HHS Public Access

Author manuscript

Am J Orthopsychiatry. Author manuscript; available in PMC 2019 January 22.

Published in final edited form as:

Am J Orthopsychiatry. 2018 ; 88(2): 151–160. doi:10.1037/ort0000301.

Gendered Race Modification of the Association between Chronic Stress and Depression Among Black and White US Adults

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Abstract

Background—Chronic stress stemming from social inequity has long been recognized as a risk factor for poor physical and psychological health, yet challenges remain in uncovering the mechanisms through which such exposures affect health outcomes and lead to racial and gender health disparities. Examination of sociocultural influences on group identity, coping, and the expression of stress may yield relevant insight into potential pathways of inequity's effect on risk for chronic disease.

Objective—To examine the relationship between chronic stress as measured by allostatic load (AL) and depression, by gendered race group.

Methods—Using NHANES 2005–2010 data, we included black and white US adults aged 18–64 years (n=6431). AL was calculated using 9 biomarkers; scores ≥4 indicated "high-risk". Depression was assessed using the PHQ-9; scores ≥10 indicated likely clinical depression. Logistic models estimated odds of depression as a function of AL for each gendered race group, adjusting for age and family poverty-to-income ratio. Effect modification was assessed by ANOVA and RERI.

Results—We observed modification on the multiplicative scale. High AL was more strongly associated with depression among white women (OR=2.1, 95% CI: 1.5, 3.0) and black men (OR=1.7 95% CI: 1.0, 2.9), than among black women (OR=1.1 95% CI: 0.6, 2.0) or white men (OR=1.4 95% CI: 0.8, 2.5).

Conclusions—A potential manifestation of high chronic stress burden, depression, differs across gendered race groups. These disparities may be due to group-specific coping strategies which are shaped by unequal social contexts.

Public policy implications—Strategies for coping with, and consequently pathological manifestation of, chronic stress may differ by race and gender as a result of social inequities, contributing to variation in disease burden across social groups. These results suggest a need for health policies targeted at mitigating group-level risk factors, such as socially-constructed identity, for maladaptive coping in addressing health disparities. Effective intervention on coping may enable greater resilience among disadvantaged groups in the context of persistent social inequity.

Keywords

chronic stress; allostatic load; depression; social inequity; culturally-influenced coping; African-Americans; European-Americans

Background

Chronic stressors such as poverty, discrimination, and other forms of social inequity have long been recognized as risk factors for poor physical and psychological health (Adler and Newman 2002; Pearlin et al. 1981; McLeod 2015). Yet, challenges remain in uncovering the mechanisms through which these exposures affect health outcomes and lead to racial and gender health disparities (Adler and Stewart 2010; Guo and Harris 2000). Contributing to this difficulty are methodological obstacles facing social and psychiatric epidemiology, in which traditional cause-effect models often fall short of accurately capturing the inter-dependency of individuals and groups within distinct psychosocial contexts (Kaufman and Cooper 1999). Such methods commonly fail to consider the dynamic influences of internal and external processes in affecting health behaviors and outcomes (Mezuk et al. 2013). The effect of chronic stress on behavior may also be partially enacted through an interaction between physiological and psychological stress responses, further complicating its measurement (Adler and Stewart 2010; Pearlin et al. 1981). Another complexity, which is the focus of this study, is variability in group responses to stress (Boyce and Ellis 2005) as a function of socially-constructed identity.

Frameworks for social disparities in health

Addressing concerns regarding theoretical limitations of health disparities research, the recently proposed Environmental Affordances (EA) Model (Mezuk et al. 2013) explicitly interrogates the interrelationships among social context, stress, health behaviors, and mental and physical health in clarifying the causes of health disparities. This framework suggests that examination of sociocultural influences on group differences in the expression of psychological distress may yield relevant insight into potential pathways of the effect of unequal social contexts on health outcomes. Evidence supporting a strong association between psychological trauma and chronic strain resulting from structural inequity (Chae, Lincoln, and Jackson 2011; Conrad and Barker 2010; Langner and Michael 1963; Meyer 2003) point to the potential role of social stratification in causing race, gender, and class disparities in mental health outcomes as an important area of investigation.

Demographic patterns in the expression of mental illness are well documented (Bresnahan et al. 2007; DiClemente et al. 2001; Kilpatrick et al. 2003; Kubrin and Wadsworth 2009; McLean et al. 2011; Odgers et al. 2008). Depression shows particularly clear disparities across socioeconomic, racial, and gender groups; extant literature supports a substantially greater proportion reporting depression in poor (Galea et al. 2007), white (Dunlop, Song, Lyons, Manheim, et al. 2003), and female (Nolen-Hoeksema et al. 1999) persons. Patterns of depression differ further by gendered race group; among black persons, the gender disparity in risk significantly attenuates when adjusting for socioeconomic status, while persisting among whites (Dunlop et al. 2003). As the literature offers compelling evidence for the

effect of stress on increased risk for psychological distress and disease (Langner and Michael 1963; Slavich and Irwin 2014), persistent patterns in psychopathology across race and gender offer a unique opportunity to investigate the nature of variability in group-level responses to stress. Understanding why these underlying demographic differences in responses to chronic stress occur can elucidate the mechanistic actions of social inequity on health (Adler and Newman 2002; Chae et al. 2011).

Social psychologists suggest that demographic patterns in stress-related psychopathology may be based partially in the role of socialized group identity in shaping self-appraisal (Franklin and Boyd-Frankling 2000; McCoy and Major 2003). Factors such as a highly centralized group identity which can increase the risk of experiencing an event or circumstance as stressful (Szymanski and Lewis 2016), and further shape the manner in which that stress is manifested (Dressler 1985; Ivanic, Overbeck, and Nunes 2011; Morrison, Plaut, and Ibara 2010), are likely related to the effect these factors have on perceptions of self-image and self-worth (Herman 1992). Race and gender identity feature strongly in both forms of self-appraisal (Cross and Madson 1997; Polce-Lynch et al. 2001; Twenge and Crocker 2002), and have been shown to significantly impact on the experience of stressful encounters and circumstances as well as the methods individuals employ to cope with them (Bosson et al. 2009; Bridges 2010). As socially-constructed categories that shape identity-defining perceptions, characteristics such as race or gender may therefore play a significant role in determining whether stress manifests predominantly in psychological, behavioral, or physical symptoms.

Studies have shown that black persons are more likely to display somatic rather than affective symptoms of depression (Ayalon and Young 2003). This disparity may be due in part to an effect of interacting racial and cultural identities on the manifestation of pathology (Brekke and Barrio 1997; Martin et al. 2013). These social identities are themselves subject to intersecting pressures of structural inequity; black persons' tendency to somaticize depression may stem directly from systemic oppressions which have forced the use of coping strategies that promote emotional fortitude but physical vulnerability within this group even as they remain as susceptible to psychological distress associated with chronic strain as other racial groups (Bridges 2010; Everett, Hall, and Hamilton-Mason 2010; Major et al. 1998; Major and O'Brien 2005; Kim 2014). Examples include hypervigilance (Mosely, Owen, Rostosky, and Reese 2016) as well as avoidant or numbing strategies such emotional eating and consumption of diets high in fat and sugar (Everett, Hall, and Hamilton-Mason 2010; Hayman, McIntyre, and Abbey 2015). In corroboration, one study identified gendered racial variance in the predictive value of socioeconomic stressors in risk for a number of health outcomes. For black women and men, education was not protective against high body mass index as was the case for white women and men. Likewise, income was protective for black and white women but not for men of either race (Assari, Nikahd, Malekahmadi, Lankarani, and Zamanian 2016).

In understanding the influence of socialized group identity on the manifestations of stress, consideration should also be given to the manner in which social inequity disparately informs dominant narratives of specific "gendered race" groups, a classification that captures the interdependency of race and gender in self-concept. An individual's self-appraisal is

concomitantly shaped by both gender and race, in addition to other identities; intersectionality theory (Crenshaw 1989) suggests that the individual contributions of one's race or gender identity to coping and, subsequently, health outcomes cannot be reasonably deciphered. Instead, causal investigation must address the unique psychosocial stressors experienced at the nexus of racial and gender identity in order to more accurately account for the role of social inequity in the distribution of disease across social groups.

Allostatic load as a measure of chronic stress

Given the group-specific nature of chronic stress stemming from social inequity, measuring stress exposure must be done in a manner that allows for comparison across groups. One method for operationalizing such complex exposures is through the use of allostatic load (AL), a physiological manifestation of "weathering" (Geronimus et al. 2006), as a proxy for chronic stress. Developed by McEwen (2003) and promoted by Cicchetti (2011), AL has been increasingly used by researchers as a measure for the cumulative effect of chronic stress on the body. Accounting for long-term physiological changes to neuroendocrine, cardiovascular, metabolic, and immune processes, AL provides a useful quantification of stress exposure that can be standardized across demographically dissimilar populations (Read and Grundy 2012). Neurological changes have been associated with high AL (McEwen 2003), linking physiological stress responses to adaptation in the neurological structures indicted in many psychiatric disorders, including PTSD and other anxiety disorders (McEwen 2003). These findings are consistent with others showing an association between AL and depression (Kobrosly et al. 2014). While the source of chronic stress may vary across gendered race groups, the cumulative effect of that stress over the lifetime can be quantified using AL, which allows for comparison across these groups.

Present study

The current analysis examines the associations between chronic stress, as operationalized in high allostatic load, and a potential health consequence of chronic stress, depression, within gendered race groups. Although race and gender (as well as sex) correlates of chronic stress, AL, and mental illness have been investigated, to our knowledge no study has examined modification of the association between AL and depression by gendered race group among US adults. We also explore theories suggesting a role for coping strategies developed in response to societal pressures in influencing these potential differences. We expect to observe a statistically significant association between AL and depression in all groups, but hypothesize that this association will be weakest among black women and men as a result of distinct coping mechanisms shaped by sociocultural factors.

Data and Methods

We used data from the National Health and Nutrition Examination Survey (NHANES), 2005–2010. Conducted by the National Center for Health Statistics, NHANES uses weighted samples to provide national estimates of health and nutritional status for the noninstitutionalized population of the United States. We selected all waves in which data on all biomarkers of interest and the depression assessment were available. NHANES data collection methodology has been described in detail elsewhere (Zipf et al. 2013).

Our analytic sample included 6,431 women and men aged 18–64 years who self-identified as non-Hispanic black or non-Hispanic white (referred to herein as “black” and “white”). Pregnant women were excluded, as pregnancy can affect biomarkers included in the allostatic load calculation (Gunderson 2009; Zamorski and Green 2001). Participants missing data on any of the nine biomarkers, depression screener, age, race, sex, or poverty-income ratio were excluded from the analysis.

Depressive symptoms

Participants completed the 9-item Patient Health Questionnaire (PHQ), a validated screen for depression (Kroenke and Spitzer 2002). Each question on this self-reported assessment of nine DSM-IV signs and symptoms of depression is scored from 0 (not at all) to 3 (nearly every day), with a total possible score of 27 calculated by summing the scores of the 9 individual questions. A total score of 10 or higher is considered indicative of clinical depression (Kroenke and Spitzer 2002).

Allostatic load

Consistent with previous research (Chyu and Upchurch 2011; Geronimus et al. 2006; Wexler Rainisch and Upchurch 2013), we used 9 biomarkers to assess allostatic load (AL). These include 3 cardiovascular biomarkers (systolic and diastolic blood pressure (BP), and pulse rate); 4 metabolic markers (glycosolated hemoglobin, body mass index (BMI), high-density lipoprotein (HDL) cholesterol, and total cholesterol); and 2 immunological markers (serum albumin and c-reactive protein (CRP)). Systolic and diastolic BP values were calculated as the average of three readings. Biomarkers with values above the 75th percentile of nationally-weighted empirical cutoffs were categorized as “high” and assigned 1 point each, with the exception of serum albumin and HDL cholesterol, which were assigned 1 point each for values below the 25th percentile empirical cutoff, as lower values of these biomarkers are considered indicative of poor physiological function. The decision to use quartiles was based on previous research indicating this as the preferred method (Geronimus et al. 2006; Wexler Rainisch and Upchurch 2013). “High” thresholds were: systolic BP 127.3 mmHG; diastolic BP >76 mmHG; pulse rate >82 bpm; glycosylated hemoglobin >5.7%, BMI >30.6; HDL cholesterol <42 mg/dL; total cholesterol >216 mg/dL; serum albumin <4.1 g/dL; and CRP >0.37 mg/dL. Following methods used in previous research (Geronimus et al. 2006), we also considered diagnosis of three chronic diseases in our AL score calculations. For participants with systolic or diastolic blood pressure, total cholesterol, and/or glycosolated hemoglobin values below high-risk thresholds, we assigned 1 point for each “yes” response to having ever been told by a doctor that they had high blood pressure, high cholesterol, or diabetes, respectively. AL scores were calculated as summation of individual biomarker points, with scores ranging from 0–9. Because previous literature (Juster et al. 2010) suggests an AL score of 3 or 4 as the threshold for differences in mortality and morbidity, we define AL scores of 4 and above as “high allostatic load”.

Covariates

The inclusion of age and family poverty-to-income ratio (PIR) as covariates in our analysis was based on prior literature showing associations of age and socioeconomic status with both depression (Kobrosly et al. 2014) and AL (Geronimus et al. 2006). The decision to

limit the number of covariates included in our models is consistent with avoidance of over-controlling for variables conceptually along a causal pathway (Kaufman and Cooper 1999). Age was stratified into 5 groups (18–24, 25–34, 35–44, 45–54, and 55–64 years) across which AL is known to vary (Kobrosly et al. 2014). PIR is an index for the ratio of household income to the federal poverty level based on family size and state of residence. NHANES provides PIR for each participant (Zipf et al. 2013). We stratified our analysis into 5 categories of PIR, "At or below (≤1x)", ">1 and ≤2x", ">2 and ≤3x", ">3 and ≤4x", and ">4x" the federal poverty threshold to better capture the distribution of our outcome across socioeconomic status.

Statistical analysis

All analyses were weighted to represent black and white women and men nationally following National Center for Health Statistics guidelines. Sample characteristics (age, family PIR) were reported by four gendered race groups: black women, white women, black men, and white men. A factorial ANOVA was conducted to compare the main effects of AL and gendered race and the interaction effect between AL and gendered race on depression. To determine which groups differed in risk of depression given high AL scores, we also conducted a pairwise comparison of marginal linear predictions using the Tukey method. To further assess the significance of this interaction, we estimated the relative excess risk due to the interaction (RERI) using the multivariate-adjusted odds ratios for each gendered race group, each compared to white men as a common referent group; RERI = $OR_{1j} - OR_{10} - OR_{0j} + 1$ where OR_{ij} is the odds ratio, i = AL risk (1 = high, 0 = low), and j = gendered race group (1 = black women, black men, or white women, 0 = white men) (Greenland et al. 2008). Corresponding confidence intervals were calculated using the delta method based on a Taylor series expansion (Hosmer and Lemeshow 1992; Rothman et al. 2008). RERI equals 0 if no interaction is present on the additive scale. Stratified logistic models were then used to estimate the probability of depression as a function of AL for each gendered race group. To test the robustness of our findings, we conducted sensitivity analyses with high AL cutoffs of ≥3 and ≥5, and an analysis using AL scores calculated without points assigned for reported hypertension, high cholesterol, or diabetes diagnoses. All analyses were conducted using STATA version 14 (StataCorp LP, College Station, Texas).

Results

Of the 14,050 participants aged 18–64 years in NHANES 2005–2010, we excluded participants whose reported race was not black or white (n=5,025), pregnant women (n=490), those missing information on depression (n=1,824), biomarkers used in the AL score calculations (n=2,530), and/or family PIR (n=1,120). All participants identified as male or female. Exclusions resulted in an analytic sample of 6,431 US adults, which represents approximately 113 million black and white women and men nationally.

Table 1 reports the proportion of US adults with depression and high AL scores by gendered race group, age, and family PIR. Black women were mostly likely to be depressed (15%) and to have a high AL score (32%), while white men were least likely to report depression (5%) or to have a high AL score (19%).

In unadjusted results, the association of AL with depression varied across gendered race groups [F (3, 6423) = 7.2, p <0.0001]. Among US adults with high AL, mean depression scores differed across several groups: black women vs. white men (contrast = 1.2, 95% CI: . 62, 1.8); black women vs. black men (contrast = 2.0, 95% CI: 1.3, 2.7); white women vs. white men (contrast = 1.8, 95% CI: 1.2, 2.3); and white women vs. black men (contrast = 2.5, 95% CI: 1.9, 3.2). There was not a statistically significant departure from additivity for any gendered race group relative to white men. The RERI statistic for black women was −0.5 (95% CI: −2.05, 1.07), for black men 0.11 (95% CI: −0.94, 1.17). While not statistically significant, results suggest the possibility of supra-additivity among white women (RERI=1.12, 95% CI: −0.02, 2.25), such that among white US adults, being a woman and having high AL may interact, resulting in higher risk of depression than the sum of risk due to either being female or having high AL alone.

After adjusting for age and PIR, black and white US adults with high AL had 1.7 times the odds of being depressed compared to those with low AL (95% CI: 1.3, 2.2; Table 2). High AL was associated with depression among white women (OR=2.1, 95% CI: 1.5, 3.0) and black men (OR=1.7, 95% CI: 1.0, 2.9), but not among black women (OR=1.1, 95% CI: 0.6, 2.0) or white men (OR=1.4, 95% CI: 0.8, 2.5).

To test the robustness of our findings, we conducted three sensitivity analyses. With a high AL score cutoff of ≥3 rather than ≥4, the association between AL and depression was similar among white participants (men, OR=1.9, 95% CI: 1.2, 2.6; women, OR=1.9, 95% CI: 1.3, 2.8), but stronger in black men (OR=1.8, 95% CI: 1.0, 3.2) than black women (OR=0.9, 95% CI: 0.6, 1.5). With a high AL score cutoff of ≥5, odds of depression as a function of AL were higher for all groups, but were not statistically significant among black women (OR=1.3 95% CI: 0.5, 2.3) or white men (OR=1.6 95% CI: 0.9, 3.1). Finally, determining high AL without including reported diabetes, high cholesterol, or high blood pressure diagnoses revealed a moderately lower odds of depression in relation to high AL among white women (1.6 vs. 2.1), and the relationship between chronic stress burden and depression became statistically insignificant among black men in addition to black women and white men (data not shown).

Discussion

We found evidence of racial and gender differences in the association between chronic stress and depression among US adults. Although black women were most likely to report depression and to have a high AL score, high AL was statistically significantly associated with depression only among white women and black men. We offer two relevant frameworks for interpreting these results. One, the Environmental Affordances (EA) model that considers the interaction of individuals with their social contexts, which posits that individual characteristics interact with environmental and societal-level factors to produce a range of health outcomes over time (Mezuk et al. 2013). Two, intersectionality theory, which describes the synergistic effects of intersecting axes of structured discrimination along race, gender, class, and sexual identities (Crenshaw 1989; Collins 2015). On the basis of these models, we offer three theories in accounting for our results: one, that subordinate social status increases vulnerability to internalizing symptoms of depression (Murphy et al. 1991;

Bandura, 1986). Two, that by having to cope with systemic racial discrimination in addition to gender inequity, black women may develop a form of psychological fortitude (Crocker 1999; Crocker and Major 1989) against the social and psychosocial factors that predispose women (Nolen-Hoeksema et al. 1999) and poor persons (Galea et al. 2007) to internalizing disorders under conditions of chronic stress. Finally, that structural racial and gender inequity interact to shape the expression of chronic stress such that symptom presentation may vary predictably according to both an individual's race and gender.

The first theory we explore, that subordinate status increases susceptibility to internalizing symptoms of depression, is consistent with extant literature supporting differential vulnerability to certain types of psychological distress as a driving factor behind gender differences in many psychopathologies (Kessler 1979; Weissman and Klerman 1977). Nolen-Hoeksema (1990) suggests considering how women's subordinate social status acts to increase susceptibility to internalizing disorders, including depression and PTSD. A strong evidence base has demonstrated that occupying a subordinate social status directly correlates with internalizing symptoms. Nolen-Hoeksema (1990) found that women's occupations, perceived societal and cultural expectations, as well as exposure to physical and sexual abuse increased their risk for rumination, somatic complaints, negative self-attribution, and feelings of helplessness--all symptoms characteristic of traditional depression conceptualizations. Our results are consonant with this theory; among both black and white participants, women were twice as likely as men to report depression.

If subordinate social status increases risk for internalizing symptoms of depression, those occupying racially subordinate status should also suffer exposure. Like women, racial minorities have been disempowered through systemic occupational discrimination, political exclusion, and physical violence. Such environments promote judgments of low-self efficacy indicated in persistence of depression (Bandura 1982). It stands to reason that black persons would be more likely to experience internalizing symptoms, a pattern ostensibly supported by our results; among men and women, black persons were more likely to report depression.

In addition to finding differential susceptibility to psychopathology across sex, Kessler (1979) also found differential exposure to chronic strain as the primary factor driving racial disparities in mental health. He argued that black persons in the U.S. were exposed to a greater amount of chronic strain than whites, results that have been confirmed by subsequent research (Jackson et al. 2010; Turner and Avison 2003; Troxel, Mathews, Bromberger, and Sutton-Tyrrell 2003). Although Kessler did not explore these racial differences by gender, the implications of such findings are unique for individuals subject to psychosocial pressures stemming from a combination of structural racial and gender inequity (Crenshaw 1989). In accordance with our findings, others have shown black women to have higher AL scores compared with black men and white women and men, even after adjusting for socioeconomic factors (Chyu and Upchurch 2011; Geronimus et al. 2006).

Higher AL and depression burden among black women suggest greater exposure to chronic stress requiring sustained coping; a lack of significant association between high AL and depression within this group suggests these coping strategies may be based in acquired sources of mental and emotional resilience. As indicated by an extensive body of literature

(Kessler and Neighbors 1986; Warheit et al. 1973), structured inequity may impact an individual's susceptibility to psychopathology in addition to increasing the risk for exposure to chronic stress associated with economic, gender, and racial inequity. Rather than increasing likelihood of psychological distress, however, we put forth the hypothesis that coping with a greater burden of chronic stress caused by structured racial and gender inequity--in addition to poverty--has forced black women to adapt psychologically. Veenstra (2011) found that "intersecting axes of inequality" can actually confer a mitigating effect on poor health, allowing some groups located at these social junctures to demonstrate better health outcomes than their individual race, gender, or class status would predict.

A key component to this theory is that this described psychological fortitude does not preclude black women from experiencing depressive symptoms, particularly those within the somatic domain (Ayalon et al. 2003). Indeed, some studies, as ours, have found that black women report higher rates of depressive symptoms than black men, white women, or white men before adjusting for socioeconomic factors (Dunlop et al. 2003; Mengesha and Ward 2012). Rather, what this proposed psychological resilience enables is an ability to redefine standards of value in a manner that challenges the socially-constructed subordinance of black and female persons, thereby potentially minimizing the likelihood of affective depressive symptoms, which have been shown to be associated with feelings of worthlessness and low-self-efficacy (Haeffel et al. 2008). Although black women are socially disempowered as a function of their race and gender and should therefore exhibit a proclivity for low self-efficacy and poor self-image, the intersection of these social identities may actually undermine the deleterious psychological effects associated with both. This hypothesis is grounded in previous research on stigma and self-esteem. Contrary to what many psychological theories predict, members of stigmatized groups tend to have comparable or higher levels of self-esteem as non-stigmatized groups (Crocker 1999; Crocker and Major 1989). The authors attribute these surprising findings to the use of self-protecting mechanisms by members of stigmatized groups: "A...mechanism by which members of stigmatized or oppressed groups may protect their self-esteem from negative feedback or negative comparisons with others…is by selectively devaluing, or regarding as less important for their self-definition, those performance dimensions on which they or their group fare(s) poorly, and selectively valuing those dimensions on which they or their group excel(s)" (Crocker and Major 1989:612–616).

Other studies have demonstrated similar results, finding that despite their marginal status, black persons tend to demonstrate higher levels of self-esteem and narcissistic tendencies than white persons (Twenge and Crocker 2002; Ziegler-Hill and Wallace 2011). Investigators have also identified differences in psychological symptoms among blacks with varying strategies for racism-related coping and levels of racial identification (Forsyth and Carter 2012), finding that in contexts of race-based stress, lower racial identification was associated with greater psychological distress. This evidence suggests that endorsement of a marginalized identity may be psychologically protective against the stress caused by such marginalization. Of particular relevance, Watson and Hunter (2015) demonstrated that indifference to stigma predicted lower levels of psychological distress in black women. It is therefore plausible that by enabling a greater sense of self-efficacy in which black women feel capable of determining for themselves standards against which their value will be

measured, discrimination against this “stigmatized” race and gender group may actually confer its members a measure of psychological protection against the need for out-group comparison in evaluating self-esteem and, consequently, the predominant adverse psychological effects of subordinate or “lesser” social status. For example, black women tend to express more body satisfaction rather than dissatisfaction and preference for larger body size compared with white women (Schooler et al. 2004; Webb, Looby, and Fults-McMurtery 2004). In thus recreating value, black women may enable themselves to retain a sense of self-efficacy and self-esteem even within a society that devalues characteristics of both their race and gender.

White men, white women, and black men, advantaged rather than disadvantaged by most current measures as a consequence of racial (white persons) and gender (males) inequity, have likely not been required to build the same psychological tolerance against the psychosocial pressures black women endure, and may experience what Kessler (1979) terms “psychic frailty” as a result. That is, these groups’ increased vulnerability to depression given chronic stress may stem from what is typically conceptualized as an “advantaged” social position: “If there are any variations in intrapsychic strengths, whites must be disadvantaged relative to nonwhites” (Kessler 1979). This psychological vulnerability Kessler observes may be due in part to the almost paradoxical nature of social privilege. Socially-constructed privilege derived from the dominant status of certain groups such as whites, males, and those of higher socioeconomic position may create a pathological dependence on comparative evaluation in building “positive” self-image for persons within these groups (Fein and Spencer 1997). As it relates to risk of depression, those who occupy the privileged social position of non-marginalization learn to rely on the elevated status these positions confer in evaluating self-efficacy and self-worth; contexts in which this positive self-image is threatened creates vulnerability to depressive symptomatology (Orth, Robins, and Roberts 2008; Assari and Lankarani, 2016a). Corroborating this theory, one study found stressful life events to be a better predictor of future major depressive episodes for men than women (Assari and Lankarani 2016b), while another demonstrated a greater role for stress in risk for depression among white men than black men (Assari and Lankarani 2016c).

Those who are both female and white, or male and non-white, occupy both subordinate and dominant social positions. It is possible that this experience of simultaneous disempowerment and privilege leads to an increased degree of identity-related psychological distress that impacts capacity for positive self-image. As part of either racially or gender dominant groups, white females and black males, in internalizing their multidimensional social status, learn to rely partially on constructed notions of value (i.e. the perceived inherent superiority of non-marginalized groups) in assessing their self-worth (Crocker 1999), especially in contexts in which their self-image is threatened (such as those of chronically stressful conditions). Yet, they are concomitantly ranked inferior according to these same social scales (Hughes and Demo 1989; Nolen-Hoeksema 1990). Because of this incongruence in the socially-constructed race and gender identities of white women and black men, as well as the prominent role these identities play in shaping both experiences of traumatic stress and coping strategies, it is likely that some similarities in patterns of chronic stress expression will be seen among these two groups, which may explain the current study’s findings. Within the current US social structure, one can both experience low self-

efficacy and poor self-image stemming from perceived subordinate status of black or female persons (Hughes and Demo 1989; Nolen-Hoeksema 1990) and higher self-efficacy and positive self-image associated with perceived superordinate social status of white or male persons (Buchanan and Selman 2008; Spence et al. 2010). This phenomenon may partially explain white women's and black men's increased risk for schizophrenia diagnoses compared with white men and black women, respectively (Metzl 2009), as well as why the association between AL and depression was statistically significant only within these two groups.

White males, however, are likely to have unique presentations of chronic stress exposure. Theoretically more at risk of psychological "frailty" than any of the other groups analyzed in this study because of lack of exposure to marginal status as a function of either race or gender, white men may be more likely to exhibit chronic stress through other forms of psychopathology. Although likely to suffer psychic frailty, white men hold both racially and gender "advantaged" social positions and are therefore less likely to be exposed to the detrimental effects of subordinate social status.

Strengths and limitations

To our knowledge, this is the first study to examine whether gendered race group modifies the relationship between chronic stress and depression among US adults, an important area of psychiatric epidemiology (McLeod 2015). A nationally representative sample allows us to estimate associations between black and white US adults. The use of AL as a measure of physiological burden allows for a standardized comparison of chronic stress across different racial and gender groups independent of chronic stress source, an important consideration given that disparate exposure to particular stressors makes comparison across these groups challenging. Further, AL is associated with several psychological and physical health conditions (McEwen, 2003; Kapczinski et al, 2008; Kobrosly et al, 2014), lending validity to our hypothesis of chronic illnesses as varied expressions of chronic stress. The use of a validated depression screener also strengthened our analysis; the PHQ-9 has strong reliability and validity among men and women (Kroenke et al. 2001), and across racial and ethnic groups (Huang et al. 2006).

Several important limitations also require acknowledgement. While the PHQ-9 has been validated within a range of racial and ethnic populations, and among men and women, the instrument assesses depression based on current symptoms. Those who suffer depression but whose symptoms are being successfully treated with medication or therapy may not be captured by the PHQ-9 even though they could hypothetically fall within our exposure group. For this reason, our estimates of the association between allostatic load and depression may be underestimated, especially among white women, who are most likely of the groups in this analysis to seek and undergo treatment for depression (Cooper et al. 2003; Möller-Leimkühler, 2002; Sussman et al. 1987). The use of cross-sectional data prevents us from assessing whether a high AL burden precedes depression within these groups. Our analysis was limited to NHANES 2005–2010 because the 2005–2006 surveys were the first to include the PHQ-9, and 2009–2010 was the latest wave to include all 9 biomarkers used to calculate AL. This limited sample size prevented further analysis of interactive effects

within stratified models. Research examining these associations among larger and more contemporary cohorts of US adults is therefore needed. An additional limitation of our study design is the process of selecting biomarkers for inclusion in AL score calculations, one limited by data availability and subject to measurement error as relevant biomarkers may be excluded. For example, no primary mediators such as cortisol measures (McEwen and Seeman 1999) were included in our AL composite score because of data availability. Still, secondary mediators (cardiovascular, metabolic, and immunologic biomarkers) have been demonstrated as defensible proxies for the primary stress processes thought to drive allostatic load (McEwen and Seeman 1999).

The number of participants excluded for missing data could cause concern about the representativeness of our sample. However, among those excluded due to missing data on AL biomarkers, missingness was distributed independently such that excluding one biomarker from the composite variable would not recover a significant number of respondents. Similarly, PIR missingness was approximately equally distributed across AL, race, and gender. Among participants excluded for missing depression scores, approximately 24% were black and 36% white. This missingness by race was distributed approximately equally across men and women, although not across income categories; participants excluded for missing information on depression had lower family poverty-to-income ratios.

Finally, our analysis is based on epidemiological data with limited information on the sociocultural and psychosocial factors that may account for gendered racial differences in the relation of allostatic load and depression. Future research in this area would be strengthened by the availability of data on coping strategies, experiences of discrimination, and the salience of racial and gender identity.

Conclusions

This study provides some insight into the pathways through which social and cultural factors perpetuate health disparities in chronic mental and physical disease. Future research should empirically examine the role of coping in mediating the relationship between chronic stress and the distribution of disease across gendered race groups, carefully considering how intersecting forces of structural inequity act to influence the types of psychological resources individuals draw from to manage social identity-based stress. In identifying socially-shaped methods for abiding chronic stress, public health policy and intervention can be tailored at promoting resilience through adaptive rather than maladaptive coping in a manner that considers the unique stressors individuals encounter as a function of their social group identity. Furthermore, by identifying certain diseases and disorders as socially and culturally-influenced manifestations of chronic stress, health practitioners may be better equipped to target the causes of chronic mental and physical illness rather than being limited to reducing the negative impact of the symptoms.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Partial support for Dr. Waring provided by NIH grant KL2TR000160.

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Table 1

Allostatic Load and Depression, Overall and by Gendered Race Group, Age, and Socioeconomic Status, among Black and White US Adults: National Health and Nutrition Examination Survey, 2005–2010, weighted % (SE)

	Depression[a]	High AL[b]
Total	7.3 (0.5)	20.4 (0.7)
Gendered race group		
White men	4.7 (0.6)	18.9 (1.0)
White women	8.6 (0.7)	19.4 (1.1)
Black men	7.1 (0.8)	25.1 (1.7)
Black women	14.6 (1.3)	32.0 (1.6)
Age, years		
18–24	5.6 (0.9)	4.5 (0.7)
25–34	6.1 (0.8)	10.0 (1.2)
35–44	7.4 (0.7)	20.4 (1.3)
45–54	9.3 (0.9)	25.1 (1.4)
55–64	6.4 (0.7)	35.0 (1.6)
Family PIR[c]		
>4	3.7 (0.5)	18.2 (1.1)
>3 and ≤4	4.1 (0.7)	18.6 (1.3)
>2 and ≤3	7.4 (0.9)	22.9 (2.2)
>1 and ≤2	12.4 (1.2)	23.0 (1.5)
≥1	17.9 (1.4)	24.8 (1.5)

[a] PHQ-9 score ≥10

[b] Allostatic load score of ≥4

[c] Ratio of household income to the US poverty threshold

Table 2

Depression in Relation to Allostatic Load by Gendered Race Group[a] among Black and White US Adults: National Health and Nutrition Examination Survey, 2005–2010

	All (n = 6431)	Black Women (n = 980)	White Women (n = 2147)	Black Men (n = 1028)	White Men (n = 2276)
Depression, % (SE)	7.3 (0.5)	14.6 (1.3)	8.5 (0.7)	7.1 (0.8)	4.9 (0.6)
Low AL[b] (0–3)	6.1 (0.5)	13.4 (1.4)	6.9 (0.7)	6.1 (0.9)	4.3 (0.6)
High AL (4–9)	11.8 (1.2)	17.1 (3.0)	15.3 (1.9)	10.1 (2.0)	7.4 (1.5)
Crude	**2.1 (1.6, 2.7)** ***	1.3 (0.8, 2.2)	**2.4 (1.7, 3.4)** ***	**1.7 (1.0, 3.0)** *	**1.8 (1.0, 3.0)** *
Adjusted[c]	**1.7 (1.3, 2.2)** ***	1.1 (0.6, 2.0)	**2.1 (1.5, 3.0)** ***	**1.7 (1.0, 2.9)** *	1.4 (0.8, 2.5)
Age, years					
18–24	ref	ref	ref	ref	ref
25–34	1.3 (.86, 2.1)	1.9 (.83, 4.3)	1.1 (.35, 3.0)	1.4 (.69, 3.0)	1.3 (.53, 3.0)
35–44	**1.7 (1.2, 2.4)** *	1.7 (.83, 3.6)	**2.2 (1.4, 3.6)** **	1.3(.51, 3.2)	.97 (.42, 2.2)
45–54	**2.4 (1.1, 4.1)** **	**2.7 (1.3, 5.5)** **	**2.3 (1.4, 3.9)** **	1.6 (.61, 4.3)	**2.8 (1.3, 5.8)** **
55–64	**1.5 (1.0, 2.3)** *	1.8 (.69, 4.6)	1.3 (.73, 2.4)	1.5 (.53, 4.2)	1.9 (.78, 4.6)
Family PIR[c]					
>4	ref	ref	ref	ref	ref
>3 and ≤4	1.1 (.84, 2.2)	1.3 (.57, 2.8)	.95 (.48, 1.9)	2.0 (.75, 5.2)	1.1 (.49, 2.5)
>2 and ≤3	**2.2 (1.5, 3.1)**	1.9 (.89, 3.8)	**1.8 (1.2, 2.7)** **	2.3 (.87, 6.2)	**2.7 (1.3, 5.5)** **
>1 and ≤2	**3.9 (2.7, 5.6)**	1.8 (.98, 3.4)	**4.4 (2.7, 7.1)** ***	**3.8 (1.3, 11.0)** ***	**3.9 (3.2, 6.7)** ***
≥1	**6.2 (4.6, 8.6)** ***	**3.4 (2.1, 5.7)** ***	**6.3 (4.1, 9.5)** ***	**4.8 (2.0, 11.7)** ***	**6.7 (4.1, 10.9)** ***

[a] Results are from five separate logistic regression models; one for the total sample and one for each gendered race group

[b] Allostatic load, calculated as a composite of nine cardiovascular, metabolic, and immune biomarkers

[c] Adjusted for five age groups and five groups of ratio of household income to the US poverty threshold, groups as defined in Table 1

* p<.05,

** p<.005,

*** p<.0001