American Psychological Association

Developmental Psychology

0012-1649/18/$12.00

2018, Vol. 54, No. 10, 1993–2006
http://dx.doi.org/10.1037/dev0000511

Discrimination, Segregation, and Chronic Inflammation: Testing the Weathering Explanation for the Poor Health of Black Americans

Ronald L. Simons, Man-Kit Lei, and Steven R. H. Beach
University of Georgia

Ashley B. Barr
State University of New York at Buffalo

Leslie G. Simons
University of Georgia

Frederick X. Gibbons
University of Connecticut

Robert A. Philibert
University of Iowa

Several studies have reported a relation between race-related stressors and the poor health of Black Americans. Such findings raise questions regarding the mediating biological mechanisms that might account for this link. The present study investigated elevated systemic inflammation, a factor shown to be a strong predictor of chronic illness and mortality in all ethnic populations, as a possible factor. Using 7 waves of data from the Family and Community Health Study, collected over a 20-year period from over 400 Black Americans, we investigated the extent to which exposure to discrimination and segregation at various points in the life course predicted adult inflammation at age 28. Our analyses examined whether cumulative stress, stress generation, or predictive adaptive response (PAR) models best accounted for any associations that existed between these race-related stressors and adult inflammation. At every wave of data collection, assessments of discrimination and segregation were related to adult inflammation. However, multivariate analyses using structure equation modeling indicated that the PAR model best explained the effect of these race-related stressors on inflammation. Exposure to discrimination and segregation during the juvenile years predicted adult inflammation and amplified the inflammatory effect of adult exposure to these race-related stressors. These effects were considerably more robust than that of traditional health risk factors such as diet, exercise, smoking, and low SES. Implications of these findings are discussed, including the limitations of the widely accepted risk factor approach to increasing the health of Black Americans.

Keywords: discrimination, segregation, inflammation, health disparity, African American

Supplemental materials: http://dx.doi.org/10.1037/dev0000511.supp

Black Americans have greater prevalence and earlier onset of disability and chronic illness and a significantly lower life expectancy than other ethnic groups (Williams, 2012). For instance, Blacks have a 30% greater chance of dying from cardiovascular disease (Department of Health and Human Services, 2012) and a twofold greater risk of diabetes (Konen, Summerson, Bell, & Curtis, 1999). Chronic illness and disability place a heavy burden upon Black families and communities and it appears that matters are getting worse, not better (Geronimus & Thompson, 2004; Williams, 2012).

In large measure, medicine and public health use a risk factor approach to explain the poor health of Black Americans. This perspective considers the chronic illness and disability experienced by Blacks to be rooted in the various health-risk factors associated

Ronald L. Simons, Department of Sociology, University of Georgia; Man-Kit Lei, Center for Family Research, University of Georgia; Steven R. H. Beach, Department of Psychology, University of Georgia; Ashley B. Barr, Department of Sociology, State University of New York at Buffalo; Leslie G. Simons, Department of Sociology, University of Georgia; Frederick X. Gibbons, Department of Psychological Sciences, University of Connecticut; Robert A. Philibert, Department of Psychiatry, University of Iowa.

This work was supported by the National Heart, Lung, Blood Institute (R01 HL118045), the National Institute on Child Health and Human Development (R01 HD080749), the National Institute on Aging (R01 AG055393), the National Institute on Drug Abuse (R21 DA034457), and the National Institute of Mental Health (R01 MH62699, R01 MH62666). The content is solely the responsibility of the authors and does not necessarily represent the official views of the National Institutes of Health.

Correspondence concerning this article should be addressed to Ronald L. Simons, Department of Sociology, University of Georgia, 324 Baldwin Hall, Athens, GA 30606. E-mail: rsimons@uga.edu

with being poor (Geronimus & Thompson, 2004; Geronimus et al., 2016) such as an unhealthy diet, lack of exercise, smoking, and the stress of economic disadvantage (Simons et al., 2016). This perspective assumes that the remedy for the poor health of Black Americans is to improve their socioeconomic standing so that they have the resources and knowledge to make better choices. Although this strategy may work for Whites, it does not seem to eliminate the health disparities faced by Black Americans. Advanced education, for example, often leads to increased discrimination and race-related challenges (Pearson, 2008). And, affluence offers no protection from the constraints of segregation (Massey, 2017). Indeed, Blacks in the top quintile of the income distribution continue to experience much neighborhood crime and disorder as they live in areas that are more segregated than Hispanics in the bottom quintile (Intrator, Tannen, & Massey, 2016). Thus although increasing the socioeconomic status (SES) of Black Americans is a laudable goal for many reasons, this objective by itself fails to address many remaining race-related strains and challenges. Not surprisingly, middle and upper-class Blacks continue to manifest high rates of chronic illness and disability (Geronimus et al., 2016).

An alternative to the risk factor perspective is the "weathering hypothesis" developed by Geronimus and her colleagues (Geronimus, 1992; Geronimus, Hicken, Keene, & Bound, 2006; Geronimus & Thompson, 2004). They posit that the health inequality suffered by Blacks is a consequence of the cumulative impact of life in a society where they suffer social, economic, and political exclusion. Although health risk behaviors are seen as having some influence, the fundamental explanation for heath inequalities in the United States is a racial divide where Black Americans occupy a marginalized, stigmatized, subordinate status in relation to Whites. The weathering hypothesis views the elevated rates of illness and disability seen among Black Americans as a physiological response to the structural barriers and daily slights, stereotypes, and other threats to one's identity that comprise the Black experience (Geronimus & Thompson, 2004; Geronimus et al., 2016). Consistent with this hypothesis, several studies have reported a relation between discrimination and poor health among Black Americans (Williams & Mohammed, 2009). Such findings suggest that the health disparities suffered by Black Americans might better be labeled "health inequities" as they represent differences that are unnecessary, avoidable, and unfair (Braveman, 2006).

To date, however, there has been little attempt to compare the importance of weathering versus a simple SES/risk factor explanation for the poor health of Black Americans. Compelling support for the weathering argument requires evidence that (a) across the life course, race-related stressors trigger biological changes predictive of chronic illness and disability; (b) this association is maintained after controlling for traditional health risk factors such as SES, smoking, poor diet, and lack of health care; and (c) the impact of race-related stressors on pathological biological processes is greater than that of traditional health risk factors. Using chronic, systemic inflammation as an indicator of biological weathering, the current study uses longitudinal data from a sample of several hundred African Americans to investigate the extent to which these three conditions hold.

We begin by briefly reviewing the evidence regarding the link between inflammation and the development of chronic illness. We then present arguments for positing that race-related stressors such as racial discrimination likely lead to chronic inflammation. Finally, we review contradictory research findings regarding this proposition and describe our strategy for circumventing the problems that have beset past studies.

Inflammation and Chronic Illness

Elevations in circulating markers of inflammation (e.g., C-reactive protein [CRP](, interleukin [IL] 6 [IL6], tumor necrosis factor α [TNFα]) have been associated with cardiovascular disorders (CVD), type II diabetes, osteoporosis, rheumatoid arthritis, Alzheimer's disease, and most cancers (Franceschi & Campisi, 2014; Morrisette-Thomas et al., 2014). And, there is evidence that Blacks tend to have higher levels of inflammation than Whites (Paalani, Lee, Haddad, & Tonstad, 2011). Such findings point to the importance of investigating the link between racism and elevated inflammation as an avenue for understanding the higher prevalence of illness among Black Americans. Modern medicine's explanation for inflammation emphasizes the role of genes, exercise, diet, and unhealthy habits such as smoking. Although these factors are associated with elevated inflammatory levels, they leave the majority of variance left unexplained.

In recent years, behavioral scientists have documented the importance of social adversity as a predictor of inflammation (Browning, Cagney, & Iveniuk, 2012; Cole, 2014). Explanations for the link between adversity and inflammation tend to emphasize the way the immune system has evolved to address threatening conditions. Cole and colleagues (Cole, 2014; Slavich & Cole, 2013), for example, note that the immune system comprises two rather distinct programs: proinflammatory cytokine genes that combat tissue damage, bacteria, and other extracellular pathogens, and antiviral genes which produce antibodies and target intracellular pathogens such as viruses. They argue that adversity (threat or danger) leads to increased expression of the inflammatory program, coupled with decreased expression of the antiviral program, as the organism prepares for possible attack and injury.

Support for this idea comes from studies reporting a link between various types of social adversity and increased transcription of inflammatory genes (Slavich & Cole, 2013; Cole, 2014). Presumably this pattern of gene expression evolved to help adapt molecular physiology to the types of sporadic and transient threats that characterized our ancestral environments (Cole, 2014). In contemporary society, however, purely symbolic or anticipated threats undermine health by fostering chronic activation of the inflammatory program and risk for inflammation-related diseases such as CVD, diabetes, arthritis, neurodegeneration, and cancer while simultaneously downregulating the antiviral program and resistance to viral infections (Cole, 2014).

Discrimination and Inflammation

Arguments such as those proffered by Cole and his colleagues suggest that social environments that pose a persistent threat of hostility, denigration, and disrespect promote chronically high levels of inflammation. This is, of course, everyday life for members of ethnic minorities living in a racially charged society. In the United States, this is particularly the case for Black Americans (Carroll, 1998). Most Whites do not recognize the multitude of ways that they unknowingly typecast, patronize, or exclude stig-

matized minorities. As a consequence, persons of color often enter situations with uncertainty and vigilance regarding how they will be perceived and treated (Geronimus et al., 2016). In other words, the orientation that Cole and others have described as giving rise to inflammation (viz., vigilance and preparedness for threat) is likely a common focus among Black Americans as they deal with the cultural and structural challenges of a racialized society. To the extent that this is true, one would expect a robust association between Blacks' reports of racial discrimination and inflammation.

The potential connection between racial discrimination and inflammation has been investigated in several studies, however, and although some of these investigations find support for this association (Brody, Yu, Miller, & Chen, 2015; Lewis, Aiello, Leurgans, Kelly, & Barnes, 2010), many do not (Albert et al., 2008; Moody, Brown, Matthews, & Bromberger, 2014). In some instances, studies report a relation for Black women but not for Black men (Kershaw et al., 2016). And, one study found that greater exposure to discrimination was associated with lower, rather than higher, levels of inflammation (Cunningham et al., 2012). In large measure, these contradictory findings are probably a consequence of the different, and often problematic, measures that have been used (Williams & Mohammed, 2009).

First of all, studies show great variability in their approach to measuring discrimination. For example, researchers using data from the Dallas Heart Study (Albert et al., 2008) failed to find an association between discrimination and inflammation but their measure of discrimination consisted of a single item ("Have you ever been discriminated against because of your race/ethnic background?") with a dichotomous response format (yes/no). Others have used the Everyday Discrimination Scale (Williams, Yan Yu, Jackson, & Anderson, 1997). This nine-item scale asks about various types of mistreatment but does not inquire as to whether the slight or injustice was related to the respondent's race/ethnicity. Thus it is unclear whether this scale is assessing racial discrimination or mistreatment based on weight, social class, or some other social characteristic. Studies using this instrument have either failed to find an association between discrimination and inflammation (Moody et al., 2014) or report mixed findings (Kershaw et al., 2016).

Some studies attempt to assess lifetime exposure to discrimination using instruments such as the Lifetime Discrimination Scale (Williams et al., 1997). This instrument asks respondents to report whether they were ever treated unfairly (no/yes) in six domains of life (e.g., work, police, education, housing, neighborhood). Although this instrument would seem to be an improvement over many of the measures used in past research, it does not identify the age of exposure or the frequency of the discriminatory events, and it requires individuals to scan across their lives, introducing opportunity for substantial recall bias. Not surprisingly, Kershaw et al. (2016) reported that scores on this measure are not related to inflammation for either males of females.

Finally, virtually all studies of the association between race-related stressors and inflammation have used a self-report measure of exposure to discrimination. There is a need for studies that use additional approaches to assessing exposure to racism. Residing in a highly segregated community, for example, is a consequence of social systemic discriminatory practices (Massey, 2007), and, like overt acts of discrimination, it conveys contempt, disrespect, and unfair treatment. Unlike self-report measures of discrimination, however, segregation is usually assessed using Census data (Massey & Denton, 1988). Findings linking racist events to increased inflammation would be more compelling if the association was shown to hold across various types and methods of assessing racism.

There have also been problems in the way that past research has measured inflammation. In recent years a profusion of studies has investigated the potential link between social adversity and elevated inflammation. Albeit whether the adversity be racial discrimination or some other stressful condition, inflammation has almost always been assessed using a single marker, usually CRP or interleukin-6 (IL-6). The result has been very modest and often inconsistent associations between social variables, including racial discrimination, and inflammation. The inflammatory system, however, is extensive and complex; one inflammatory protein (cytokine) often stimulates and amplifies the production of others, setting up a cascade of reactions (Abbas, Lichtman, & Pillai, 2015). Further the system consists of both proinflammatory and anti-inflammatory factors, with the latter serving to regulate and limit the inflammatory process. A wealth of studies has indicated that it is the balance of pro- and anti-inflammatory cytokines that is crucial for health (Andargie & Ejara, 2015; Wojdasiewicz, Poniatowski, & Szukiewicz, 2014). Indeed, it has been suggested that most of the inconsistent and insignificant findings associated with research on inflammation is a consequence of using only one or two biomarkers to assess such a broad and complex biological system (Morrisette-Thomas et al., 2014).

Thus the contradictory findings that have been reported regarding a potential link between discrimination and elevated inflammation may be partially or perhaps largely a consequence of measurement problems. In support of this view, a recent study that used a more comprehensive measure of discrimination and a multibiomarker assessment of inflammation found a robust association between the two constructs (Brody et al., 2015). Still, even though this study avoided the measurement problems that have been described, it suffered from a limitation extant in most all research on discrimination and health. Past research on this topic has restricted its focus to a particular segment of the life course. Therefore, when a positive association is found, it is difficult to determine the manner in which discrimination exerts its effect. For example, is childhood or adult exposure to discrimination more consequential for health? Is cumulative exposure to racism more important for health than exposure during any particular stage of life? Does childhood exposure to discrimination enhance a person's response to such events as an adult? Such questions relate to the various life course models that have been proposed regarding the impact of social adversity on biological processes and health. Testing these models as they pertain to the effect of racism on inflammation requires longitudinal data covering various phases of the life course.

Life Course Models Linking Social Adversity and Health

Research regarding the effect of social adversity on health-related markers of biological dysregulation such as inflammation has been informed by three theoretical frameworks. Perhaps the most popular of these frameworks is the predictive adaptive response model (PAR). The PAR model views childhood and early

adolescence as sensitive periods, or stages of plasticity, during which cognitive and biological systems are programmed to prepare the organism for the future environment that is likely to be encountered (Miller, Chen, & Parker, 2011; Rickard & Lummaa, 2007). It is assumed that adverse conditions such as harsh or unpredictable family environments, provide cues about the severe circumstances that are likely to be faced throughout the life course (Ellis, Figueredo, Brumbach, & Schlomer, 2009). At the cognitive level, harsh environmental conditions are viewed as fostering a distrustful, vigilant orientation that prepares the person for anticipated threats (Miller et al., 2011). At the biological level, early adversity is thought to act as a programming agent that calibrates monocytes/macrophages to be more responsive to challenge (Miller et al., 2011).

The PAR model makes two predictions regarding the impact of early adversity on adult inflammation. First, it posits that exposure to childhood adversities will elevate adult levels of inflammation irrespective of subsequent risk exposure. The distrustful schemas and monocyte programming acquired during childhood are seen as fostering an elevated inflammatory response that continues unabated into adulthood. Second, harsh childhoods are seen as heightening sensitivity to subsequent stressors encountered across the life-course. Distrustful schemas prime individuals to perceive adult events as threatening and a hypersensitive immune system increases the probability that perceived threats will result in a strong inflammatory response. The result is an interaction effect whereby early stressors, given their impact on cognitive and biological programming, enhance the effect of adult stressors on level of inflammation.

A second model of the process whereby stress becomes biologically embedded as inflammation is the cumulative stress (conditional stimulus [CS]) model. Contrary to the PAR model, this perspective posits that individuals are at greatest physiological risk when they are exposed to persistent or cumulative stress across the life course (Gruenewald et al., 2012; Pollitt et al., 2008). Support for the CS model is provided by studies that find cumulative measures of SES across childhood and adulthood to be a stronger predictor of physiological risk than measures from single periods in the life course (Gruenewald et al., 2012; Loucks et al., 2010; Pollitt et al., 2008). A key assumption of the cumulative stress model is that childhood and adult stressors combine additively; they do not have interactive or multiplicative effects (Hostinar, Lachman, Mroczek, Seeman, & Miller, 2015).

Still a third framework is represented by the stress generation (SG) model. This perspective argues that it is level of stress during the preceding several months or years that best predicts physiological risk. Although childhood stressors may exert some effect on biomarkers such as inflammation, adult stressors are seen as having a more powerful effect (Gruenewald et al., 2012; Pearlin, Schieman, Fazio, & Meersman, 2005). A variant of the SG model is the argument that childhood adversity increases the probability of experiencing stressful conditions as an adult. This proliferation of stressors across the life course might account for the association between childhood experiences and adult health outcomes (Pearlin et al., 2005). When examined alone childhood stressors might be functioning as proxies for cumulative stress exposures thereby masking their explanatory effects. Stated in terms of a hypothesis, adult stressors would be expected to increase inflammation irrespective of childhood adversities and potentially operate as a mediating mechanism for childhood stressors. Any one of these models, or perhaps some combination of them, may describe the association between discrimination and elevated inflammation.

The Present Study

Using longitudinal data from a sample of several hundred Black respondents, the present study attempts to improve upon past research in several respects. First, we use a multiple-item index of racial discrimination that gets at various types of maltreatment, including insults and racial slurs, disrespectful treatment by community members, and mistreatment by fellow employees or the police. This instrument was administered at Wave 1, when the respondents averaged 10 years of age, through Wave 6, when they averaged 26 years of age.

In addition to this self-report index, level of neighborhood segregation is used as a second indicator of exposure to racism. At each wave of data collection segregation was assessed using U.S. Census data (Massey & Denton, 1988). More than half of all Black metropolitan residents live in neighborhoods that are highly segregated; indeed, no other group in the history of the United States has experienced the degree of segregation that has been forced upon Black Americans (Massey, 2017). Residential segregation is a consequence, in large measure, of institutionalized prejudice and discrimination in the real estate and banking industries, including practices such as profiling, redlining and selective marketing (Massey, 2007). Racial segregation in housing, in turn, leads to concentrated poverty and racial separations in job opportunities, educational resources, and social connections (Massey, 2007). Such social conditions, like the events on the self-report discrimination index, are likely to bolster feelings of mistreatment, anger, and distrust, with the result being increased risk for dysregulated physiological processes such as chronically elevated inflammation. Therefore, we expect self-reported discrimination and segregation to be related to inflammation in a similar fashion. And, to the extent that this is the case, we will combine the two instruments to form a composite measure of racist treatment.

Further, we attempt to improve on past research by utilizing a more comprehensive assessment of inflammation. At Wave 7, when they were roughly 28 years of age, the respondents were asked to contribute blood for purposes of assaying their inflammatory status. Roughly 92% agreed to do so. The blood samples were used to assay 14 inflammatory cytokines that were used to form an index of pro- and anti-inflammatory factors.

Finally, having several waves of data will allow us to investigate which theoretical model best explains the link between racism and inflammation. The PAR model will be supported to the extent that child and adolescent exposure to discrimination/segregation have a direct effect upon adult inflammation and serve to amplify the effect of adult discrimination/segregation on inflammation. The CS model will be supported if both childhood and adult exposure to discrimination/segregation have an independent effect on inflammation. And, lastly, the SG model will be supported if adult discrimination/segregation is a significant predictor of inflammation and any effect of discrimination/segregation during childhood or adolescence is indirect through the experience of such events during adulthood.

If the weathering perspective is correct, our analysis will demonstrate a significant effect of racial discrimination and segrega-

tion on inflammation through one or more avenues described by the three theoretical models. Support for the weathering perspective requires that these effects remain after controlling for social class (income, education) and health risk behaviors (smoking, heavy alcohol consumption, diet, exercise, and access to health care). Importantly, the weathering perspective not only asserts that discrimination and segregation will continue to evince an effect on inflammation after controlling for these factors, it also suggests that the impact of our two measures of racism will be more robust than the effect of SES and the traditional health risk behaviors. Indeed, based on arguments by Geronimus and her colleagues (Geronimus et al., 2016; Geronimus & Thompson, 2004; Pearson, 2008), there is reason to expect that SES measures such as income and education will have little or no association with inflammation, a finding quite different than what has been found for Whites (Marmot, 2015).

Finally, it should be noted that Geronimus et al. (2010) have posited that middle-age Black women are especially vulnerable to the weathering effect given that they often reside in communities with high rates of male unemployment and incarceration and must support multiple generations of dependents with resources provided by low income jobs (Burton & Whitfield, 2003). Our sample, however, is only 28 years of age and hence they have not yet encountered many of the challenges and stressors that they will have to endure during middle age. Thus, we do not anticipate finding gender differences in our models.

Method

Sample

Our research utilizes the seven waves of data that have been collected for the Family and Community Health Study (FACHS), a multisite (Georgia and Iowa) investigation of neighborhood and family processes that contribute to African American children's development in families living in a wide variety of community settings (see Gibbons, Gerrard, Cleveland, Wills, & Brody, 2004; Simons et al., 2002). The FACHS sample consists of several hundred African American families living in Georgia and Iowa at the initiation of the study. Each family included a child who was in 5th grade at the time of recruitment (see Gibbons et al., 2004; Simons et al., 2011). The first wave of the FACHS data were collected in 1997–1998 from 889 African American, fifth-grade children (467 from Iowa and 422 from Georgia), their primary caregiver, and a secondary caregiver when one was present in the home. Primary caregivers' mean age was 37, 93% were female, 84% were the target's biological mothers, and 44% identified themselves as single parents. Their educational backgrounds ranged from less than a high school diploma (19%) to a bachelor's or advanced degree (9%).

The second through sixth waves of data were collected when the target children were ages 12–13, 14–15, 18–19, 21–22, and 24–25, respectively. Of the 889 targets interviewed at Wave 1, 699 (78% of the original sample) were reinterviewed at Wave 6. In 2014–2015 when the targets were roughly 28 years of age, a seventh wave of data was collected that included blood draws. Given the logistics of scheduling visits by phlebotomists, only members of the sample residing in Georgia, Iowa, or a contiguous state were identified as eligible. After also excluding persons who were deceased, incarcerated, or otherwise unreachable, we were left with a pool of 479 individuals, 413 (86%) of whom agreed to be interviewed and to provide blood. Average education for these individuals was 13.1 years (9% < high school, 38% high school/GED, 18% vocational school, 24% 2–3 years of college, 14% college graduate, 2% graduate school). Median income was roughly $25,000 (30% < $13,000, 20% > $36,000, 9% > $52,000).

Analyses indicated that those individuals who did not participate in Waves 6 and 7 did not differ significantly from those who participated with regard to Wave 1 scores on caregivers' education, household income, family structure, or neighborhood characteristics. Compared to Wave 1, however, a higher percentage of those interviewed at Waves 6 and 7 were female.

Procedures

The protocol and all study procedures were approved by the University Institutional Review Board of the University of Georgia (Title: FACHS IV; Protocol # Study00000172). African American university students and community members served as field researchers served as interviewers. Questions were administered in the respondent's home using computer assisted interviewing (Gibbons et al., 2004; Simons et al., 2011). At Wave 7, participants were also asked to provide a blood sample. A certified phlebotomist drew five tubes of blood at each participant's home. Two of the tubes were spun immediately to separate serum into three cryo-vials that were then frozen and stored in a −80° freezer until used for the analyses described in the Measures section.

Measures

Elevated inflammation. Seventeen cytokines (e.g., IL-1, IL-6, TNFα) were assayed using a traditional enzyme-linked immunosorbent assay (ELISA; see online supplemental materials for details). Three cytokines were excluded as they were undetectable in most of the samples (≥95%). Of the remaining 14, three were anti-inflammatory and 10 proinflammatory. Using approaches recommended in the literature (Conraads et al., 2006), cytokines with no detectable values were coded as 1, those with values below the upper quartile were coded as 2, and those above or equal to this value were coded as 3. To capture the relative balance of proinflammatory to anti-inflammatory activity, the sum for the proinflammatory cytokines was divided by the sum of the anti-inflammatory cytokines. Using this ratio, higher scores indicated increased dominance of the inflammatory response. As expected, level of cytokines covaried across all pro and anti-inflammatory cytokines with an intraclass correlation between all cytokines used in the measure of .740.

Racial discrimination. At each wave of data collection, respondents completed 13 items from the Schedule of Racist Events (Landrine & Klonoff, 1996). This instrument has strong psychometric properties and has been used extensively in studies of African Americans (Brody et al., 2006; Burt, Simons, & Gibbons, 2012; Simons et al., 2002). The items assess the frequency, on a scale of 1 (*never*) to 4 (*several times*) with which various discriminatory events have been experienced. The items focused on being the victim of racial slurs, being hassled by the police, disrespectful treatment by sales clerks, false accusations by authority figures,

and exclusion from social activities because of being African American. At Wave 1, respondents were asked to report how often they had experienced each of these events in the past, whereas at Waves 2–7 they were asked to report how often they had experienced each of these events during the previous year. Coefficient alpha for the scale was above .75 at every wave.

Racial residential segregation. In Waves 1 through 3, racial residential segregation was assessed with the 2000 Census tract and county data, which was geocoded with participants' residential addresses in 1997, 1999, and 2001. At Waves 4 through 6, the measure was created using the tract-level and county-level data from the U.S. Census Bureau's American Community Survey 5-year estimate for 2006–2010, which was mapped onto the geocodes for our study participants' residential addresses in 2004, 2007, and 2011. Following Massey and Denton (1988), we assessed racial residential segregation using the widely accepted index of isolation (P_t). Isolation is defined as:

$$P_t = \sum_{i=1}^{n} \left[\left(\frac{x_i}{X} \right) \left(\frac{x_i}{t_i} \right) \right]$$

where n is the number of tracts in a county, x_i is the population of Blacks residing in census tract i, X is the total population of Blacks in a county, and t_i is the total population in census tract i. The index is based on pairwise comparisons and measures the distribution of non-Hispanic Blacks and Whites across tracts in a county. Scores range from 0 (*no segregation*) to 1 (*complete segregation*). Thus, a higher score indicates that Blacks have a greater probability of being isolated from other racial groups. It should be noted that we also performed our analyses using other indices for calculating segregation (e.g., dissimilarity index; Massey & Denton, 1988) and the pattern of results were always the same.

Control variables: Social class and health risk factors. Several statistical covariates that have been linked to health, race/ethnicity, and/or discrimination were included in order to minimize risk of confounding in the associations of interest. Gender is controlled in all analyses and is examined in exploratory analyses as a potential source of differential response. Demographic controls included level of education (8th grade thru post graduate study), age (in years), weekly income (in dollars) and work status (1 = employed). Respondents reported whether they were married or cohabiting with a romantic partner (no, yes). Respondents reported whether during the preceding three months they had suffered the symptoms of an illness such as the flu or a cold. This variable was included as acute infections foster temporary increases in inflammation. Health insurance was assessed by a single item that asked whether the respondent had health insurance (no, yes) at the time of the interview.

Respondents also reported on various health risk behaviors. They indicated whether during the prior 12 months they had smoked cigarettes (0 = *no*, 1 = *yes*). Responses to this item were summed across Waves 4–7 (ages 18 – 28). They also reported frequency of alcohol consumption, ranging from 0 (*never*) to 5 (*every day*) during the past year, and responses to this item were also summed across Waves 4–7. Healthy diet was assessed using two items that asked about frequency of fruit and vegetable consumption during the previous 7 days. The relationship between the two diet items was significant ($r > .25$ at each wave). Responses ranged from 1 (*none*) to 5 (*twice a day or more*) and were averaged to form the healthy diet variable. Again, scores were summed across Waves 4–7. Exercise was measured with two items: On how many of the past 7 days did you exercise or participate in physical activity for at least 30 min that made you breathe hard such as running or riding a bicycle fast? And, on how many of the past 7 days did you exercise or participate in physical activity for at least 30 min that did not make you breathe hard, but was still exercise such as fast walking, slow bicycling, skating, pushing a lawn mower, or doing active household chores? The response categories ranged from 1 (*0 days*) to 5 (*all 7 days*). These two items were correlated ($r > .45$ at each wave). Scores on the two items were averaged to obtain a score for each wave and scores were then summed across Waves 4–7.

Analytic Approach

We will construct a measure of juvenile discrimination by summing discrimination scores across Waves 1–3, and a measure of adult discrimination by summing discrimination scores across Waves 4–6. A similar approach will be used to form measures of juvenile and adult segregation. To test the predictions of the PAR, CS, and AS models, we will first run hierarchical regression models with robust standard errors using STATA 14.0 (StataCorp, 2015) to examine the unique effects of juvenile and adult exposure to racial discrimination on inflammation. Next, we will run a set of regression models to investigate the impact of juvenile and adult exposure to segregation on inflammation. Because missing data might influence our findings, we will use the last observation carried forward approach for imputing missing values. And, given that racial segregation is measured by the index of dissimilarity for the residential county, standard errors will be adjusted for clustering at the county level to avoid overestimating our results. To the extent to that the discrimination and segregation regressions produce similar results, we will use standardized scores to combine the instruments for each time period to form an index of juvenile exposure to racism and an index of adult exposure to racism.

The mediated-moderation model available in M*plus* 7.04 (Muthén & Muthén, 2015) will be used to examine the extent to which the impact of juvenile racism is indirect through adult racism, as well as whether the effect of adult racism on inflammation is amplified by juvenile racism. When interaction effects are present, we will examine simple slopes to interpret the results (Aiken & West, 1991). Finally, we will test for differences between the models for men and women using the multiple group analysis option in M*plus*. To assess goodness-of-fit, we will use Steiger's root mean square error of approximation (RMSEA; Browne & Cudeck, 1992) and the comparative fit index (CFI; Bentler, 1990). The CFI is truncated to range from 0 to 1 and values close to 1 indicate a very good fit (Bentler, 1990). An RMSEA smaller than .05 indicates a close fit; an RMSEA between.05 and .08 suggests a reasonable fit (Browne & Cudeck, 1992).

Results

We began by examining the zero-order correlations between both discrimination and segregation and adult inflammation at each wave of data collection (Waves 1–6). As shown in Table 1, assessments of discrimination and segregation are significantly

Table 1
Correlations of Racial Discrimination/Racial Segregation and Inflammation (n = 409)

Racism variables	*r*	*p* value
Racial discrimination		
Age 10	.135	.006
Age 13	.163	.001
Age 15	.174	.000
Age 18	.109	.027
Age 21	.137	.006
Age 24	.158	.001
Racial segregation		
Age 10	.190	.000
Age 13	.189	.000
Age 15	.241	.000
Age 18	.159	.001
Age 21	.188	.000
Age 24	.202	.000

related to adult inflammation at every wave. Next, to examine the various theoretical models that might account for these associations, we summed the discrimination scores and segregation scores across Waves 1–3 (ages 10–15) to obtain measures of juvenile discrimination and segregation, respectively. Scores were also summed across Waves 4–6 (ages 18–25) to obtain measures of discrimination and segregation during adulthood. Table 2 presents the means, standard deviations, and zero-order correlation matrix for these newly formed variables, inflammation, social class and health risk variables. The table shows that both juvenile and adult discrimination, as well as juvenile and adult segregation, are significantly associated with adult inflammation. The correlations range from .158 to .219. Further, as expected, there are significant associations between the discrimination and segregation measures, consonant with the idea that they are both indicators of exposure to race-based mistreatment. As expected, neither education nor income is related to inflammation, suggesting that increased SES has no health advantage for our sample. Income is not related to the discrimination or segregation variables, whereas education is positively related to segregation. The various health risk variables, although correlated with each other and in some cases with the discrimination and segregation measures, show little association with inflammation. The exception is acute illness and high alcohol consumption, both of which have been linked to elevated inflammation in prior studies. Contrary to expectation, however, exercise is positively related to inflammation and smoking shows no effect.

Table 3 shows the results of using hierarchical regression with robust standard errors to examine the effect of juvenile and adult discrimination and segregation on inflammation. Model 1A shows that juvenile discrimination is associated with inflammation ($\beta = .163$) after taking into account all of the various controls, and Model 1B shows that this is also the case for adult discrimination ($\beta = .143$). Model 1C presents the results of entering both juvenile and adult discrimination as predictors. When each of these variables is considered while taking into account the effect of the other, only juvenile discrimination has a significant effect ($\beta = .123$). The effect of adult discrimination does not even approach significance. Although the effect of sex approaches significance, none of the SES or health risk variables shows a significant association with inflammation.

Table 2
Correlations, Means, and Standard Deviations Among the Study Variables (n = 409)

Variable	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15
1. Inflammation	—														
2. Juvenile racial discrimination	.192**	—													
3. Adult racial discrimination	.158**	.576**	—												
4. Juvenile racial segregation	.219**	.279**	.289**	—											
5. Adult racial segregation	.199**	.240**	.251**	.866**	—										
6. Males	−.083†	−.018	.150**	−.106*	−.062	—									
7. Acute illness (age 28)	.121*	.199**	.255**	.253**	.243**	−.243**	—								
8. Education (age 28)	.045	−.008	.037	.099*	.116*	−.066	.064	—							
9. Income (age 28)	.050	−.041	.052	−.041	−.013	.100*	.037	.245**	—						
10. Married or cohabiting (age 28)	.065	−.061	.002	.054	.062	.007	.041	.076	.172**	—					
11. Health insurance (age 28)	.070	−.012	−.016	.070	.087†	−.120*	.156**	.211**	.141**	.057	—				
12. Healthy diet (ages 18–28)	.088†	.147**	.031	−.018	−.030	−.168**	.037	.154**	−.043	.025	.092†	—			
13. Exercise (ages 18–28)	.097*	.186**	.181**	.089†	.094†	.292**	.007	.138**	.104*	−.021	.040	.254**	—		
14. Alcoholic drinks (ages 18–28)	.085†	.206**	.304**	.242**	.233**	.164**	.202**	.076	.113*	−.046	.042	−.091†	.045	—	
15. Cigarette use (ages 18–28)	.046	.189**	.200**	.148**	.123*	.077	.072	−.295**	−.086†	−.064	−.060	−.114*	.024	.333**	—
M	3.737	21.474	21.268	.795	.802	.380	5.303	13.095	479.269	.259	.822	6.044	4.896	2.037	1.938
SD	.778	5.868	6.244	.179	.157	.486	6.404	1.755	509.659	.439	.383	1.650	1.433	.972	1.314

† $p \leq .10$. * $p \leq .05$. ** $p \leq .01$ (two-tailed tests).

Table 3
Regression Coefficients and Standard Errors for Racial Discrimination and Racial Segregation Regressed on Inflammation

	Racial discrimination						Racial resident segregation					
	Model 1A		Model 1B		Model 1C		Model 2A		Model 2B		Model 2C	
Predictors and controls	b	β	b	β	b	β	b	β	b	β	b	β
Juvenile racial discrimination	.020 (.007)**	.154			.016 (.008)*	.121						
Adult racial discrimination			.016 (.007)*	.130	.008 (.008)	.064						
Juvenile racial segregation							.798 (.270)**	.182			.696 (.291)*	.154
Adult racial segregation									.800 (.367)*	.162	.136 (.439)	.032
Males	−.158 (.090)†	−.099	−.198 (.091)*	−.124	−.175 (.091)†	−.109	−.118 (.068)†	−.081	−.137 (.066)*	−.092	−.119 (.067)†	−.082
Acute illness	.005 (.006)	.043	.004 (.007)	.035	.004 (.007)	.033	.005 (.003)	.037	.006 (.003)†	.039	.005 (.003)	.036
Education (age 28)	−.001 (.024)	−.001	−.003 (.024)	−.007	−.001 (.024)	−.003	−.009 (.018)	−.021	−.008 (.019)	−.021	−.009 (.018)	−.022
Income (age 28)	.001 (.001)	.035	.001 (.001)	.028	.001 (.001)	.034	.001 (.001)	.048	.001 (.001)	.043	.001 (.001)	.048
Married or cohabiting (age 28)	.124 (.088)	.070	.111 (.088)	.063	.120 (.088)	.068	.083 (.072)	.050	.086 (.071)	.051	.082 (.072)	.049
Health insurance (age 28)	.076 (.103)	.037	.076 (.103)	.037	.080 (.103)	.039	.038 (.116)	.027	.033 (.115)	.025	.037 (.116)	.027
Healthy diet (ages 18–28)	.014 (.025)	.029	.020 (.025)	.043	.014 (.025)	.030	.029 (.016)†	.062	.029 (.016)†	.062	.029 (.016)†	.063
Exercise (ages 18–28)	.045 (.030)	.083	.051 (.030)†	.094	.044 (.030)	.082	.045 (.021)*	.084	.048 (.021)*	.089	.045 (.021)*	.084
Alcoholic drinks (ages 18–28)	.042 (.044)	.052	.041 (.045)	.051	.036 (.045)	.045	.027 (.041)	.040	.033 (.042)	.047	.027 (.041)	.040
Cigarette use (ages 18–28)	.009 (.033)	.015	.012 (.033)	.020	.007 (.033)	.012	.009 (.041)	.017	.012 (.040)	.022	.009 (.042)	.018
Constant	2.806 (.358)**		2.901 (.355)**		2.775 (.360)**		2.678 (.377)**		2.650 (.413)**		2.655 (.403)**	
R-squared	.068		.061		.070		.075		.068		.075	

Note. Unstandardized (*b*) and standardized (β) coefficients shown with standard errors in parentheses; the standard error of racial segregation is adjusted for clustered at county-level ($n = 52$). $N = 409$.
† $p \leq .10$. * $p \leq .05$. ** $p \leq .01$ (two-tailed tests).

The impact of segregation on inflammation is investigated in Models 2A–2C in Table 3. Given that racial segregation was measured by the index of isolation for the residential county, standard errors were adjusted for clustering at the county level to avoid overestimating our results. Model 2A shows that juvenile segregation is associated with inflammation (β = .182) after taking into account the various controls, and Model 1B shows that this is also the case for adult segregation (β = .162). Consistent with the findings for discrimination, Model 1C indicates that only juvenile segregation has an effect (β = .154) on inflammation when both juvenile and adult segregation are in the model. Again, SES and the various risk factors show little or no impact on inflammation. These findings provide partial support for the PAR model in that they indicate that exposure to racism (whether assessed through discrimination or segregation) during late childhood/early adolescence directly predicts adult inflammation. The fact that neither exposure to discrimination nor exposure to segregation during adulthood demonstrated a significant effect once juvenile experiences were taken into account, is contrary to the predictions of both the CS and SG models.

Given that the discrimination and segregation measures were correlated and produced similar results (viz., juvenile experiences were more important than adult experiences in predicting inflammation), we combined the juvenile discrimination and segregation measures to obtain a composite measure of racist experiences during late childhood and adolescence. We did the same for our adult measures of discrimination and segregation. This approach had the advantages both of parsimony and of providing composite measures of juvenile and adult racism that included both self-report and census-based indicators.

Table 4 shows the results of using hierarchical regression with robust standard errors to examine the effect of juvenile and adult composite racism on inflammation. The results replicate those shown in Table 3. Model 3A shows that juvenile composite racism is associated with inflammation (β = .223) after taking into account all of the various controls, and Model 3B shows that this is also the case for adult discrimination (β = .200). Model 3C shows, however, that only juvenile composite racism is significant when both the juvenile and adult racism measures are in the model, and Model 3D shows that this pattern of findings remains the same when the various SES and health risk variables are added to the model. Model 3D also shows that none of the SES or health risk variables are significantly related to inflammation once the effect of the racism measures are taken into account. This indicates that these various controls are neither confounds nor mediators of the association between composite racism and inflammation. These findings support the weathering perspective's assertion that race-related stressors are more important predictors of the health of Black Americans than SES or traditional health risk behaviors.

Next, we used the mediated-moderation model available in M*plus* 7.04 (Muthén & Muthén, 2015) to further test the predictions of the various theoretical models. The structural equation model presented in Figure 1 depicts the effects of juvenile and adult racism, plus the interaction of these the two variables in predicting inflammation. The various fit indices indicate a good fit of the data. Consonant with the PAR model, juvenile racism has a direct effect on inflammation (β = .167) after taking into account the various controls, adult racism, and the interaction of juvenile and adult racism. Contrary to both the CS and SG models, adult racism is not significantly related to inflammation once the other variables are taken into account. Further, contrary to the SG model, there is no evidence that juvenile racism has an indirect effect on inflammation through its impact on adult racism. A test of this indirect effect using bootstrapping with 1,000 replications did not approach statistical significance.

Lastly, as predicted by the PAR model, Figure 1 shows that inflammation is predicted by the interaction of juvenile and adult racism (β = .096). As an aid to interpretation, Figure 2 presents a graph of this interaction. Based on the simple slope test (Aiken & West, 1991), the association between adult racism and inflammation is significant (β = .119, $p > .001$) for respondents exposed to high levels of racism as juveniles, whereas the relation between adult racism and inflammation does not approach significance (β = .016, $p = .758$) for those who experienced low levels of racism as juveniles. This pattern of findings supports the PAR model's assertion that childhood adversity calibrates the immune

Table 4
Regression Coefficients and Standard Errors for Composite Racism Regressed on Inflammation

	Model 3A		Model 3B		Model 3C		Model 3D	
Predictors and controls	b	β	b	β	b	β	b	β
Juvenile composite racism	.217** (.052)	.223			.177* (.076)	.181	.168* (.078)	.172
Adult composite racism			.197** (.053)	.200	.078 (.078)	.080	.069 (.080)	.070
Males	−.129 (.089)	−.080	−.180* (.089)	−.112	−.107 (.081)	−.067	−.142 (.091)	−.088
Acute illness	.003 (.006)	.022	.002 (.007)	.014	.003 (.006)	.029	.002 (.007)	.014
Education (age 28)	−.007 (.024)	−.015	−.010 (.024)	−.022			−.008 (.024)	−.019
Income (age 28)	.001 (.001)	.049	.001 (.001)	.039			.001 (.001)	.048
Married or cohabiting (age 28)	.104 (.087)	.058	.094 (.087)	.053			.099 (.087)	.056
Health insurance (age 28)	.070 (.102)	.034	.067 (.102)	.033			.070 (.102)	.034
Healthy diet (ages 18–28)	.020 (.025)	.042	.025 (.025)	.053			.021 (.025)	.045
Exercise (ages 18–28)	.036 (.030)	.067	.043 (.030)	.079			.036 (.030)	.066
Alcoholic drinks (ages 18–28)	.022 (.044)	.028	.023 (.045)	.029			.018 (.045)	.022
Cigarette use (ages 18–28)	.002 (.033)	.003	.006 (.033)	.010			.001 (.033)	.002
Constant	3.392** (.350)		3.393** (.354)		3.760** (.064)		3.430** (.353)	
R-squared	.087		.078		.074		.089	

Note. Unstandardized (*b*) and standardized (β) coefficients shown with robust standard errors in parentheses. $N = 409$.
* $p \leq .05$. ** $p \leq .01$ (two-tailed tests).

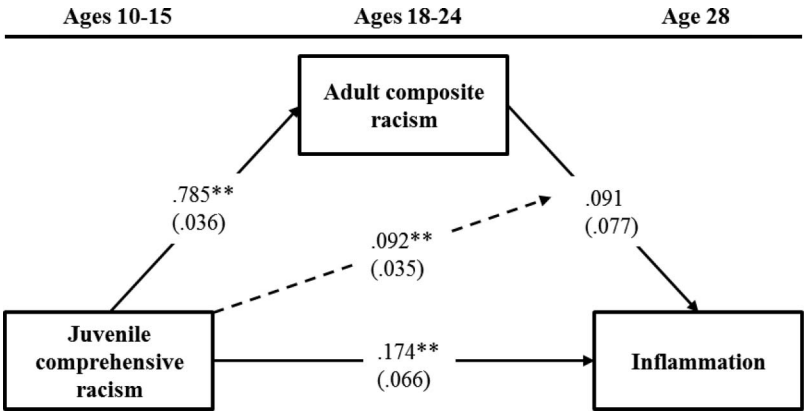

Figure 1. Mediated moderation of the relationship between juvenile and adult composite racism on inflammation. Chi-square = 104.396, *df* = 29, *p* = .000; comparative fit index = .965; root mean square error of approximation = .080. Values are standardized parameter estimates and standard errors are in parentheses. Gender, illness, education, income, married or cohabiting, health insurance, healthy diet, exercise, alcoholic drinking, and cigarette use are controlled. The standard error is adjusted for clustered. $N = 409$. ** $p \leq .01$ (two-tailed tests).

system to be more responsive to threatening events, thereby amplifying the effects of adult stressors on the inflammatory response.

Finally, we used the multiple group analysis option in M*plus* to examine the extent to which the findings presented in Figure 1 varied by sex. This procedure compares the chi square for each path when it is constrained to be equal for males and females versus when it is freed to differ. Table 5 shows that the change in chi square was not significant for any of the paths in our structural equation model. This is as expected given that the men and women in our sample are only in their late 20s. The gender differences described by Geronimus et al. (2010) would not be expected until the sample reaches middle age.

Discussion

It is well documented that Black Americans suffer from greater prevalence and earlier onset of chronic illness and disability than other ethnic groups (Williams, 2012). In large measure, medicine and public health have used a risk factor approach to explain the

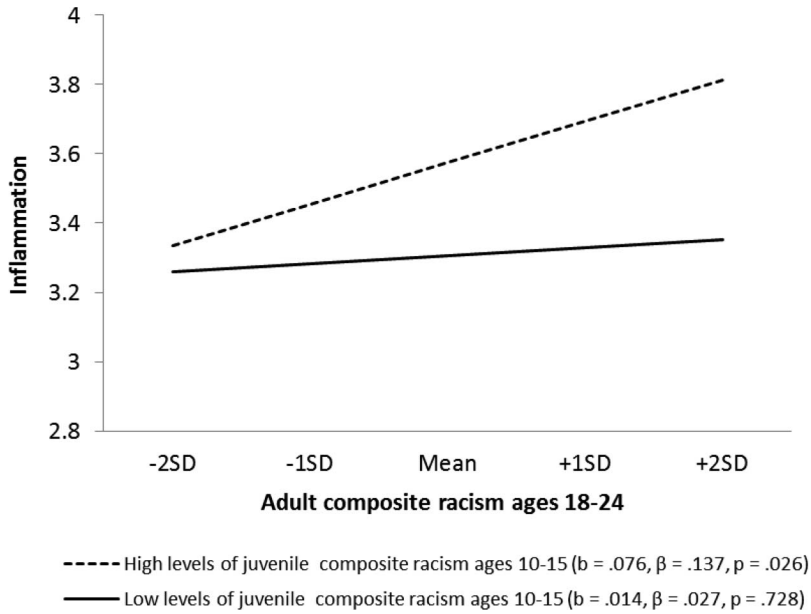

Figure 2. The effect of racial discrimination/segregation ages 18–24 on inflammation by levels of racial discrimination/segregation ages 10–15. The lines represent the regression lines for different levels of racial discrimination/segregation ages 10–15 (low: 1 *SD* below the mean; high: 1 *SD* above the mean). Numbers in parentheses refer to simple slopes.

Table 5
Comparison of the Paths for Males and Females

Paths	Δ Chi square	*df*	*p* value
Juvenile composite racism → Adult composite racism	2.550	1	.110
Juvenile composite racism → Inflammation	1.799	1	.180
Adult composite racism → Inflammation	.400	1	.527
Juvenile × Adult Composite Racism → Inflammation	.456	1	.499

poor heath of Black Americans. This perspective considers the chronic illness and disability experienced by Blacks to be rooted in the various health-risk factors associated with being poor such as an inadequate diet, lack of exercise, smoking, deficient health care, and economic hardship (Geronimus & Thompson, 2004; Geronimus et al., 2016). It is therefore assumed that the best remedy is to improve the socioeconomic standing of Black Americans so that they have the resources and knowledge to make better choices. Most of the research supporting the health-risk approach, however, is based upon White samples, and recent studies indicate that significant health inequities continue to exist between Blacks and other ethnic groups after adjusting for health risk factors and social status (Geronimus et al., 2006; Phelan & Link, 2015). Indeed, whereas social class is a robust predictor of better health among Whites, there is evidence that this relationship is weak or nonexistent for Blacks (Geronimus et al., 2016; Pearson, 2008). Such findings have led to speculation that in large measure the health inequities of Black Americans may be a consequence of race-related stressors. It is argued that the poor health of Blacks is rooted in the physiological weathering they experience in response to the chronic discrimination and marginalization of living in a race-conscious society (Geronimus & Thompson, 2004; Geronimus et al., 2006).

Recent research suggests that such weathering is likely to involve, at least in part, chronic, systemic inflammation. Inflammation tends to accrue with age (Morrisette-Thomas et al., 2014) and has been identified as a significant risk factor for virtually all of the chronic illnesses of old age including cardiovascular heart disease (CHD), diabetes, stroke, dementia, and cancer (Franceschi & Campisi, 2014). Further, studies have also linked inflammation to exposure to adverse social conditions such as loneliness, loneliness, low SES, bereavement, PTSD (Slavich & Cole, 2013). Indeed, increased inflammation appears to be the immune system's evolved response to a threatening environment (Cole, 2014). Given this biological wiring, one would expect the insults and mistreatment experienced by many Black Americans to increase their levels of inflammation. Consonant with this expectation, studies have reported that Blacks tend to have higher levels of inflammation than Whites (Chyu et al., 2011; Paalani et al., 2011). Contrary to this argument, however, research on the association between discrimination and inflammation has produced mixed results. Albeit, these studies have suffered from a variety of methodological problems that preclude drawing firm conclusions from their findings. Fortunately, the present study was able to overcome many of these limitations. We used a validated, multi-item index of discrimination, included a census variable measure of segregation, and we used longitudinal data that allowed us to test various competing life course models regarding the effect of racism on inflammation.

Our pattern of findings was the same whether self-report discrimination or community segregation was used to assess exposure to racism. Contrary to both the cumulative stress and the stress generation models, exposure to racism during the adult years was not related to inflammation once the effect of juvenile exposure was taken into account. On the other hand, juvenile exposure to racism showed a direct effect on adult levels of inflammation, even after controlling for a variety of health-related behaviors such as unhealthy diet, lack of exercise, and smoking. Further, we found evidence of an interaction effect where juvenile exposure to racism amplified the effect of exposure to racism during adulthood. That is, individuals who experienced a high degree of exposure during the juvenile years responded to adult racist events with increased inflammation. In contrast, exposure to racism as an adult had no significant effect on inflammation for those individuals who had experienced low levels of exposure to racism as a juvenile.

These results provide strong support for the predictive adaptive response model with its emphasis on early calibration of cognitive and biological systems during the sensitive periods of childhood and adolescence. This model posits that evolution has equipped humans to use juvenile experiences to predict the environmental conditions that they are likely to encounter throughout life. Thus a threatening childhood and adolescent milieu indicates a need to be vigilant and on guard, a perspective that fosters calibration of the immune system to a hypersensitive mode. This early programming is predicted to foster vigilance and elevated inflammation throughout the life course. Further, this early programming would be expected to amplify the effects of adult exposure to potentially racist events in two ways. First, a vigilant cognitive orientation would increase the probability that ambiguous stimuli might be perceived as racist and, second, a highly reactive immune system would display a robust inflammatory response when such perceptions occur (Miller et al., 2011). Conversely, adults who experienced minimal racism as children would be less likely as adults to display a chronic attitude of vigilance for discrimination and would respond with a less robust inflammatory response when such events occur.

These findings regarding the importance of early programming may explain why past studies have often failed to find an association between discrimination and inflammation. Most studies have investigated the impact of adult exposure to discrimination. Our findings suggest that it is juvenile exposure to racism that elevates an individual's risk for adult inflammation and that adult exposure to racist events has little effect upon the inflammatory levels of individuals who experienced minimal discrimination as children and adolescents.

In addition, our results supported the contention made by some that, contrary to the experience of Whites, increases in income and education often do little to improve the health of Blacks (Geroni-

mus et al., 2016; Pearson, 2008). Neither income nor education was related to inflammation in our analysis. Further, none of the traditional health risk behaviors were associated with adult inflammation. These findings lend credence to the contention that a medical/public health model that restricts its focus to increasing SES and reducing health risk behaviors is likely to have little impact on the ill health of Black Americans.

Past research has documented a status gradient for a wide variety of biomarkers of health (cortisol, cholesterol, blood pressure, CHD). The lower a person's social status, the worse their biomarkers. These gradations are evident in all countries, are less pronounced in countries with less inequality, remain after controlling for traditional health risk factors, and are even evident across the status hierarchies of other primates (Marmot, 2004, 2015; Marmot & Sapolsky, 2014). Given the consistency of these social gradient findings, it is not surprising that increases in social status lead to improved health for Whites. Blacks, however, face a different situation. They continue to be stigmatized and marginalized even when they achieve higher levels of SES. Regardless of achieved status, Blacks must deal with structural barriers and cultural stereotypes that degrade and discredit their social identities, with the result being biological responses such as chronic inflammation. Thus it would seem that any effective approach to addressing their poorer health needs to address their status problems in a racialized society.

Limitations

Although our study overcame many of the limitations of past research, it also suffered from certain shortcomings. First of all, our data set contains no measures of early childhood exposure to discrimination. Our first assessment of discrimination was at age 10. The PAR model emphasizes the importance of early programming and it may be that exposure to discriminatory treatment during the preschool or early elementary school years has an even bigger impact on adult inflammation than we were able to document using assessments from late childhood and early adolescence. A second limitation is that we were not able to link our assessment of inflammation to an increased risk for illness. At the last wave of data collection, our respondents were only 28 years of age and almost none of them had been diagnosed with a serious illness. It will be important for subsequent studies to establish that the effect of racial mistreatment on illness is mediated, at least in part, by inflammation.

Conclusions and Implications

Our study findings might be viewed as containing both good news and bad news. The good news is that minimal exposure to racist events during the juvenile years seems to promote a type of biological resilience to experiences of adult racism. At least, that is the pattern when inflammation is used as a biomarker of health risk. It may be that other markers of health will tell a different story. But, inflammation is an important marker and it appears to be stable in the face of racially based mistreatment during adulthood if the individual had limited exposure to such events during their formative years.

The bad news from our study is that early exposure to racism produces lasting effects on an individual's risk for elevated inflammation. Early exposure elevates inflammation during adulthood and increases the likelihood that any adult exposure will amplify inflammation still further. This finding suggests that "weathering" starts early in life and continues to exert a deleterious effect on health throughout the adult years. It may be, however, that an adult social environment characterized by high and consistent levels of nurturance and support might lead to recalibration and reduced levels of inflammation. Consistent with this observation, past research has shown that cognitive–behavioral stress management programs and gratitude exercises can reduce proinflammatory gene expression and/or reduce levels of inflammation in adults facing life adversities (Cole, 2014). This suggests that future studies should investigate the extent to which naturally occurring supportive relationships can counter the proinflammatory consequences of having faced early discrimination.

References

Abbas, B. K., Lichtman, A. H., & Pillai, S. (2015). *Cellular and molecular Immunology* (8th ed.). Philadelphia, PA: Elsevier.

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Newbury Park, CA: Sage.

Albert, M. A., Ravenell, J., Glynn, R. J., Khera, A., Halevy, N., & de Lemos, J. A. (2008). Cardiovascular risk indicators and perceived race/ethnic discrimination in the Dallas Heart Study. *American Heart Journal, 156,* 1103–1109. http://dx.doi.org/10.1016/ahj.2008.07.027

Andargie, T. E., & Ejara, E. D. (2015). Pro- and anti-inflammatory cytokines in visceral leishmaniasis. *Journal of Cell Science and Therapy, 6*(3), 1–8. http://dx.doi.org/10.4172/2157–7013.1000206

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological Bulletin, 107,* 238–246. http://dx.doi.org/10.1037/0033-2909.107.2.238

Braveman, P. (2006). Health disparities and health equity: Concepts and measurement. *Annual Review of Public Health, 27,* 167–194. http://dx.doi.org/10.1146/annurev.publhealth.27.021405.102103

Brody, G. H., Chen, Y.-F., Murry, V. M., Ge, X., Simons, R. L., Gibbons, F. X., . . . Cutrona, C. E. (2006). Perceived discrimination and the adjustment of African American youths: A five-year longitudinal analysis with contextual moderation effects. *Child Development, 77,* 1170–1189. http://dx.doi.org/10.1111/j.1467-8624.2006.009

Brody, G. H., Yu, T., Miller, G. E., & Chen, E. (2015). Discrimination, racial identity, and cytokine levels among African-American adolescents. *Journal of Adolescent Health, 56,* 496–501. http://dx.doi.org/10.1016/j.jadohealth.2015.01.017

Browne, M. W., & Cudeck, R. (1992). Alternative ways of assessing model fit. *Sociological Methods & Research, 21,* 230–258. http://dx.doi.org/10.1177/0049124192021002005

Browning, C. R., Cagney, K. A., & Iveniuk, J. (2012). Neighborhood stressors and cardiovascular health: Crime and C-reactive protein in Dallas, USA. *Social Science & Medicine, 75,* 1271–1279. http://dx.doi.org/10.1016/j.socscimed.2012.03.027

Burt, C. H., Simons, R. L., & Gibbons, F. X. (2012). Racial discrimination, ethnic-racial socialization, and crime: a micro-sociological model of risk and resilience. *American Sociological Review, 77,* 648–677. http://dx.doi.org/10.1177/0003122412448648

Burton, L. M., & Whitfield, K. E. (2003). Weathering towards poorer health in later life: Co-morbidity in urban low-income families. *The Public Policy and Aging Report, 13,* 13–18. http://dx.doi.org/10.1093/ppar/13.3.13

Carroll, G. (1998). Mundane extreme environmental stress and African American families: A case for recognizing different realities. *Journal of Comparative Family Studies, 29,* 271–284.

Chyu, L., & Upchurch, D. M. (2011). Racial and ethnic patterns of allostatic load among adult women in the United States: Findings from the National Health and Nutrition Examination Survey 1999–2004. *Journal of Women's Health, 20,* 575–583. http://dx.doi.org/10.1089/jwh.2010.2170

Cole, S. W. (2014). Human social genomics. *PLOS Genetics, 10*(8), e1004601. http://dx.doi.org/10.1371/journal.pgen.1004601

Conraads, V. M., Denollet, J., De Clerck, L. S., Stevens, W. J., Bridts, C., & Vrints, C. J. (2006). Type D personality is associated with increased levels of tumour necrosis factor (TNF)-α and TNF-α receptors in chronic heart failure. *International Journal of Cardiology, 113,* 34–38. http://dx.doi.org/10.1016/j.ijcard.2005.10.013

Cunningham, T. J., Seeman, T. E., Kawaschi, I., Gortmaker, S. L., Jacobs, D. R., Kiefe, C. I., & Berkman, L. F. (2012). Racial/ethnic and gender differences in the association between self-reported experiences of racial/ethnic discrimination and inflammation in the CARDIA Cohort of 4 US communities. *Social Science and Medicine, 75,* 922–931. http://dx.doi.org/10.1016/j.socscimed.2012.04.027

Department of Health and Human Services. (2012). *Heart disease and African Americans*. Washington, DC: U.S. Department of Health and Human Services. Retrieved from http://minorityhealth.hhs.gov/templates/content.aspx?lvl=2&lvlID=51&ID=3018

Ellis, B. J., Figueredo, A. J., Brumbach, B. H., & Schlomer, G. L. (2009). Fundamental dimensions of environmental risk: The impact of harsh versus unpredictable environments on the evolution and development of life history strategies. *Human Nature, 20,* 204–268. http://dx.doi.org/10.1007/s12110-009-9063-7

Franceschi, C., & Campisi, J. (2014). Chronic inflammation (inflammaging) and its potential contribution to age-associated diseases. *The Journals of Gerontology. Series A, Biological Sciences and Medical Sciences, 69*(Suppl. 1), S4–S9. http://dx.doi.org/10.1093/gerona/glu057

Geronimus, A. T. (1992). The weathering hypothesis and the health of African-American women and infants: Evidence and speculations. *Ethnicity and Disease, 2,* 207–221.

Geronimus, A. T., Hicken, M., Keene, D., & Bound, J. (2006). "Weathering" and age patterns of allostatic load scores among blacks and whites in the United States. *American Journal of Public Health, 96,* 826–833. http://dx.doi.org/10.2105/AJPH.2004.060749

Geronimus, A. T., Hicken, M. T., Pearson, J. A., Seashols, S. J., Brown, K. L., & Cruz, T. D. (2010). Do U.S. black women experience stress-related accelerated biological aging? *Human Nature, 21,* 19–38. http://dx.doi.org/10.1007/s12110-010-9078-0

Geronimus, A. T., James, S. A., Destin, M., Graham, L. A., Hatzenbuehler, M., Murphy, M., . . . Thompson, J. P. (2016). Jedi public health: Co-creating an identity-safe culture to promote health equity. *SSM-Population Health, 2,* 105–116. http://dx.doi.org/10.1016/j.ssmph.2016.02.008

Geronimus, A. T., & Thompson, J. P. (2004). To denigrate, ignore, or disrupt: Racial inequality in health and the impact of policy-induced breakdown of African American communities. *Du Bois Review, 1,* 247–279. http://dx.doi.org/10.1017/S1742058X04042031

Gibbons, F. X., Gerrard, M., Cleveland, M. J., Wills, T. A., & Brody, G. (2004). Perceived discrimination and substance use in African American parents and their children: A panel study. *Journal of Personality and Social Psychology, 86,* 517–529. http://dx.doi.org/10.1037/0022-3514.86.4.517

Gruenewald, T. L., Karlamangla, A. S., Hu, P., Stein-Merkin, S., Crandall, C., Koretz, B., & Seeman, T. E. (2012). History of socioeconomic disadvantage and allostatic load in later life. *Social Science & Medicine, 74,* 75–83. http://dx.doi.org/10.1016/j.socscimed.2011.09.037

Hostinar, C. E., Lachman, M. E., Mroczek, D. K., Seeman, T. E., & Miller, G. E. (2015). Additive contributions of childhood adversity and recent stressors to inflammation at midlife: Findings from the MIDUS study. *Developmental Psychology, 51,* 1630–1644. http://dx.doi.org/10.1037/dev0000049

Intrator, J., Tannen, J., & Massey, D. S. (2016). Segregation by race and income in the United States 1970–2010. *Social Science Research, 60,* 45–60. http://dx.doi.org/10.1016/j.ssresearch.2016.08.003

Kershaw, K. N., Lewis, T. T., Roux, A. V. D., Jenny, N. S., Liu, K., Penedo, F. J., & Carnethon, M. R. (2016). Self-reported experiences of discrimination and inflammation among men and women: The multi-ethnic study of atherosclerosis. *Health Psychology, 35,* 343–350. http://dx.doi.org/10.1037/hea0000331

Konen, J. C., Summerson, J. H., Bell, R. A., & Curtis, L. G. (1999). Racial differences in symptoms and complications in adults with type 2 diabetes mellitus. *Ethnicity & Health, 4,* 39–49. http://dx.doi.org/10.1080/13557859998182

Landrine, H., & Klonoff, E. A. (1996). The schedule of racist events: A measure of racial discrimination and a study of its negative physical and mental health consequences. *Journal of Black Psychology, 22,* 144–168. http://dx.doi.org/10.1177/00957984960222002

Lewis, T., Aiello, A. E., Leurgans, S., Kelly, J., & Barnes, L. L. (2010). Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. *Brain, Behavior, and Immunity, 24,* 438–443. http://dx.doi.org/10.1016/j.bbi.2009.11.011

Loucks, E. B., Pilote, L., Lynch, J. W., Richard, H., Almeida, N. D., Benjamin, E. J., & Murabito, J. M. (2010). Life course socioeconomic position is associated with inflammatory markers: The Framingham Offspring Study. *Social Science & Medicine, 71,* 187–195. http://dx.doi.org/10.1016/j.socscimed.2010.03.012

Marmot, M. G. (2004). *The status syndrome*. New York, NY: Henry Holt.

Marmot, M. G. (2015). *The health gap: The challenge of an unequal world*. New York, NY: Bloomsbury.

Marmot, M. G., & Sapolsky, R. (2014). Of baboons and men: Social circumstances, biology, and the social gradient of health. In M. Weinstein & M. A. Lane (Eds.), *Sociality, heirarchy, health: Comparative biodemogrphy—a collection of papers* (pp. 365–388). Washington, DC: National Academic Press.

Massey, D. S. (2007). *Categorically Unequal: The American Stratification System*. New York, NY: Russell Sage Foundation.

Massey, D. S. (2017). Why death haunts black lives. *Proceedings of the National Academy of Sciences of the United States of America, 114,* 800–802. http://dx.doi.org/10.1073/pnas.1620083114

Massey, D. S., & Denton, N. A. (1988). The dimensions of residential segregation. *Social Forces, 67,* 281–315. http://dx.doi.org/10.1093/sf/67.2.281

Miller, G. E., Chen, E., & Parker, K. J. (2011). Psychological stress in childhood and susceptibility to the chronic diseases of aging: Moving toward a model of behavioral and biological mechanisms. *Psychological Bulletin, 137,* 959–997. http://dx.doi.org/10.1037/a0024768

Moody, D. L. B., Brown, C., Matthews, K. A., & Bromberger, J. T. (2014). Everyday discrimination prospectively predicts inflammation across 7-years in racially diverse midlife women: Study of Women's Health across the Nation. *Journal of Social Issues, 70,* 298–314. http://dx.doi.org/10.1111/osi.12061

Morrisette-Thomas, V., Cohen, A. A., Fülöp, T., Riesco, É., Legault, V., Li, Q., . . . Ferrucci, L. (2014). Inflamm-aging does not simply reflect increases in pro-inflammatory markers. *Mechanisms of Ageing and Development, 139,* 49–57. http://dx.doi.org/10.1016/j.mad.2014.06.005

Muthén, L. K., & Muthén, B. O. (2015). *Mplus version 7.04: Base program and combination add-on*. Los Angeles, CA: Author.

Paalani, M., Lee, J. W., Haddad, E., & Tonstad, S. (2011). Determinants of inflammatory markers in a bi-ethnic population. *Ethnicity & Disease, 21,* 142–149.

Pearlin, L. I., Schieman, S., Fazio, E. M., & Meersman, S. C. (2005). Stress, health, and the life course: Some conceptual perspectives. *Jour-*

nal of Health and Social Behavior, 46, 205–219. http://dx.doi.org/10.1177/002214650504600206

Pearson, J. A. (2008). Can't buy me whiteness: New lessons from the Titanic on race, ethnicity, and health. *Du Bois Review, 5,* 27–48. http://dx.doi.org/10.1017/S1742058X0808003X

Phelan, J. C., & Link, B. G. (2015). I racism a fundamental cause of inequalities in health? *Annual Review of Sociology, 41,* 311–330. http://dx.doi.org/10.1146/annurev-soc-073014-112305

Pollitt, R. A., Kaufman, J. S., Rose, K. M., Diez-Roux, A. V., Zeng, D., & Heiss, G. (2008). Cumulative life course and adult socioeconomic status and markers of inflammation in adulthood. *Journal of Epidemiology and Community Health, 62,* 484–491. http://dx.doi.org/10.1136/jech.2006.054106

Rickard, I. J., & Lummaa, V. (2007). The predictive adaptive response and metabolic syndrome: Challenges for the hypothesis. *Trends in Endocrinology and Metabolism, 18,* 94–99. http://dx.doi.org/10.1016/j.tem.2007.02.004

Simons, R. L., Lei, M. K., Beach, S. R. H., Brody, G. H., Philibert, R. A., & Gibbons, F. X. (2011). Social environmental variation, plasticity genes, and aggression: Evidence for the differential susceptibility perspective. *American Sociological Review, 76,* 883–912. http://dx.doi.org/10.1177/0003122411427580

Simons, R. L., Lei, M. K., Beach, S. R., Philibert, R. A., Cutrona, C. E., Gibbons, F. X., & Barr, A. (2016). Economic hardship and biological weathering: The epigenetics of aging in a U.S. sample of black women. *Social Science & Medicine, 150,* 192–200. http://dx.doi.org/10.1016/j.socscimed.2015.12.001

Simons, R. L., Murry, V., McLoyd, V., Lin, K. H., Cutrona, C., & Conger, R. D. (2002). Discrimination, crime, ethnic identity, and parenting as correlates of depressive symptoms among African American children: A multilevel analysis. *Development and Psychopathology, 14,* 371–393. http://dx.doi.org/10.1017/S0954579402002109

Slavich, G. M., & Cole, S. W. (2013). The emerging field of human social genomics. *Clinical Psychological Science, 1,* 331–348. http://dx.doi.org/10.1177/2167702613478594

StataCorp. (2015). *Stata statistical software: Release 14*. College Station, TX: Author.

Williams, D. R. (2012). Miles to go before we sleep: Racial inequities in health. *Journal of Health and Social Behavior, 53,* 279–295. http://dx.doi.org/10.1177/0022146512455804

Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine, 32,* 20–47. http://dx.doi.org/10.1007/s10865-008-9185-0

Williams, D. R., Yan, Y. U., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socio-economic status, stress and discrimination. *Journal of Health Psychology, 2,* 335–351. http://dx.doi.org/10.1177/135910539700200305

Wojdasiewicz, P., Poniatowski, L. A., & Szukiewicz, D. (2014). The role of inflammatory and anti-inflammatory cytokines in the pathogenesis of osteoarthritis. *Mediators of Inflammation, 2014,* 1–19. http://dx.doi.org/10.1155/2014/561459

Received December 9, 2016
Revision received November 14, 2017
Accepted December 30, 2017 ■