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How does race get "under the skin"?: Inflammation, weathering, and metabolic problems in late life

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ABSTRACT

Using nationally representative data from the 2005–2006 U.S. National Social Life, Health, and Aging Project, this study queries the mechanisms underlying worse metabolic outcomes—blood-sugar control and cardiovascular health—among black than white men ages 57–85. Results indicate that contrary to much of the academic literature as well as media accounts—implicitly rooted in a "culture of irresponsibility" model—older black men's social isolation, poor health behaviors, or obesity may not play a major role in their worse metabolic problems. Instead, these outcomes seem to derive more consistently from a factor almost unexamined in the literature—chronic inflammation, arguably a biological "weathering" mechanism induced by these men's cumulative and multi-dimensional stress. These findings highlight the necessity of focusing attention not simply on proximal behavioral interventions, but on broader stress-inducing social inequalities, to reduce men's race disparities in health.

Introduction

An established literature documents greater diabetic and cardiovascular problems among black men at any age, and especially in late life (Heron, 2007). Recent biomedical studies suggest a syndromal clustering of these two metabolic conditions—suggesting common, possibly social, antecedents (Goossens, 2008; Grundy, Brewer, Cleeman, Smith, & Lenfant, 2004; Yudkin, 2003). The sparse sociological literature on underlying mechanisms generally examines prevalence of diagnosed disease (Dupre, 2008; Morenoff et al., 2007)—problematic given common underdiagnosis of these issues among minority groups (Geiss et al., 2006; Timmermans & Haas, 2008). Few studies directly examine blood-sugar control and cardiovascular health, and compare multiple mechanisms. Moreover, both the academic literature and news media frequently posit obesity—conceived as a result of poor social control and unhealthy behaviors—as the "biological gateway" through which social and behavioral mechanisms affect these distal metabolic outcomes. Recent studies on stress-physiology linkages, however, suggest other biosocial pathways (Geronimus, 2001; McDade, 2005). Due to a lack of large-sample surveys containing requisite social as well as biological indicators, these mechanisms remain underexamined.

Using data from the 2005–2006 U.S. National Health and Social Life Project (NSHAP)—a nationally representative probability sample of adult Americans aged 57–85—the present study begins to fill these gaps. Specifically, it examines and compares a series of social, behavioral, stress-process, and biological mediators of the impact of race on men's blood-sugar control and cardiovascular health.

Race and metabolic problems

Four potential mechanisms can be extracted from the sociological and biomedical literatures, for older black men's worse metabolic outcomes: (1) social support and/or control; (2) health behaviors; (3) obesity, possibly as a consequence of social and behavioral factors; and (4) a stress-inflammation chain.

Social support and control

An established literature indicates the health benefits of more and stronger social ties—whether through more control of unhealthy behaviors, or through emotional and instrumental support, including caregiving (Coleman, 1988; Kawachi, Kennedy, & Glass, 1999). These control and support mechanisms can work through multiple features of one's egocentric network—starting with network size, which may simply index a greater number of social alters invested in one's health. Similarly, the likelihood of such investments arguably increases with the strength of the social

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relationship. Multiple studies indicate that especially at older ages, extra-family networks shrink, with retirement and the curtailment of social activities due to functional or health problems (McPherson, Smith-Lovin, & Brashears, 2006; Schnittker, 2007). In this life stage, strong ties are most likely with one's relatives—especially those related by blood—who may become crucial sources of health-monitoring and caregiving (Shaw, Krause, Liang, & Bennett, 2007). Hence, networks richer in consanguineal or blood relatives arguably have more control and support potential. Finally, studies have long emphasized the importance of "closure"—the degree to which network members are interconnected—in enhancing social control, by allowing alters to combine forces to sanction a focal individual's adverse behaviors (Coleman, 1988).

Sociological literature is more ambiguous about men's race differentials in these health-producing social assets. Some studies indicate greater social isolation among black men—with accounts of underlying mechanisms reflecting an implicit moralistic narrative of these men's non-engagement in social and especially familial roles (Burton & Snyder, 1998; Jarrett, Roy, & Burton, 2002). In contrast, a separate literature suggests that black men may be *more* strongly embedded than their white counterparts in densely interconnected network structures, centered in church membership and the matriarchal kin group (Hill, 1999; Sarkisian & Gerstel, 2004). Consistent with the isolation argument, Cornwell, Schumm, and Laumann (2008) combined-gender NSHAP study (additively including gender and ethnicity) finds smaller networks among all black participants. However, it also finds few race differentials in other egocentric network attributes—with existing differentials generally favoring black participants. Finally, if race associations are gender-differentiated—with black men having worse and black women better social ties—combined analyses may possibly mask the former deficits.

To test these competing arguments about the role of older black men's social deficits in mediating their worse diabetic and cardiovascular outcomes, Hypothesis 1 (Table 1) is included.

Behaviors, obesity, inflammation: culture or stress?

Next, an established literature suggests that black men may have worse health behaviors (Williams, 2001; Williams & Collins, 1995). In turn, diabetic and cardiovascular outcomes have been linked to both actively risky behaviors such as smoking (Ockene & Miller, 1997) and alcoholism (Lakka et al., 2002)—as well as passive ones including too much (Wilson, 2005) or too little sleep (Cappuccio et al., 2007; Miller et al., 2009), and inactivity (Haskell et al., 2007). A final health-related behavior that might affect these conditions is underutilization of healthcare, which studies suggest is particularly common in black communities—arguably due to low social compatibility with and/or trust in medical practitioners (van Ryn, 2002). These conjectures lead to Hypothesis 2 (Table 1).

Moreover, especially in the sociological literature, many of these behaviors are assumed to flow through obesity. Whether due to more cultural acceptance of weight (Bennett & Wolin, 2006), neighborhood cultural patterns (Boardman, Saint Onge, Rogers, & Denney, 2005), or fatalism about health (Plowden, 2003), black individuals' obesity-inducing behaviors have been presented as a major cause for their worse health outcomes. Or in other words, obesity has implicitly or explicitly been portrayed as *the* "biological gateway" through which behavioral mechanisms affect metabolic problems. Indeed, the "obesity epidemic" in recent decades has been depicted—in both the news media and academic literature—as key to increases in these chronic conditions (Campos, Saguy, Ernsberger, Oliver, & Gaesser, 2006; Saguy & Almeling, 2008).

Taken together, these conceptions arguably represent an implicit "culture of irresponsibility" model that depicts black men's unhealthier social and behavioral propensities as distal antecedents that, acting partly through obesity, lead to their greater morbidity and mortality (Oliver, 2006; Saguy & Gruys, 2010). Consistent with this notion, an extensive literature does link worse health behaviors (diet, inactivity) with obesity, and documents the causal role of obesity in blood-sugar control, diabetes, and heart disease (Grundy et al., 2004; Yudkin, 2003). However, while a range of studies indicates greater obesity among black individuals of all ages (Denney, Krueger, Rogers, & Boardman, 2004; Mokdad et al., 2003), recent evidence suggests a gender-differentiated pattern—with black men *less* obese than their white counterparts, and the reverse holding for women (Chang & Lauderdale, 2005). Moreover, some biomedical literature also suggests that obesity, in itself, may not be as responsible for metabolic problems as previously thought (Abbasi, Brown, Lamendola, McLaughlin, & Reaven, 2002; Reaven, 1995). Accordingly, Hypothesis 3 (Table 1) is included to help adjudicate between these conflicting accounts of obesity's mediatory role.

Rather than race-specific cultural patterns, moreover, metabolic pathologies may also derive from older black men's greater stress-induced "weathering" (Geronimus, 2001; Geronimus, Hicken, Keene, & Bound, 2006). Weathering refers to the cumulative health impact of black individuals' repeated experiences with social, economic, or political exclusion—with the presence of these stressors in multiple life dimensions, and consequent high-effort coping, potentially inducing morbidity both directly and through unhealthy behaviors. Thus, an extensive literature links stress to multiple behavioral issues, including smoking (Feldner, Babson, & Zvolensky, 2007; Koenen et al., 2005), alcohol use (Davis, Uezato, Newell, & Frazier, 2008; Grant & Harford, 1995), poor sleep (American Psychiatric Association, 2000) as well as physical inactivity (Verger, Lions, & Ventelou, 2009)—factors, as noted above, linked to metabolic problems. More directly, studies indicate race disparities in "allostatic load"—multi-systemic physiological wear-and-tear through long-term exposure to stress-induced fluctuations or elevations in neuroendocrine response (Geronimus et al., 2006; McEwen, 1998; Singer, Ryff, & Seeman, 2004; Sterling & Eyer, 1981). Finally, recent biodemographic literature suggests that low-grade chronic inflammation, triggered by prolonged exposure to stressful environments, may be a key mechanism through which weathering works, and produces allostatic load. Multiple studies indicate a pathway from extended psychosocial stress to inflammation (McDade, Hawkley, & Cacioppo, 2006; Melamed, Shirom, Toker, Berliner, & Shapira, 2006; Weinstein, Vaupel, & Wachter, 2007)—possibly due to norepinephrine-driven gene expression of inflammatory mediators (Bierhaus et al., 2003; Kiecolt-Glaser, Gouin, & Hantsoo, 2010). Accordingly, C-reactive protein (CRP), an inflammation indicator, is increasingly being used as a biomarker of chronic stress—whether in the British Whitehall II studies (Owen, Poulton, Hay, Mohamed-Ali, & Steptoe, 2003), Chicago (McDade et al., 2006), or in the United States (Alley et al., 2006). Downstream, inflammation has a demonstrated causative role in cardiovascular problems, as well as poor blood-sugar control due to insulin resistance (Grundy et al., 2004; Yudkin,

Table 1
Summary of hypotheses.

Relative to their white counterparts, older black men's worse diabetic and cardiovascular outcomes will be mediated by their:
1. worse social deficits—indicated by smaller networks, with less close ties, less closure, and smaller proportions of blood relatives.
2. worse health behaviors—both active (smoking, alcohol consumption) and passive (inactivity, poor sleep).
3. greater obesity.
4. worse inflammation, due to greater chronic stress.