables 11.2a and 11.2b, the steep decline in rates in the 1965–1980 period was especially notable in the populations of color, and especially in the two lowest income quintiles. The combined impact of class and color is further underscored by our results showing that between 1960 and 2002, had everyone experienced the same yearly age-specific mortality rates as whites in the highest-income county quintile, then 14 percent of white premature deaths, yet fully 30 percent of the premature deaths among populations of color, would have been averted.

At one level, our finding that risk of premature mortality increased with economic deprivation and racial inequality obviously is not new; research documenting these social facts easily extends back to the late eighteenth century (Rosen 1958/1993; Krieger 2000d). That said, what our results newly underscore is that contemporary U.S. inequities are not immutable: they shrunk considerably between 1965 and 1980, and increased or stagnated thereafter. The early trends give grounds for hope; the latter augur poorly for the Healthy People 2010 objective of eliminating U.S. socioeconomic and racial/ethnic health disparities.

Table 11.2a

U.S. premature mortality rates (death before age sixty-five) per one hundred thousand, 1960–2002

		Premature mortality rate by year (95% CI)					Average change in rate per year		
Race/ethnicity	Income quintile	1960	1970	1980	1990	2000	1960–1965	1966–1980	1981–2002
Total	Q1	377 (375, 380)	417 (414, 419)	335 (333, 337)	291 (289, 292)	268 (267, 270)	**12.6**	**–6.2**	**–3.0**
	Q2	426 (423, 428)	398 (396, 400)	310 (308, 312)	265 (263, 267)	224 (222, 225)	**3.0**	**–8.4**	**–4.0**
	Q3	404 (401, 406)	368 (365, 370)	284 (283, 286)	271 (270, 273)	225 (223, 226)	**–0.7**	**–7.6**	**–2.5**
	Q4	384 (381, 386)	371 (369, 373)	296 (294, 298)	252 (250, 253)	200 (199, 202)	**3.1**	**–6.2**	**–4.7**
	Q5	342 (340, 344)	311 (309, 313)	250 (249, 252)	204 (203, 206)	163 (162, 164)	**–1.8**	**–5.1**	**–4.3**
White	Q1	318 (316, 320)	367 (364, 369)	297 (295, 299)	261 (259, 263)	244 (243, 246)	**13.0**	**–4.9**	**–2.6**
	Q2	379 (376, 381)	356 (353, 358)	279 (277, 281)	239 (237, 241)	210 (208, 211)	**2.7**	**–7.3**	**–3.2**
	Q3	377 (375, 380)	341 (339, 344)	268 (266, 270)	239 (238, 241)	204 (203, 206)	**–0.7**	**–7.0**	**–2.8**
	Q4	366 (363, 368)	338 (336, 340)	271 (269, 273)	230 (228, 231)	182 (181, 184)	**0.6**	**–6.1**	**–4.4**
	Q5	322 (319, 324)	298 (296, 300)	241 (239, 243)	195 (194, 197)	157 (155, 158)	**–1.3**	**–4.4**	**–4.3**
Of color	Q1	672 (664, 680)	691 (683, 699)	518 (512, 524)	460 (454, 466)	371 (366, 375)	**15.9**	**–14.1**	**–6.3**
	Q2	774 (764, 784)	699 (690, 708)	530 (523, 538)	431 (425, 437)	298 (294, 303)	**–0.3**	**–15.6**	**–10.6**
	Q3	701 (689, 713)	637 (627, 647)	450 (442, 458)	441 (435, 447)	318 (314, 322)	**0.3**	**–15.6**	**–6.2**
	Q4	592 (581, 603)	611 (603, 620)	437 (431, 444)	360 (355, 365)	294 (289, 298)	**16.0**	**–13.8**	**–6.5**
	Q5	579 (568, 590)	476 (466, 487)	336 (329, 343)	262 (257, 267)	194 (191, 198)	**–4.4**	**–15.1**	**–6.3**

Note: Premature mortality rates were age-standardized to the year 2000 standard million. Changes in rate per year in boldface are statistically significant at the $p < 0.05$ level. Q1 = lowest county family income quintile; Q5 = highest county family income quintile.

Source: Krieger et al. (2008b).

Table 11.2b

U.S. infant death rates (per one thousand persons under age one), 1960–2002

Race/ethnicity	Income quintile	Premature mortality rate by year (95% CI) 1960	1970	1980	1990	2000	Average change in rate per year 1960–1965	1966–1980	1981–2002
Total	Q1	29 (29, 30)	26 (25, 26)	15 (15, 16)	10 (10, 11)	9 (8, 9)	**–0.2**	**–1.0**	**–0.3**
	Q2	30 (29, 30)	23 (23, 24)	14 (14, 15)	10 (10, 10)	7 (7, 8)	**–0.9**	**–0.9**	**–0.3**
	Q3	28 (27, 28)	21 (21, 21)	13 (13, 14)	10 (10, 11)	8 (7, 8)	**–1.1**	**–0.7**	**–0.2**
	Q4	26 (25, 26)	21 (21, 22)	14 (14, 15)	10 (10, 10)	7 (7, 8)	**–0.8**	**–0.6**	**–0.3**
	Q5	22 (22, 22)	17 (17, 17)	12 (11, 12)	8 (7, 8)	6 (5, 6)	**–0.9**	**–0.5**	**–0.3**
White	Q1	23 (23, 23)	22 (21, 22)	13 (12, 13)	9 (8, 9)	7 (7, 7)	**0.0**	**–0.8**	**–0.2**
	Q2	26 (25, 26)	20 (20, 21)	12 (12, 13)	8 (8, 9)	6 (6, 6)	**–0.7**	**–0.8**	**–0.2**
	Q3	25 (25, 26)	19 (19, 19)	12 (12, 12)	8 (8, 8)	6 (6, 6)	**–1.0**	**–0.7**	**–0.2**
	Q4	24 (24, 24)	19 (18, 19)	12 (12, 12)	8 (8, 8)	6 (6, 6)	**–1.0**	**–0.6**	**–0.3**
	Q5	20 (20, 20)	15 (15, 16)	10 (10, 11)	7 (6, 7)	5 (5, 5)	**–0.8**	**–0.4**	**–0.3**
Of color	Q1	49 (48, 50)	39 (38, 40)	24 (23, 25)	16 (16, 17)	13 (12, 13)	**–0.5**	**–1.6**	**–0.5**
	Q2	51 (50, 52)	37 (35, 38)	24 (23, 25)	17 (17, 18)	12 (12, 13)	**–2.3**	**–1.3**	**–0.5**
	Q3	45 (44, 47)	36 (35, 37)	23 (22, 24)	18 (17, 19)	12 (12, 13)	**–1.2**	**–1.2**	**–0.5**
	Q4	40 (39, 41)	36 (35, 37)	25 (24, 25)	16 (16, 17)	13 (12, 13)	**–0.3**	**–1.0**	**–0.5**
	Q5	40 (38, 41)	31 (30, 33)	19 (18, 20)	13 (12, 14)	9 (8, 9)	**–1.0**	**–1.2**	**–0.4**

Note: Changes in rate per year in boldface are statistically significant at the $p < 0.05$ level. Q1 = lowest county family income quintile; Q5 = highest county family income quintile.

Source: Krieger et al. (2008b).

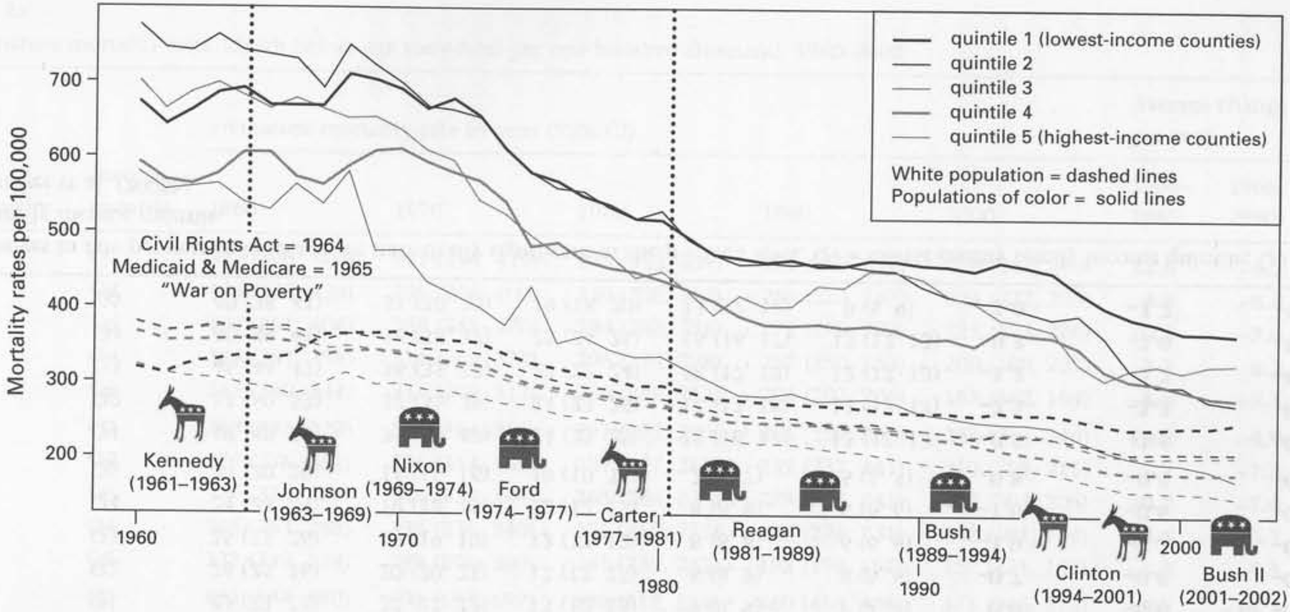

Figure 11.3

U.S. trends in racial/ethnic and socioeconomic inequities in premature mortality, 1960–2002: Data and interpretation.

Source: Krieger et al. (2008b; political events and symbols added).

In our published study, we exhaustively consider competing hypotheses that might explain our findings (Krieger et al. 2008b). Here, I summarize them, and I start by noting that the finding of a declining—then increasing—gap is unlikely to be an artifact of inaccurate numerator or denominator data. Since 1960, 99 percent of all U.S. deaths and births have been registered (Hetzel 1997). The U.S. census undercount (disproportionately affecting lower-income populations and populations of color) has declined considerably (e.g., for blacks, from 6.6% in 1960 to 2.8% in 2000), a trend that would increasingly reduce, not inflate, estimates of social disparities in mortality (Clark and Moul 2004). Results are also unlikely to be affected by racial/ethnic misclassification, given the broad groupings employed; rather, demographic trends should have lowered risk of premature mortality among U.S. populations of color, given the increase in foreign-born U.S. populations of color and their associated "healthy immigrant" effect, and the corresponding proportional decline in the U.S. African American population (from 92% of U.S. populations of color in 1960 to 72% in 2000; Singh and Siapush 2001; U.S. Bureau of the Census 2009b).

What, then, might explain the observed trends? First, the rising U.S. per capita gross domestic product (GDP) likely contributed to the overall decline in premature mortality rates (e.g., GDP grew by 32% during 1961–1970, 23% during 1970–1980, 25% during 1980–1990, and 22% during 1990–2000; Centre for the Study of Living Standards 2009). But this rising GDP cannot explain the observed pattern of a diminishment and then increase in the socioeconomic gradient. Nor can the observed trends be explained simply by relative positioning in a social hierarchy, a hypothesis some have proposed to explain social gradients in health (Phelan and Link 2005; Wilkinson 2005; Adler and Rehkopf 2008), because such an explanation would not account for either the overall falling rates of premature mortality or the shrinking and then widening of the gap. It is also unlikely that purely individual-level behavioral factors can explain the faster and then slower decline in premature mortality among persons in the lower income quintiles, unless an argument can be made that health promotion efforts in this group were more successful in the earlier, rather than later, time period, which is unlikely.

As an alternative explanation, the most plausible one involves two likely major societal determinants of health: economic priorities and civil rights (Krieger et al. 2008b). Likely contributing to the 1965–1980 improvements are the positive impact of the "War on Poverty" and the civil rights legislation that expanded economic opportunity and resources, and also availability of health services, for both the poor and populations of color, especially African Americans (Davis and Schoen 1978; O'Connor 2001; Fairclough 2001; Quadagno and McDonald 2003; Almond, Chay, and Greenstone 2006; Lefkowitz 2007). Conversely, the subsequent slowdown likely reflects the adverse impact of post-1980 neoliberal and neoconservative policies to roll back the welfare state (O'Connor 2001; Auerbach, Card, and Quigley 2006; Navarro

2007). Here, I mean cutting federal responsibility and funds for public health and antipoverty programs, opposing affirmative action, blocking rises in the minimum wage, and selectively decreasing taxes on the wealthy, coupled with rising medical uninsurance and persistent racial/ethnic disparities in quality of care (O'Connor 2001; Smedley, Stith, and Nelson 2003; Auerbach, Card, and Quigley 2006; Navarro 2007).

To summarize, our results refute the view that widening health disparities necessarily accompany improvements in population health. Death is inevitable; premature mortality is not. One potentially hopeful hypothesis accordingly prompted by our findings is that if addressing social injustice and its embodied health consequences becomes a priority, we can make progress. Another implication is that we need to understand not only how class inequalities harm health within each and every racial/ethnic group, but also how racial inequality harms health, including within and across class strata. As our data illustrate, the point is not race versus class—both matter, and we need data on both. The implication is that we need more than simply indirect studies to investigate how racism harms health: it is also necessary to consider the direct impact of racial discrimination on health, including within and across socioeconomic strata.

Direct Approach: Self-Reported Experiences of Racial Discrimination and Health

The next set of examples accordingly focuses on the inclusion of data on self-reported experiences of racial discrimination in public health research. I begin with a description of an instrument to measure exposure to racial discrimination and then discuss its application in two studies.

The Experiences of Discrimination (EOD) instrument (see table 11.3), which we validated (in both English and Spanish) in 2005 (Krieger et al. 2005b), is based on an instrument I first developed in 1990 (Krieger 1990; Krieger and Sidney 1996) and have used with slight modification since. It is among the two most widely used self-report measures in empirical studies on self-reported experiences of racial discrimination and health (Krieger 2000a; Paradies 2006). The approach of the EOD is to ask participants, explicitly, about whether they have "experienced discrimination, been prevented from doing something, or made to feel inferior in any of the following situations because of your race, ethnicity or color." The nine situations then listed each pertain to commonly identified domains in which people report having experienced racial discrimination, that is, involving education, work, housing, health care, public settings, and the police. Respondents who answer yes to any given item are then asked a follow-up question about frequency. Additional questions ask about response to unfair treatment. As shown in the validation study, which was conducted among a cohort of low-income workers in the greater Boston area, the EOD has a high Cronbach's alpha (0.8 or higher among black Americans and Latinos), high test-retest reliability (over 0.7), and no response item bias; moreover, confirmatory factor analysis indicated that there was an acceptable fit to the data for a single underlying factor (Krieger et al. 2005b). Also of note, we included several single-item measures of experiences of discrimination in the validation study, all of which we found had very low test-retest reliability (correlations of 0.4 or less), a result that cautions against using single-item questions about exposure to racial discrimination, as currently used, for example, in the U.S. Behavioral Risk Factor Surveillance Survey (National Center for Chronic Disease Prevention and Health Promotion 2009).

Relevant to the question, what's the use of race? in the validation study, all three racial/ethnic groups reported having experienced racial discrimination, albeit at different frequencies. Thus 38 percent and 24 percent of the black and Latino participants reported having experienced discrimination in three or more of the nine situations, versus only 12 percent of the white participants; a similar difference in magnitude was evident for the frequency score (Krieger et al. 2005b). Moreover, self-reports of racial discrimination were associated with the odds of having smoked cigarettes only among the black and Latino workers, but not among the white workers; self-reports also tended to be associated with the risk of being psychologically distressed among the black workers and, to a lesser extent, the Latino workers, but once again, not the white workers. These findings, along with the literature on "reverse discrimination"(Pincus 2003; Bonilla-Silva 2003; Crosby 2004), underscores a key problem with the suggestions offered by some researchers to "abandon" (Fullilove 1998) race as a variable in health research and to instead use only measures pertaining to racial discrimination (Fullilove 1998) or other social measures (e.g., socioeconomic position; Bhopal and Donaldson 1998; Stolley 1999): without data on race/ethnicity, it becomes impossible to offer meaningful interpretation of the self-report data on racial discrimination or to analyze if it is similarly or differentially associated with health status across all racial/ethnic groups. Thus, as increasingly recognized in the literature distinguishing between the use of race/ethnicity as a descriptive versus ascriptive category (Kaplan and Bennett 2003; Braun et al. 2007), studies that ask about self-reported experiences of discrimination for the purpose of testing causal hypotheses necessarily must also employ appropriate descriptive racial/ethnic categories.

Also important to note is evidence indicating that people with fewer resources tend to report less racial discrimination compared to those with more resources. In the case of the EOD, for example, self-reports of having ever experienced racial discrimination were notably higher, compared to self-reports by the low-income, working-class participants in the EOD validation study (Krieger et al. 2005b), in three investigations with more affluent populations: the proportion was nearly one and a half times higher in the CARDIA study (52%), a multicity, population-based, longitudinal investigation concerned with black-white differences in risk of cardiovascular disease (Krieger and Sidney 1996), and over two times higher (80%) in both a study of pregnant women with health insurance (Dominguez et al., 2009) and a Web-based study in which 60

Table 11.3
Experiences of Discrimination instrument

Measure	Question	Stem
English version		
Experience of discrimination	Introduction: "This next section is going to ask about how you and others like you are treated, and how you typically respond."	
Response to unfair treatment	If you feel you have been treated unfairly, do you usually: (please select the best response)	1. Accept it as a fact of life 2. Try to do something about it
	If you have been treated unfairly, do you usually: (please select the best response)	1. Talk to other people about it 2. Keep it to yourself
Discrimination	Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior in any of the following situations because of your race, ethnicity, or color? 1. At school? 2. Getting hired or getting a job? 3. At work? 4. Getting housing? 5. Getting medical care? 6. Getting service in a store or restaurant? 7. Getting credit, bank loans, or a mortgage? 8. On the street or in a public setting? 9. From the police or in the courts?	For each situation to which the participant replied yes (vs. no), the follow-up question was, How many times did this happen? 1. Once 2. Two or three times 3. Four or more times
Spanish version		
Experience of discrimination	Introduction: "En esta sección se le preguntará acerca de cómo usted, y otros como usted, son tratados, y cómo usted responde típicamente."	
Response to unfair treatment	En caso de sentir que ha sido tratado de manera injusta, usted normalmente: (por favor elija la mejor respuesta)	1. Lo toma como un hecho de su vida 2. Trata de hacer algo al respecto
	Si usted ha sido tratado injustamente, usted normalmente: (por favor elija la mejor respuesta)	1. Habla acerca de esto con otras personas 2. Se lo guarda para sí mismo

Table 11.3
(continued)

Measure	Question	Stem
Discrimination	¿Alguna vez ha experimentado discriminación, no se le ha permitido hacer algo, se le ha molestado o hecho sentir inferior en alguna de las siguientes situaciones debido a su raza, etnia o color? 1. ¿En la escuela? 2. ¿Al ser contratado u obtener un empleo? 3. ¿En el trabajo? 4. ¿Al obtener una casa? 5. ¿Al obtener asistencia médica? 6. ¿El requerir servicio en una tienda o restaurante? 7. ¿Al obtener crédito, préstamos bancarios o hipotecarios? 8. ¿En la calle, en un lugar público? 9. ¿De la policía o en las cortes?	For each situation to which the participant replied *sí* (vs. *no*), the follow-up question was, ¿Cuántas veces ocurrió esto? 1. Una vez 2. Dos o tres veces 3. Cuatro o más veces

Note: The Experiences of Discrimination instrument and instructions for its use are freely available online (http://www.hsph.harvard.edu/faculty/nancy-krieger/).
Source: Krieger et al. (2005b).

percent of the respondents had a bachelor's degree or higher (Krieger et al., in press). The positive association between socioeconomic resources and self-reports of racial discrimination is a robust finding (Krieger 2000a; Paradies 2006; Williams and Mohammed 2009). One possible explanation is that people with more power and resources may be more able and willing to identify experiences of racial discrimination (Krieger 1990; Krieger and Sidney 1996; Krieger 2000a; Blank, Dabady, and Citro 2004). An important implication is that research on racial discrimination cannot be concerned only about race/ethnicity, but must also take into account socioeconomic position.

Demonstrating that it does make a difference, empirically, to include data on self-reported experiences of racial discrimination in epidemiologic research are results of a CARDIA study on racial discrimination and risk of preterm delivery, in which I was a coinvestigator (Mustillo et al. 2004). Motivated by the unanswered question of what accounts for higher rates among U.S. black women, as compared to white women, of preterm delivery—a major determinant of low birth weight and infant mortality (Krieger et al. 1993; Schempf et al. 2007)—in this study, we sought to assess whether including data on self-reported experiences of racial discrimination, in addition to major known conventional risk factors, would affect the magnitude of the observed black-white disparities.

The study population consisted of 367 women who gave birth between the year 7 and year 10 CARDIA exams (Mustillo et al. 2004). From the year 7 exam, we obtained prospective data on self-reported experiences of racial discrimination (using a prior version of the EOD), socioeconomic position, and other relevant baseline characteristics; from the year 10 exam, we obtained data on birth outcomes. The four key results were as follows (see table 11.4):

1. As shown in model 1, overall, the odds of black women having a preterm delivery were two and a half times higher as compared to white women.
2. When we additionally added data on self-reported experiences of racial discrimination, we found that the black excess risk was reduced to 1.7, and also that increased exposure to racial discrimination was associated with a twofold higher odds of risk of preterm delivery (model 2).
3. In analyses that controlled for other major risk factors for preterm delivery, such as income, education, smoking, alcohol, and depression (model 3), we found that adjusting for these risk factors somewhat reduced the initially observed black-white difference (model 1), but the odds of black women having a preterm delivery were still about two times more than compared to white women.
4. Last, when we additionally included the data on self-reported experiences of racial discrimination (model 4), we found that there was no longer any racial/ethnic difference in risk: the odds of black women and white women having a preterm delivery *were the same* (model 4). We also found that, as compared to women reporting no racial discrimination, the odds of having a preterm delivery were two times higher among women who reported experiencing moderate racial discrimination (in one or two situations) and three times higher among those reporting high exposure (in three or more situations).

Table 11.4

Self-reported experiences of racial discrimination and risk of preterm delivery: The CARDIA study (1992–1995)

	Model: Odds ratio (95% CI)			
Characteristic	1	2	3[a]	4[a]
Black vs. white	2.5 (1.3, 4.8)	1.7 (0.8, 3.5)	1.9 (0.8, 4.1)	1.1 (0.5, 2.4)
Racial discrimination				
1–2 vs. 0 domains		2.0 (0.9, 4.4)		2.0 (0.9, 4.5)
3+ vs. 0 domains		2.4 (1.0, 5.7)		3.0 (1.3, 7.3)

[a] Controlling for income, education, smoking, alcohol, and depression.

Note: Study population comprised 152 black and 200 white women who gave birth between the year 7 exam (1992) and the year 10 exam (1995).

Source: Mustillo et al. (2004).

Thus what our results showed—for the first time—was that experiences of racial discrimination not only predicted risk of preterm delivery, but also explained the excess black-white risk that the other conventional risk factors could not.

Prior to our investigation, however, most studies had included only conventional risk factors, like those we included in model 3, thereby concluding that the remaining unexplained black excess risk of premature delivery must be due to some innate biological difference between black and white women (Krieger et al. 1993; Giscombé and Lobel 2005). But we show that this interpretation is wrong because our study clearly demonstrated that by including information on experiences of racial discrimination, we could explain the observed black-white disparities. This is a very powerful finding—and it adds further evidence to the claim that the source of racial/ethnic disparities is the injustice in our society, not innate biology.

The last empirical example serves as a reminder that racial discrimination, however important it may be as a determinant of racial/ethnic health inequities, is not the only adverse type of social hazard to which people of color are exposed. Thus, drawing on the full cohort of 1,202 low-income employed workers that served as the population with which we validated the EOD, we found that participants reported not only racial discrimination, but also workplace abuse and sexual harassment (Krieger et al. 2006, 2008a). More specifically, among the black workers, the two most common combinations, together reported by over half the women and men, were, first, racial discrimination combined with workplace abuse, followed by all three types combined, with the latter most common among the black women. Among the Latino workers, a different set of combinations were the two most common, again reported by over half the women and men: first, racial discrimination plus workplace abuse, followed by workplace abuse alone. Last, among the white workers, the most common category by far was workplace abuse alone, reported by slightly over 40 percent of the women and men. Additional analyses showed that, in the case of sexual harassment, one additional social category was relevant: that of sexuality, with the lesbian, gay, bisexual, and transgender (LGBT) workers reporting twice as much sexual harassment as their heterosexual counterparts.

The net implication is that racial discrimination matters—and does not occur in isolation. This, too, is the point of the ecosocial construct of embodiment and its recognition that each and every day, our bodies daily integrate our experiences. Hence, to analyze the determinants of health inequities, including racial/ethnic health disparities, we have to remember that we are not one day a woman or a man, another day white or a person of color, another day working class or a professional, another day straight or LGBT, and still another day U.S. born or foreign born: we are all of these at once, with the implication being that our research must reckon with diverse, yet combined and embodied, aspects of social position (Krieger 1994, 2005b, 2006).

Conclusion: From "What's the Use of Race" to the Need for Research and Action on Racism and Health

As the arguments and examples of this chapter make clear, in the case of population health and racial/ethnic health inequities, the meaningful question becomes, what's the use of race in research on racism and health? This is because (1) racism and the social categories of race/ethnicity that it creates are socially real and (2) racism harms health, and does so differentially by race/ethnicity, thereby producing racial/ethnic health inequities. Consequently, to measure the impact of racism on health, we need to employ (1) appropriate racial/ethnic categories to distinguish its targets—and perpetrators—and (2) relevant measures of exposure to structural, institutional, and interpersonal racism. These same considerations pertain to research regarding the links between class and health, gender and health, and any other social construct implicated in health inequities: we need categories to identify the populations defined by these social relations, and we need data to quantify the relevant measures of exposure to adverse conditions. The corollary is that calls for being color-blind in data collection are not color neutral; rather, they are an expression of denial and bias about the seriousness of how racism harms individuals and societies.

One final question, then, ought be asked: what's the use of research on racism and health? The clear-cut answer is that this research is necessary to understand and provide evidence relevant to addressing racial/ethnic health inequities. Although scientific knowledge, like any knowledge, can by itself change nothing, when people organize to apply it, change can happen. The example I have provided regarding the shrinking of U.S. socioeconomic and racial/ethnic health inequities between the mid-1960s and 1980 is a case in point: descriptive and analytic knowledge about the existence of these inequities and their causes was critical to spurring and guiding action to rectify them.

Consider, too, the recent the Public Broadcasting Service series *Unnatural Causes . . . Is Inequality Making Us Sick?* (California Newsreel 2009), aired nationally in spring 2008 (and for which I served, by way of disclosure, as one of the scientific advisors and participants). The series's clear message is that racism and economic deprivation harm health and can be challenged and countered. Widely acclaimed, it was bestowed the prestigious 2009 Alfred I. duPont–Columbia University Award, broadcast journalism's equivalent of the Pulitzer Prize (California Newsreel 2009). Its power derived in part from its ability to link the findings of extant research about racism, economic deprivation, and health to individual stories that simultaneously illuminated and gave credence to the experiences of those now suffering the brunt of health inequities. The series likewise succeeded in breaking through the individualism so rampant in our society, by making clear that—in the old-fashioned language of the day—the personal is, in fact, political. By this I mean that the series revealed how people's health woes—including those involving racial/ethnic health inequities—are at once individual and societal: we experience them as the unique individuals we are, within a context of societally structured options and constraints. Already shown at over twelve thousand community and organizational meetings by the end of 2008 (California Newsreel 2009), the series is being used by health professionals and health advocates, nationally and locally, to educate the broader public and affected communities about how social injustice harms health—and what can be done to change this. As should be obvious, the series's insights, evidence, and recommendations for action are premised on meaningful use of data about race/ethnicity, racism, and health; without using categories of race/ethnicity, the series would not have been possible.

In conclusion, the use of "race"—that is, racial/ethnic categories—is essential for the science and epidemiology of racism and health. The rationale for this work is to generate knowledge that, if put into action, can inform current efforts to eliminate health inequities and improve population health. To do this work, it is not an option either to ignore categories of race/ethnicity or to analyze them without reference to their societal context and the impact of racism on health. Only by grappling with the paradox of using categories of race/ethnicity to vanquish racial injustice, rather than attempting to proscribe their use, do we stand a chance in furthering our goals of racial justice and social equity in health.

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