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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 to 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.

Keywords

USA; race disparities; black; weathering; blood sugar; diabetes; cardiovascular; inflammation

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 et al., 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.

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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 to 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 et al., 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 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 et al., 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 et al., 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 et al., 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 et al.’s (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 et al., 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 et al., 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 et al., 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 et al., 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 et al., 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 et al., 2007; Koenen et al., 2005), alcohol use (Davis et al., 2008; Grant & Harford, 1995), poor sleep (American Psychiatric Association, 2000) as well as physical inactivity (Verger et al., 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 et al., 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 et al., 2006; Melamed et al., 2006; Weinstein et al., 2007)—possibly due to norepinephrine-driven gene expression of inflammatory mediators (Bierhaus et al., 2003; Kiecolt-Glaser et al., 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 et al., 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, 2003). More specifically, recently developed high-sensitivity CRP assays suggest that chronic, low-grade inflammation (indicated by lower CRP levels) is linked to subsequent incidence of cardiovascular disease (Danesh et al. 2000), type 2 diabetes (Pradhan et al., 2001), and the “metabolic syndrome” (McDade & Hayward, 2009; Ridker et al. 2003). Moreover, in their combined-gender NSHAP study (additively including gender and ethnicity), McDade et al. (2011) find higher inflammation/CRP among older black than white adults. Thus, in addition to obesity, inflammation represents a second—and underexamined—“biological gateway” through which older black men’s social environments may affect their diabetic and cardiovascular outcomes.

Apart from stress, however, inflammation has also been linked to obesity (Yudkin, 2003), smoking and heavy alcohol-consumption (Chiu et al., 2008; Mukamal, 2006), and lack of sleep (Meier-Ewert et al., 2004). Much of this evidence comes from cross-sectional studies, leaving ambiguity over whether inflammation is a consequence of these factors—or, as a long-term stress marker, a cause. It is argued, therefore, that inflammation represents a stress-induced biological weathering mechanism to the extent that the pathways through which it mediates race differentials in metabolic problems do not include obesity and/or health behaviors as causal antecedents. To test this potential mediatory role of inflammation, Hypothesis 4 (Table 1) is included.

To summarize, then, the four mechanisms drawn from the literature fall into two categories. A first set of implicitly moralistic approaches sees men’s black-white differentials in diabetic and cardiovascular health as stemming from social deficits due to disengagement from social and family roles; from unhealthy behaviors as a function of race-specific cultural patterns; and (consequently) from obesity. A second emphasizes long-term biological weathering induced by older black men’s cumulative and multi-dimensional stress—whether in directly causing metabolic pathologies via chronic inflammation; or through unhealthy behaviors.

METHODS

Data

Data are from the 2005–2006 U.S. National Social Life, Health, and Aging Project. NSHAP is a nationally-representative probability sample of 1550 women and 1455 men aged 57 to 85, with an oversampling of blacks, hispanics, men, and those 75 to 85. In addition to self reports, data include assessments of physical function, height and weight, as well as salivary and blood samples—all collected at the time of interview by non-medically trained interviewers. The survey has an unweighted response rate of 74.8% and a weighted response rate of 75.5% (O’Muirheartaigh & Smith, 2007). Participant consent was obtained prior to interview. Institutional review boards at the Division of the Social Sciences and NORC at the University of Chicago approved data collection procedures.

Measures

Table 2 presents summary statistics for all variables used in the analyses.

Predictors and controls—A participant’s *race* is indexed by a set of dummy variables for *black* and *hispanic/other*, with *non-hispanic white* as the reference category. Seventy percent of men in the hispanic/other category are non-black hispanics, with the remainder

comprised of American Indians or Alaskan natives, Asian or Pacific Islanders, and “other.” All analyses linearly control one’s *age* (in years). Models including less healthcare utilization also add *no healthcare access*, a dummy self-report indicating that a participant has no place to go when sick. Finally, Tables 4 and 5 include controls for *less education*—an integer score ranging from 1 (Bachelors degree or more) to 4 (less than a high school education)—and any lifetime diagnosis of *diabetes* or *hypertension* by a medical doctor.

Depression—A current *depressed* state is used to validate stress-inflammation linkages in preliminary analysis. NSHAP depression items are derived from the Iowa form of the Center for Epidemiological Studies Depression Scale (CES-D; Shiovitz-Ezra et al., 2009), with each 4-category Likert item indexing experiences over the preceding week of one of 11 depressive states. Those with scores above 9.0 on the full additive scale are categorized as depressed (Zauszniewski & Graham, 2009).

Mediators—Four attributes of a participant’s egocentric social network tap deficits in support and monitoring/control. All are based on nominations of alters during the network-roster portion of the face-to-face interview (Cornwell et al., 2008). First, *fewer alters* is a simple reverse-coded count of one’s network members, and runs from 1 (5 or more) to 6 (no alters). The next three variables all range continuously from 0 to 1, with higher values denoting worse ties. *Less close ties* indicates a participant’s average (self-reported) lack of closeness with his/her alters. *Less network closure* is proxied by the average (participant-reported) frequency of contact among all members of one’s network—including the frequency of contact among nominated alters (in alter-alter dyads) as well as between a participant and each alter (in ego-alter dyads). Finally, *smaller kin proportion* is indexed by the proportion of one’s alters who are consanguineal kin (i.e., related by blood).

Measures of poor health behaviors include *poor sleep*—based on self-reports of “usually” sleeping less than 7 or more than 9 hours a night. Physical *inactivity* is measured by one’s self-reported frequency of participation—on a regular basis—in activities such as walking, dancing, gardening, physical exercise or sports. This variable ranges from 1 (3 or more times per week) to 5 (never). Next, studies indicate race differences in underreports of smoking in large-sample surveys (Fisher et al., 2008). Hence, a participant’s smoking habits are measured through his cotinine levels (Drum et al., 2009). Cotinine is a metabolite of nicotine, derived from saliva samples taken during the biomeasure collection portion of the interview. Based on the Wells-Stewart method (Wells, 1993), men with cotinine greater than 100 ng/ml are classified as regular *smokers*. Next, a participant’s alcohol consumption is indexed by two ordinal self-reports. *More alcohol* indicates average weekly drinking frequency, and runs from 0 (none) to 3 (daily or more). *Binge drinking* indicates the number of days in the preceding three months a participant has had four or more drinks on one occasion, and runs from 0 (none) to 2 (two or more). Finally, *less healthcare utilization* is indexed by one’s ordinal self-reported frequency of seeing a doctor in the preceding 12 months, and ranges from 0 (30 or more) to 7 (none).

Of the two posited biological mediators, abdominal *obesity* is indicated by waist size (in inches). Next, in keeping with previous studies (Alley et al., 2006; Owen et al., 2003; McDade et al., 2006), low-grade chronic *inflammation* is indexed by C-reactive protein (CRP)—derived from high-sensitivity assays of dried blood spots collected through capillary finger-sticks (Williams & McDade, 2009). Due to heavy right skew, raw CRP values are log-transformed, with values greater than 8.6 mg/l excluded from analysis as more indicative of acute (short-term) infection than prolonged stress-exposure (McDade et al., 2004).

Metabolic outcomes—Diabetic problems, or issues with *blood sugar control*, are indicated by log-transformed Hemoglobin A1c (HbA1c)—glycosylated hemoglobin as a percentage of total hemoglobin. As with CRP, HbA1c is derived from dried blood spots, and measures plasma glucose concentration, with higher levels indicating worse blood sugar control. Next, cardiovascular health is indexed by *systolic* and *diastolic BP* (blood pressure, in mm Hg), and *heart rate* (in beats per minute)—each indicator representing the mean of two successive readings.

Analytic plan

The analysis is set up in stages, aimed at progressively eliminating posited mechanisms. While the tables present results for “hispanic or other” men, this is solely for baseline information. Given the theoretical focus of the study, the account below focuses on black-white differentials.

The first stage (Table 3) explores men’s age-adjusted race differentials in posited social, behavioral, and biological (obesity, inflammation) mediators, as well as metabolic outcomes (blood sugar control and the three cardiovascular factors). As noted, the model examining healthcare utilization also adjusts for access to care. A wider array of controls is deliberately not used in these analyses, to avoid overcontrolling race differentials. As the last section clarifies, these dependent variables include continuous, ordinal, as well as dichotomous indicators. Accordingly, results are from OLS, ordinal logit, and logistic regression models. To facilitate pattern-visualization, coefficients rather than Odds Ratios are presented for categorical outcomes.

Next, associations between each posited mediator with a significant black-white differential, and each distal metabolic outcome, are examined one-at-a-time in Table 4. As noted, the proposed mechanisms are potentially interrelated. Hence, these “gross models” also analyze interdependencies between these factors. To adjust for potential endogeneity, additional controls are added in Table 4 to models examining biological outcomes (inflammation, metabolic states). These include any lifetime diagnosis of diabetes or (separately) hypertension, which net out feedback from health conditions—and more generally, the possibility that observed associations are confounded by prior disease. (To the extent that current social and behavioral factors—and especially chronic inflammation—index long-term processes responsible for race differentials in the prevalence of diagnosed diabetes and hypertension, these analyses are overcontrolled, and yield conservative estimates of mediator-outcome linkages.) To additionally control reverse causation from current spikes in health status, healthcare access and utilization in the preceding year are added—on the argument that behavioral and stress responses to such issues largely occur through medical notification. These factors are not adjusted in models with social mediators as independent variables, since it is partly by inducing changes in healthcare behaviors that social ties may affect biological outcomes. To avoid data loss—and solely as control variables in these and subsequent path analyses—no healthcare access (111) and less healthcare utilization (109) have missing values replaced with 0, with the maximum number of substitutions in parentheses. Finally, whether in representing less knowledge of health issues or lower material resources, less education may potentially confound examined associations, and is therefore controlled.

In the final analytic stage, a path model is used to jointly test potential mediators linked to metabolic states in the “gross models” (Table 4), for their role in mediating men’s race differentials in these outcomes. Results for this “net model” are presented in Table 5. (To avoid analytic problems due to large variances, some variables (age, systolic and diastolic BP, and heart rate) are first divided by 10, solely in these analyses.) Final inferences about mediation are drawn from postestimation analyses of specific indirect effects—each

subsuming all paths leading from race to a distal metabolic outcome through a specific mediator (Muthén & Muthén, 1998–2010). The model also examines interdependencies between mechanisms, by specifying covariances/correlated residuals between the mediators as well as proximal controls from Table 4—thus avoiding assumptions about causal order. While this conservative strategy precludes statistical testing of indirect effects routed through multiple mechanisms, direct associations between mediators do yield tentative inferences about such pathways.

With the exception of the path model, all analyses are conducted with the STATA 12.1 statistical package (Stata Corp., 2011). Results are weighted using *svy* methods, first using population weights that adjust for the intentional oversampling of blacks and hispanics, and also incorporate a non-response adjustment based on age and urbanicity (O’Muirheartaigh & Smith, 2007). Standard errors are adjusted for sample stratification (sampling strata independently) and clustering (sampling individuals within each of 100 primary sampling units). Path analyses are conducted with the Mplus Version 6 statistical package (Muthén & Muthén, 1998–2010), also adjusting for sample stratification, clustering, and unequal probability of selection.

RESULTS

As noted, an extensive literature now documents inflammatory responses to antecedent and prolonged psychosocial stress. To further validate this linkage, preliminary age-adjusted analysis examines inflammation’s association with a current depressed state. In the results, inflammation is significantly higher among depressed men (Coeff. = 0.16, $p < 0.01$).

Race differentials: social ties, behaviors, biology

Table 3 contains age-adjusted OLS models for men’s race differences in posited social, behavioral, and biological mediators—as well as current metabolic states. Consistent with arguments about older black men’s greater social isolation, these men report significantly fewer network members. However, the proportion of these alters who are blood relatives is significantly higher among black than white men. In other words, the findings suggest that older black men have smaller networks rich in ties to those most likely to provide caregiving, emotional, and instrumental support. As with social factors, behavioral results only partly conform to expectations. Specifically, older black men are no more likely than their white counterparts to smoke regularly, and less likely to consume alcohol or binge drink—actively unhealthy behaviors that the biomedical literature links to metabolic problems. Underutilization of healthcare is also less common among black men. These men are, however, more likely to have passive unhealthy behaviors—physical inactivity and poor sleep habits.

Next, contrary to both media reports and some academic literature, there is no statistically significant black-white differential in older men’s obesity. Indeed, the direction of the effect suggests a “negative” differential, with black men thinner than their white counterparts. (Separate analysis, not shown, suggests similar results for BMI.) In contrast, older black men are significantly more likely to have chronic inflammation (with the magnitude of this association increasing, in supplementary analysis, with obesity controlled). Finally, as expected, all four distal metabolic outcomes—blood sugar control, systolic and diastolic blood pressure, and heart rate—are significantly worse among older black men. (As with inflammation, the magnitude of each of these effects also increases in separate analysis controlling obesity—further indicating a lack of mediation by this posited “gateway”).

Associations: mediator-mediator, mediator-outcome

Next, Table 4 presents results for models examining the associations of men's diabetic and cardiovascular states with the remaining potential mediators (inflammation, smaller networks, inactivity, and poor sleep habits), added one-at-a-time to control variables. As argued, these social, behavioral, and biological factors are all potentially interdependent—and may play either mediatory or confounding roles among themselves. Hence, the tables also examine such interdependencies. These models include some mediators as dependent and some as independent variables. This ordering is solely to simplify analysis and presentation, and does not reflect causal assumptions. As noted, subsequent path analysis more correctly specifies covariances/correlated residuals between mediators, entered as a block along with proximal controls.

Results (Table 4) are inconsistent with network size as a potential mediator. While a smaller network has a weak association with older men's physical inactivity, it is not linked to any distal metabolic outcome. Among behaviors, poor sleep habits and inactivity are both associated with more inflammation. Whether this linkage represents a causal effect, or a feedback from prolonged stress to these behaviors, remains unclear. Moreover, while sleep is not associated with any diabetic or cardiovascular outcome (and is thus arguably not a mediator), inactivity does have a positive linkage with a higher heart rate. In contrast, inflammation has consistent positive associations with all four metabolic outcomes.

Path analysis

As noted, in the final analytic stage, a path model jointly examines the role of the remaining potential mechanisms (inactivity, inflammation) in mediating men's race-metabolic linkages (Table 5). The model's Comparative Fit Index (CFI) and Tucker-Lewis Index (TLI) are 1.00, while the Root Mean Square Error of Approximation (RMSEA) is 0.00, suggesting excellent fit.

Findings suggest that much of the race effect passes through lifetime diabetes or hypertension. However, even net of these conditions as well as inactivity, inflammation is correlated with all four current distal metabolic states (Table 5). Postestimation analyses of specific indirect effects support conjectures about inflammation's mediatory role—with significant indirect effects leading from black men, through their inflammation, to blood sugar control (Coeff. = 0.003, $p < 0.05$), systolic (Coeff. = 0.03, $p < 0.10$) and diastolic BP (Coeff. = 0.02, $p < 0.05$), and heart rate (Coeff. = 0.05, $p < 0.01$). In contrast, inactivity does not mediate any of men's indirect race effects. However, as in Table 4, inactivity is directly associated with inflammation—suggesting that inflammation is either a confounder (indexing chronic stress) of the inactivity-heart rate linkage from Table 4, or a mediator through which inactivity has an indirect association with heart rate. (In separate path analysis excluding only inflammation, inactivity retains its significant direct association with heart rate (Coeff. = 0.10, $p < 0.05$), and also mediates a significant indirect race-effect on this outcome (Coeff. = 0.01, $p < 0.05$)—also suggesting that it is only the inclusion of inflammation that suppresses this linkage.)

DISCUSSION

This study began with four overlapping mechanisms for black-white differentials in older men's diabetic and cardiovascular outcomes: (1) deficits in social support and/or control; (2) poor health behaviors, potentially due to these social deficits; (3) obesity, possibly as a consequence of social and behavioral factors; and (4) inflammation, potentially induced by prolonged stress-exposure. Results were most uniformly consistent with the inflammation mechanism.

To recall, while the health benefits of social resources are well established, the literature yields competing arguments about men's race differences in these assets. Some studies suggest greater social isolation among black men—potentially leading to less support and caregiving, less monitoring and control of negative behaviors, and, ultimately, worse health outcomes. Others indicate their greater embeddedness in densely interconnected networks centered in the matriarchal kin group. Hypothesis 1 (Table 1) was included to test these conjectures. Results ran counter to the social-deficits argument. Consistent with Cornwell et al.'s (2008) combined-gender findings, black men had smaller social networks (Table 3). However, this factor did not have any significant linkages with metabolic problems (Table 4). Moreover, the proportion of blood kin (those most likely to provide caregiving as well as instrumental and emotional support) was in fact *greater* among black men than their white counterparts. It is possible, however, that the NSHAP data omitted important social precursors to metabolic problems, such as the quality (and hence support potential) of one's network ties.

Next, it was argued that whether due to social deficits, or cultural factors, older black men would have worse health behaviors, and that these behaviors would mediate their worse metabolic outcomes (Hypothesis 2). At least for the list of behaviors available in NSHAP, the results were generally inconsistent with these arguments. Older black men were no more likely than their white counterparts to smoke, and less likely to consume alcohol or binge drink (Table 3)—actively unhealthy behaviors that the biomedical literature strongly links to diabetic and cardiovascular problems. However, other passively unhealthy behaviors—physical inactivity and poor sleep habits—were more likely among black than white men, with both factors linked to chronic inflammation (Table 4). Poor sleep had no associations with either men's blood sugar control or their cardiovascular problems—while the “gross” association between inactivity and heart rate (Table 4) was suppressed in men's path results (Table 5) net of inflammation. Overall, then, contrary to both media accounts and academic literature, poor health behaviors do not seem to consistently mediate older black men's worse diabetic and cardiovascular health.

A third argument centered in obesity. To recall, both the sociological and biomedical literatures frequently portray obesity as the “biological gateway” through which black men's poor health behaviors lead to their worse metabolic outcomes (Hypothesis 3). It was argued that these assumptions implicitly represent a moralistic “culture of irresponsibility” model for race disparities in men's health. Findings (Table 3) ran completely contrary to this hypothesis—with older black men *less* likely than their white counterparts to be obese.

Rather than social or behavioral factors, or obesity, it was the fourth and least examined mechanism—inflammation, posited as an alternative “gateway” through which cumulative and multi-dimensional stress may induce biological weathering—that most consistently accounted for older black men's greater metabolic problems (Hypothesis 4). Not only were these men more likely to have chronic inflammation (Table 3), but in both the “gross” (Table 4) and path (Table 5) results, inflammation was the one mediator consistently associated with blood sugar control as well as all three cardiovascular outcomes. Moreover, analysis of specific indirect effects confirmed that inflammation significantly mediated men's black-white differentials in all four distal metabolic states. To recall, it was noted that some cross-sectional studies also link inflammation to obesity and specific health behaviors, with the causal direction unclear. It was argued, then, that inflammation represents a stress-induced weathering mechanism to the extent that its role in mediating race differentials in metabolic outcomes is not confounded or causally influenced by these factors. In the results, obesity was, as noted, (non-significantly) lower among older black than white men, and behaviors other than inactivity were either no higher among black men or had no associations with their diabetic or cardiovascular health. Hence, it is argued that the

pathways through which inflammation induces black men's worse metabolic states may not involve these factors. The only potential exception is inactivity. If, as noted above, the association between this factor and men's higher heart rate was mediated by inflammation, then the role of inflammation for this outcome was (at least partly) that of a behaviorally-induced mechanism, and not (only) a stress-metabolic "gateway." Alternately, inflammation may have confounded the linkage between heart rate and this passively unhealthy current behavior precisely in marking prolonged stress-exposure. Moreover, this potential behavioral-induction applied solely to a single metabolic outcome—with only inflammation playing a plausible mediatory role for black men's worse blood sugar control, and their higher systolic and diastolic BP. Along with the depression-inflammation associations noted above, this weight of evidence suggests that the inflammation results may indeed reflect the direct metabolic impact of older black men's greater long-term stress.

While this study provides important new information on the biosocial mechanisms behind a major set of ethnically stratified health issues in late life, it also has several limitations. First, the list of health behaviors examined—while based on current biomedical literature—could have missed important causal factors, with the same true of possible network precursors to metabolic issues. In the absence of direct measures, most also had to be crudely proxied through self-reports—leading to potential bias from race patterns in reporting. Next, efforts were made to properly conceptualize potential feedback pathways, and confounding—and to include relevant controls. However, the NSHAP data were cross-sectional, making it difficult to examine causal order. In particular, there remains a possibility of residual feedback from health states to posited mediators. Moreover, the social, behavioral, and biological mechanisms outlined above were potentially interrelated. All three sets of factors had possible autonomous linkages with metabolic outcomes. Additionally, social deficits could lead to obesity and inflammation through lower control of unhealthy behaviors, and to inflammation via less alleviation of chronic stress. Worse behaviors—whether active (smoking, drinking), or passive (inactivity, poor sleep)—could be associated with diabetic and cardiovascular health, as well as with obesity and inflammation. Finally, obesity had a potential biological linkage with inflammation. In other words, the role of each factor in accounting for black-white differentials in metabolic outcomes could either be mediated or confounded by the other mechanisms. It was thus vital to examine associations not just between single mechanisms and outcomes, but among mechanisms, as well as potential indirect pathways leading from race to metabolic pathologies via multiple mechanisms. While these cross-sectional analyses explored such interdependencies, and presented tentative inferences, comprehensive accounts of confounding and mediation must await longitudinal data. Perhaps most importantly, the web of interrelationships between social and metabolic pathologies has only begun to be examined—suggesting additional potential for confounding by unexplored pathways. The present study should thus be seen as a broad analysis that establishes baseline linkages and lays the groundwork for more detailed exploration.

CONCLUSION

Standard mechanisms in the literature do not consistently account for men's black-white differentials in diabetic and cardiovascular problems. Specifically, some academic studies as well as media accounts take an implicit "culture of irresponsibility" approach—implicating black men's greater social isolation, worse health behaviors, and obesity as the key factors behind their worse health outcomes. In contrast, findings from a nationally representative study of older Americans suggest that older black men are less obese than their white counterparts, have social networks rich in blood relatives, and their few unhealthier behaviors may not play a major role in their current metabolic health. 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 older black men's cumulative and multi-dimensional stress. In other words, these problems seem at least in part to be directly rooted in the systems of social stratification in which these men have lived their lives. As sociologists have long argued (Link & Phelan, 1995), reducing race disparities in health will require refocusing attention to these fundamental causes of disease.

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- This study queries the mechanisms underlying worse metabolic problems among black than white U.S. men ages 57 to 85.
- Contrary to literature, older black men's social isolation, poor health behaviors, or obesity may not play a role in these problems.
- The mechanism seems to be chronic inflammation, indexing the direct metabolic effects of cumulative and multi-dimensional stress.

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.

Table 2

Descriptive Statistics for Variables Used in Analyses.

Variable	Mean (SE)	Percentage	Range ^d	N
Race/ethnicity				
White (non-Hispanic) ^b	0.81 (0.02)	80.78%	0 – 1	1451
Black ^b	0.09 (0.01)	9.17%	0 – 1	1451
Hispanic/other ^b	0.10 (0.02)	10.04%	0 – 1	1451
Control variables				
Age ^c	67.55 (0.28)		57 – 85 (years)	1455
Less education ^d	2.26 (0.05)		1 – 4	1455
Diabetes ^b	0.22 (0.01)	21.51%	0 – 1	1455
Hypertension ^b	0.53 (0.02)	53.43%	0 – 1	1455
No healthcare access ^b	0.09 (0.01)	9.08%	0 – 1	1344
Depression				
Depressed ^b	0.16 (0.01)	15.92%	0 – 1	1436
Social deficits				
Fewer alters ^d	2.74 (0.05)		1 – 6	1455
Less close ties ^c	1.88 (0.01)		1 – 4	1409
Less network closure ^c	0.54 (0.01)		0 – 1	1410
Smaller kin proportion ^c	0.64 (0.01)		0 – 1	1411
Poor health behaviors				
Poor sleep ^b	0.39 (0.02)	38.96%	0 – 1	1450
Inactivity ^d	1.67 (0.03)		1 – 5	1453
Smoker ^b	0.21 (0.02)	20.98%	0 – 1	1185
More alcohol ^d	1.37 (0.07)		0 – 7	1452
Binge drinking ^d	0.38 (0.02)		0 – 2	1444
Less health care utilization ^d	4.52 (0.05)		0 – 7	1346
Biological mediators				
Obesity ^c	40.64 (0.20)		27.00 – 66.00 (inches)	1412
Inflammation ^{c,e}	0.89 (0.03)		0.00 – 2.25 (log scale)	879
Metabolic outcomes				
Blood sugar control ^c	1.80 (0.01)		1.44 – 2.65 (log scale)	839
Systolic BP ^c	137.13 (0.45)		85.00 – 212.67 (mm Hg)	1424
Diastolic BP ^c	80.45 (0.36)		44.50 – 127.67 (mm Hg)	1424
Heart rate ^c	70.50 (0.46)		40.00 – 133.33 (beats/min)	1422

Note: All values are specific to men. Italicization denotes reference category in subsequent analyses. All estimates are weighted to account for differential probabilities of selection and differential nonresponse. Design-based standard errors are given in parentheses. For dummy variables, both means and percentages are provided, as baseline information. Subsequent analyses use a standard 0/1 coding for these measures.

^aRange specific to men.

^bDummy variable.

^cContinuous variable.

^dOrdinal variable

^eObservations of CRP greater than 8.6 mg/l excluded from analysis.

Table 3

Associations of Race With Social, Behavioral, and Biological Factors Among U. S. Men Aged 57–85: Coefficients (Standard Errors).

	Social deficits				Poor health behaviors			
	Fewer alters ^a	Less close ties ^b	Less network closure ^b	Smaller kin proportion ^b	Poor sleep ^c	Inactivity ^a	Smoker ^c	More alcohol ^a
Race (ref: non-Hispanic White)								
Black	0.51 ^{**} (0.15)	−0.02 (0.04)	−0.05 (0.04)	−0.06 (0.04)	0.72 ^{**} (0.13)	0.47 [*] (0.19)	0.05 (0.24)	−0.50 [*] (0.21)
Hispanic/other	0.72 ^{**} (0.19)	0.00 (0.05)	−0.14 ^{**} (0.03)	−0.07 ⁺ (0.04)	−0.04 (0.21)	0.30 (0.21)	−0.27 (0.32)	−0.39 [*] (0.17)
	1451	1405	1406	1407	1446	1449	1181	1448
	Poor health behaviors (contd.)			Biological mediators		Metabolic outcomes		
	Binge drinking ^a	Less health care utilization ^{a,d}	Obesity ^b	Inflammation ^{b,e}	Blood sugar control ^b	Systolic BP ^b	Diastolic BP ^b	Heart rate ^b
Race (ref: non-Hispanic White)								
Black	−0.64 [*] (0.29)	−0.57 ^{**} (0.18)	−0.88 (0.55)	0.24 ^{**} (0.07)	0.12 [*] (0.05)	3.79 [*] (1.81)	2.06 [*] (0.83)	2.16 ⁺ (1.20)
Hispanic/other	0.18 (0.18)	0.17 (0.23)	−1.33 [*] (0.56)	−0.01 (0.06)	0.06 ⁺ (0.03)	−2.37 (1.59)	−0.71 (0.97)	1.50 (1.58)
	1440	1336	1408	878	838	1420	1420	1418

Note: All models control a participant's age. All estimates are weighted to account for differential probabilities of selection and differential nonresponse. Design-based standard errors are given in parentheses.

^a Ordinal outcome. Results are from ordinal logit regression model.

^b Continuous outcome. Results are from OLS regression model.

^c Dummy outcome. Results are from logistic regression model.

^d Lack of healthcare access controlled.

^e Observations of CRP greater than 8.6 mg/l excluded from analysis.

⁺ $p < .10$;

^{*} $p < .05$;

$p < .01$
**

Table 4

Associations Between Potential Mediators, and Between Mediators and Metabolic Outcomes, Among Men: Coefficients (Standard Errors).

Poor health behaviors		Biological mediator	Metabolic outcomes			
Poor sleep ^a	Inactivity ^b	Inflammation ^{c,d,e}	Blood sugar control ^{c,d}	Systolic BP ^{c,d}	Diastolic BP ^{c,d}	Heart rate ^{c,d}
Social deficits						
Fewer alters	-0.06 (0.05)	0.00 (0.02)	0.00	0.37 (0.42)	0.11 (0.21)	0.22 (0.30)
N	1446	878	838	1420	1420	1418
Poor health behaviors						
Poor sleep		0.11 ^{*f} (0.05)	-0.02 ^{+f} (0.01)	-0.27 ^f (1.39)	0.42 ^f (0.92)	0.00 ^f (1.05)
N		876	836	1417	1417	1415
Inactivity		0.08 ^{**f} (0.02)	0.00 ^f (0.01)	-0.85 ^{+f} (0.43)	-0.35 ^f (0.35)	0.75 ^{*f} (0.31)
N		877	837	1419	1419	1417
Biological mediator						
Inflammation			0.03 ^{**f} (0.01)	3.11 ^{*f} (1.27)	1.85 ^{*f} (0.84)	4.39 ^{**f} (1.06)
N			784	875	875	874

Note: Variables entered one-at-a-time into models controlling a participant's age and education. All estimates are weighted to account for differential probabilities of selection and differential nonresponse. Design-based standard errors are given in parentheses.

^aDummy outcome. Results are from logistic regression model.

^bOrdinal outcome. Results are from ordinal logit regression model.

^cContinuous outcome. Results are from OLS regression models.

^dAny lifetime diagnosis of diabetes or (separately) hypertension controlled.

^eObservations of CRP greater than 8.6 mg/l excluded from analysis.

^fLack of healthcare access and less health care utilization controlled.

⁺ $p < .10$;

Das

*
**

 $p < .05$
 $p < .01$

Table 5

Path Model: Coefficients (Standard Errors)

		Poor health behaviors	Biological mediator	Blood sugar control ^b	Metabolic outcomes		
		Inactivity ^a	Inflammation ^{b,c}		Systolic BP ^b	Diastolic BP ^b	Heart rate ^b
Race (ref: non-Hispanic White)							
Black	Direct effect	0.11 [*] (0.05)	0.09 ^{**} (0.03)	0.01 ⁺ (0.01)	0.14 (0.10)	0.15 ⁺ (0.08)	0.02 (0.07)
	Total effect	0.14 ^{**} (0.05)	0.11 ^{**} (0.03)	0.04 ^{**} (0.01)	0.19 [*] (0.08)	0.11 [*] (0.04)	0.10 ⁺ (0.05)
Hispanic/other	Direct effect	0.05 (0.07)	-0.03 (0.03)	0.02 (0.01)	-0.11 (0.08)	-0.02 (0.05)	0.05 (0.09)
	Total effect	0.09 (0.06)	-0.01 (0.03)	0.02 ⁺ (0.01)	-0.14 (0.09)	-0.05 (0.05)	0.07 (0.09)
Poor health behaviors							
Inactivity	Direct = total effect		0.12 ^{**} (0.02)	0.00 (0.01)	-0.18 ^{**} (0.07)	-0.07 ⁺ (0.04)	0.04 (0.04)
Biological mediator							
Inflammation	Direct = total effect			0.03 ^{**} (0.01)	0.30 ⁺ (0.16)	0.17 ⁺ (0.09)	0.45 ^{**} (0.09)

Note: Numbers are weighted coefficients. Sample size is 1455. The only exogenous control is a participant's age. Endogenous controls include less education, any lifetime diagnosis of diabetes or (separately) hypertension, no healthcare access, and less health care utilization. To condense presentation, only effects for theoretically-relevant paths are reported. Full results available on request. All estimates are weighted to account for differential probabilities of selection and differential nonresponse. Design-based standard errors are given in parentheses.

^a Ordinal variable

^b Continuous variable.

^c Observations of CRP greater than 8.6 mg/l excluded from analysis.

⁺ $p < .10$;

^{*} $p < .05$;

^{**} $p < .01$.