



# Structural racism and myocardial infarction in the United States



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## ABSTRACT

There is a growing research literature suggesting that racism is an important risk factor undermining the health of Blacks in the United States. Racism can take many forms, ranging from interpersonal interactions to institutional/structural conditions and practices. Existing research, however, tends to focus on individual forms of racial discrimination using self-report measures. Far less attention has been paid to whether structural racism may disadvantage the health of Blacks in the United States. The current study addresses gaps in the existing research by using novel measures of structural racism and by explicitly testing the hypothesis that structural racism is a risk factor for myocardial infarction among Blacks in the United States. State-level indicators of structural racism included four domains: (1) political participation; (2) employment and job status; (3) educational attainment; and (4) judicial treatment. State-level racial disparities across these domains were proposed to represent the systematic exclusion of Blacks from resources and mobility in society. Data on past-year myocardial infarction were obtained from the National Epidemiologic Survey on Alcohol and Related Conditions (non-Hispanic Black:  $N = 8245$ ; non-Hispanic White:  $N = 24,507$ ), a nationally representative survey of the U.S. civilian, non-institutionalized population aged 18 and older. Models were adjusted for individual-level confounders (age, sex, education, household income, medical insurance) as well as for state-level disparities in poverty. Results indicated that Blacks living in states with high levels of structural racism were generally more likely to report past-year myocardial infarction than Blacks living in low-structural racism states. Conversely, Whites living in high structural racism states experienced null or lower odds of myocardial infarction compared to Whites living in low-structural racism states. These results raise the provocative possibility that structural racism may not only harm the targets of stigma but also benefit those who wield the power to enact stigma and discrimination.

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## Introduction

Over the past few decades, researchers have directed considerable attention toward the study of racial discrimination and its effects on the health of Blacks in the United States. Much of this research has been undertaken with the goal of explaining racial disparities in morbidity and mortality. Indeed, despite adjustments for socioeconomic status and health behaviors, racial disparities persist in such outcomes as life expectancy and mortality from leading causes of death including heart disease, hypertension, and diabetes (CDC, 2011).

Researchers have postulated that racial discrimination is an important factor undermining the health of Blacks relative to Whites (e.g., Jones, 2000; Krieger, 2012; Williams & Mohammed, 2009). Krieger (2012), for example, proposes an “ecosocial” theory of racism and health. According to this model, racism becomes “embodied” over the life-course, adversely affecting the health of oppressed populations through multiple pathways, ranging from exposure to toxins to economic and social deprivation (Krieger, 2012). Importantly, Krieger’s model highlights the potential duality of the impact of racism on health—a process that both harms subordinate social groups while providing benefits to dominant ones.

The power dynamic central to the ecosocial model is consistent with other theories of structural stigma (Link, 2014; Link & Phelan, 2001) and systemic racism (Feagin, 2000; 2006; Feagin & Bennefield, 2013). Racism may be conceptualized as a tool employed by those in power to maintain privilege and control over resources (for example, wealth, knowledge, prime land and

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housing) that ultimately benefit health (Link, 2014; Link & Phelan, 1995). Furthermore, as Bonilla-Silva (1997) asserts, the pervasive nature of racism, and the “racialized social systems” (p. 469) that define it, extend beyond ideology and class conflict to permeate the structure of society and shape “life chances” (p. 470) in a racialized way across multiple domains (e.g., political, social, and economic). Although structural racism as proposed in the ecosocial model is hypothesized to harm the health of Blacks while potentially bolstering that of Whites, few empirical studies have directly assessed this relationship. In outlining the ecosocial hypothesis, Krieger, Chen, Koshelva, and Waterman (2012) provide some data examining the effects of Jim Crow legislation on mortality in the United States. Comparing mortality among Whites and persons of color in states with and without Jim Crow legislation, a tiered relationship emerged (namely in the decade between 1960 and 1970), with the highest rates of mortality occurring in populations of color within Jim Crow states and the lowest rates of mortality occurring among Whites in these highly racist environments. The current study expands the literature on structural racism and encompasses two primary aims. First, we examine whether structural racism serves as a risk factor for myocardial infarction among Blacks in the United States. Second, consistent with the above theories, we evaluate whether the cardiac health of Whites is enhanced under conditions of structural racism against Blacks.

Cardiovascular health has been of particular interest to discrimination researchers (Williams & Mohammed, 2009; Wyatt et al., 2003). Indeed, multiple lines of evidence document associations between reporting discrimination and markers for cardiovascular disease among Blacks, including hypertension (Davis, Liu, Quarells, & Din-Dzietharn, 2005; Guyll, Matthews, & Bromberger, 2001; Krieger, 1990; Krieger & Sidney, 1996; Roberts, Vines, Kaufman, & James, 2007; Sims et al., 2012), subclinical carotid disease (Troxel, Matthews, Bromberger, & Sutton-Tyrrell, 2003), coronary artery calcification (Lewis et al., 2006), coronary artery obstruction (Ayotte, Hausmann, Whittle, & Kressin, 2012), elevated low-density lipoprotein (LDL) cholesterol, visceral abdominal fat deposits (Lewis, Kravitz, Janssen, & Powell, 2011), increased C-reactive protein (Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010), and, in experimental designs, cardiovascular reactivity in response to acute discriminatory stress (Lepore et al., 2006; Smart Richman, Bennett, Pek, Siegler, & Williams, 2007). One of the reasons that cardiovascular health has garnered such attention among discrimination researchers is that both theoretical and empirical work indicates that discrimination serves as a chronic stressor for Black Americans (Clark, Anderson, Clark, & Williams, 1999). As such, discrimination can be linked to poor cardiovascular outcomes through stress-response systems (Dimsdale, 2008; Sawyer, Major, Casad, Townsend, & Mendes, 2012; Williams & Mohammed, 2009; Wyatt et al., 2003), providing a plausible set of biological mechanisms through which discrimination may influence myocardial infarctions. Methodologically, we focused the present study on myocardial infarctions because, unlike other self-reported health outcomes, measures of heart attack and angina have been found to be highly reliable (Bergmann, Byers, Freedman, & Mokdad, 1998; Bush, Miller, Golden, & Hale, 1989; Lampe, Walker, Lennon, Whincup, & Ebrahim, 1999).

#### *Measuring interpersonal and structural forms of racism*

Racism acts through discrimination at various levels of society, from interpersonal events (e.g., victimization) to structural (also called institutional) practices and conditions (Krieger, Rowley, Herman, Avery, & Phillips, 1993; Meyer, 2003). Interpersonal discrimination can include actions that are intentional and unintentional, and it manifests itself in several different ways,

including “lack of respect, suspicion, devaluation, scapegoating, and dehumanization” (Jones, 2000, p. 1213). In contrast to forms of discrimination that occur on an individual or interpersonal level, structural discrimination refers to macro-level conditions that constrain the opportunities, resources, and well-being of socially disadvantaged groups (Link & Phelan, 2001). These conditions are embedded in structural relations that maintain and perpetuate greater social influence among majority group members (Bonilla-Silva, 1997; Jones, 2000; Link & Phelan, 2001) and are therefore considered independent of individual-level discrimination (Bonilla-Silva, 1997; Meyer, Schwartz, & Frost, 2008).

The vast majority of studies examining racial discrimination and health, including cardiovascular health, have relied on self-report measures of interpersonal events (Paradies, 2006; Williams & Mohammed, 2009). The most commonly used measures of interpersonal discrimination, or *perceived discrimination*, query respondents about whether they have been discriminated against in a variety of major life domains (e.g., healthcare, education, employment) or in everyday circumstances (e.g., followed in stores, called names or insulted), as a result of their race (Williams & Mohammed, 2009). These self-report, check-list measures, however, are vulnerable to measurement error. For instance, individuals who experience discrimination may not be willing to report these sensitive events (Krieger, 1999; Meyer, 2003; Williams & Mohammed, 2009) or may vary in their perceptions and interpretations of these events (Krieger, 1999; Meyer, 2003), potentially resulting in biased estimates of the relationship between discrimination and health (Krieger 1999; Meyer, 2003; Williams & Mohammed, 2009).

In addition, measures of perceived discrimination often cannot capture structural forms of discrimination (Hatzenbuehler, McLaughlin, Keyes, & Hasin, 2010; Meyer, 2003). Although a number of researchers have called for the development of such measures (Krieger, 2012; Lauderdale, 2006; Shavers et al., 2012; Williams & Mohammed, 2009), few measures of structural discrimination are currently available. There are at least three reasons for the relative absence of structural measures in the extant literature. One is the tendency of public health research to focus on individual-level risk factors, often perceived as more amenable to intervention (Feagin & Bennefield, 2013; Susser, Schwartz, Morabia, & Bromet, 2006).

A second formidable barrier lies in the identification and development of measures that legitimately represent this construct. Indeed, measuring structural racism presents a significant challenge to researchers given the shift over the past half-century from overt, and often legally sanctioned, forms of discrimination to largely “aversive” ones characterized by avoidance of racial/ethnic minorities and implicit expressions of racism (Bonilla-Silva, 1997; Dovidio & Gaertner, 2004; Gaertner & Dovidio, 1986; Krieger, 2012). Because aversive forms of racism often exist outside of conscious awareness, traditional self-report measures cannot be used to reliably evaluate this construct. Structural racism can also be obscured or “misrecognized” (Bourdieu, 1979) through processes such as White racial framing (Feagin, 2006) and “stigma power” (Link, 2014). Given the difficulty of capturing structural forms of racism that are frequently concealed, researchers have been hard pressed to find individual examples of structural racism that have adequate construct validity. Because of this challenge, the development of multiple (rather than single) indicators that represent a pattern of racial inequity at a structural level might strengthen the body of evidence in the field. Third, structural racism is often ubiquitous, making it especially difficult to identify measures that capture sufficient variation to predict health outcomes.

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