Mechanisms of Racial Health Disparities in Inflammation: A Test of the Differential Stress Exposure and Differential Stress Vulnerability Hypotheses

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Extended Abstract

BACKGROUND

Despite decades of research and public health intervention, disparities in cardiovascular diseases among social groups persist and are only partially explained by parallel differences in socioeconomic status or conventional risk factors, such as smoking. Relative to non-Hispanic whites, racial minorities are particularly at risk of developing and dying from cardiovascular diseases (Rogers et al., 2011). How do we explain this enduring gap in health status and what more can be done to curtail this disparity? Answering these questions necessitates acknowledging that the social patterning of cardiovascular diseases is not a chance occurrence; rather, racial disparities in health are rooted in a system of social stratification that consistently and systematically disadvantages certain subgroups of the population (Adler & Rehkopf, 2008) through the unequal distribution of fiscal and material resources, on the one hand (Williams & Mohammed, 2009), and through the unequal distribution of stressors and coping resources, on the other hand (Pearlin, 1989; Williams & Mohammed, 2009).

Discrimination is a chronic stressor that disproportionately affects racial minorities (Kessler, Mickelson & Williams, 1999). It manifests as discrete life events (i.e., major lifetime discrimination) that significantly hinder opportunities for status attainment and as day-to-day experiences of unfair treatment (i.e., everyday discrimination), such as being treated with less respect than others or being threatened or harassed (Williams, Yu, Jackson, & Anderson, 1997). Prior research has linked exposure to discrimination to multiple adverse health outcomes including low infant birth weight, depressive symptoms, all-cause mortality, and risk factors for cardiovascular diseases, such as high blood pressure (for a review of this literature see: Mays, Cochran & Barnes, 2007; Pascoe & Richman, 2009; Williams & Mohammed, 2009). Therefore, racial disparities in cardiovascular diseases may be attributed to differences in exposure to discrimination.

The current study sheds light on the mechanisms through which group differences in exposure to and the impact of discrimination generate disparities in cardiovascular diseases. We specifically look at its effects on C-reactive protein (i.e., CRP), a protein produced in response to exposure to stressors. It is a marker of systemic inflammation that is positively associated with cardiovascular diseases such as stroke, atherosclerosis, and myocardial infarction (Ridker, Cushman, Stampfer, Tracy, & Hennekens, 1997; Ridker, Hennekens, Buring, & Riafai, 2000). The current study goes beyond a basic description of the population-level distribution of CRP by focusing on psychosocial factors that mediate and/or moderate these relationships. We specifically examine: (1) race and gender differences in CRP levels; (2) the extent to which these differences are explained by parallel differences in exposure and/or vulnerability to discrimination; and (3) the extent to which perceived social support—a coping resource—dampens these effects thereby helping to explain group differences in cardiovascular outcomes.

METHODS

We use data from the Health and Retirement Study (HRS), a multi-cohort longitudinal survey of a large and nationally representative sample of adults age 51 years and older. The HRS aims to identify and better understand the social, economic, psychosocial, and physical factors that influence and result from retirement. Since its inception in 1992, data have been collected every two years on the original HRS cohort and on subsequent cohorts added to the study. The "core" HRS interview is conducted face-to-face at baseline and collects data on demographic characteristics; housing and family structure; employment, income, assets, and insurance; health care, health status, cognition, and disability; and life expectations. Beginning in 2004, HRS respondents were asked to complete a psychosocial questionnaire that included measures of everyday and lifetime discrimination, among other factors, and in 2006 biomarkers—including CRP—were collected from one half of the study sample; biomarkers were collected from the other half in 2008. To maintain sufficient statistical power to detect differences in CRP, multivariate analyses are conducted with data from a hybrid 2006/2008 sample. The analysis involves statistical methods needed to appropriately adjust for the complex sampling design of HRS and a mediated moderation analysis to test the intricate causal pathways leading to race differences in CRP.

PRELIMINARY RESULTS

In line with our hypotheses, African Americans have higher levels of CRP than non-Hispanic whites and reported greater exposure to everyday and lifetime discrimination. Hispanics do not significantly differ from non-Hispanic whites in CRP levels or in their reports or everyday and lifetime discrimination. In unadjusted models, everyday and major lifetime discrimination are positively associated with CRP. This association remains for lifetime discrimination in fully adjusted models but not for everyday discrimination. Both everyday and lifetime discrimination mediate racial/ethnic and gender differences in inflammation. Lifetime discrimination mediates these differences in and of itself, and in conjunction with other factors, particularly waist circumference. Everyday discrimination also mediates group differences in inflammation in conjunction with waist circumference. Vulnerability to discrimination does not differ by race/ethnicity or by gender.

CONCLUSION

This study supports the presence of racial differences in CRP—an indicator of systemic inflammation and a clinical risk factor for cardiovascular diseases—on a population-level. The findings also suggest that exposure to discrimination, a chronic stressor that disproportionately affects racial minorities, is associated with higher levels of CRP and partially explains racial and gender differences in inflammation. In all, these findings provides further support for the significant contributions of the social environment on health and health disparities.

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