Pathways from racial discrimination to cortisol/DHEA imbalance: Protective role of religious involvement

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Abstract

Objective: Racial discrimination (RD) is hypothesized to dysregulate the production of stress reactive hormones among African Americans. Psychological processes that may mediate the association between RD and such dysregulation (e.g., cortisol/DHEA ratio) are not well articulated. Organizational religious involvement (ORI) has been discussed as a psychological protective factor within the context of RD, but our understanding of ORI as a physiological protective factor remains limited. We evaluated whether RD was directly and indirectly (through depressive symptoms) associated with an imbalance of cortisol and DHEA hormones, and whether ORI buffered these direct and/or indirect pathways.

Design: Data were drawn from the Flint Adolescent Study, an ongoing interview study of youth that began in 1994. Participants were 188 African American emerging adults (47.3% Female, ages 20–22). We used mediation and moderated-mediation analyses, as outlined by Hayes (2012), to evaluate the study aims.

Results: We found that depressive symptoms mediated the association between RD and the cortisol/DHEA ratio. We also found that depressive symptoms mediated the association between RD and the cortisol/DHEA ratio for individuals reporting low and moderate levels of ORI, but not at high levels.

Conclusions: Our findings support the socio-psychobiological model of racism and health (Chae et al., 2011) and suggest that the psychological toll of RD can confer physiological consequences. Moreover, ORI may disrupt pathways from RD to cortisol/DHEA ratio by buffering the psychological toll of RD.

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Keywords

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Epidemiological research has firmly established that African Americans bear a disproportionate share of health problems (see Lewis et al., 2015). To understand the causes of racial disparities in health, scholars have pointed to racial discrimination (RD) as a psychosocial stressor that contributes to poor health among African Americans (see Williams & Mohammed, 2013). Guided by Chae and colleagues' (2011) socio-psychobiological conceptual model of racism and health, we posit that one mechanism through which RD may lead to poor health is through dysregulation of stress-reactive biological systems (e.g., the hypothalamic-pituitary-adrenal (HPA) axis; indexed by the glucocorticoid cortisol; Adam et al., 2015; Bergus & Sarnyai, 2015; Lee et al., 2017). Disrupted stress biology is proposed to underlie many health conditions (Cohen, Janicki-Deverts, & Miller, 2007), and scholars have reported that RD can disrupt the production of hormones such as cortisol and dehydroepiandrosterone (DHEA) that are involved in the stress process (Lucas et al., 2017; Ratner, Halim, & Amodio, 2013). To our knowledge, no study, has examined whether RD can contribute to an imbalance of multiple stress reactive hormones. Yet, imbalances in the cortisol/DHEA ratio have been associated with various mental (Sollberger & Ehlert, 2016) and physical health problems (Guder et al., 2007), suggesting that this type of dysregulated stress response might be one pathogenic pathway by which RD deteriorates health.

Despite these interesting findings, research on RD and stress reactive hormones is limited in two important ways. First, although research consistently reveals that chronic RD generates vulnerability for mental health problems (e.g., Hurd et al., 2014), and that mental health problems contribute to the imbalance of the cortisol/DHEA ratio (Sollberger & Ehlert, 2016), these associations have not been examined in the same study. Furthermore, evidence of the association between RD and biological stress regulation (i.e., cortisol, DHEA) has been mixed, with several researchers reporting no direct effect (Fuller-Rowell et al., 2012; Lee et al., 2017). We posit that one reason for the inconsistency in prior work is that the association between RD and dysregulation of biological stress indicators like the cortisol/DHEA ratio may become established through indirect psychological pathways, such as depressive symptoms. To better understand the risk and resilience processes, we propose to test this model in the current study.

The second limitation pertains to the limited number of studies that examine protective factors in pathways that link RD to physiological stress responses. Organizational religious involvement (hereafter we use ORI) has been identified as a key protective factor that can moderate the adverse psychological effects of RD among African Americans (e.g., Hope, Assari, Cole-Lewis, & Caldwell, 2017). Researchers have mostly theorized mechanisms by which ORI might offer physiological protection from RD (Assari et al., 2015). Scholars, for instance, posit that ORI may moderate the physiological consequences of RD by mitigating the psychological consequences of RD (Assari, 2013; Assari, 2014; Assari et al., 2015), but studies have not examined both processes in the same model. Thus, we do not know whether

ORI reduces the physiological toll of RD directly or by moderating the psychological influence of RD (see Figure 1 for conceptual model).

To address these gaps in the literature, our study entailed two aims. First, we investigated whether RD was directly and indirectly (through depressive symptoms) associated with an imbalance in the cortisol/DHEA ratio. Second, we explored whether ORI moderated the direct and indirect pathway from RD to the cortisol/DHEA ratio (see Figure 1).

Racial Discrimination and the HPA axis

RD, defined as "differential treatment on the basis of race that disadvantages a racial group" (National Research Council, 2004, p. 4), is a ubiquitous stressor among African Americans. With over 90% of African American emerging adults reporting multiple RD experiences in a given year (e.g., race-related microaggressions), researchers have consistently found that RD exacts psychological and physiological tolls (Lewis et al., 2015). Chae and colleagues (2011) have proposed a socio-psychobiological framework of racial health disparities, in which RD can set in motion psychological, behavioral, and biological vulnerabilities that, in combination, can accelerate health deterioration. Among African Americans, RD exposure has been linked with poorer mental health outcomes (Hurd, Varner, Caldwell, & Zimmerman, 2014), higher allostatic load (Geronimus, 2006), shorter telomere length (Lee, Kim, & Neblett, 2016), dysregulation of the HPA axis (Lee et al., 2017), and poorer health behaviors (Jackson, Knight, & Rafferty, 2010). RD, to this end, may contribute substantially to racial disparities in rates of disease morbidity, comorbidity, severity, and mortality (Williams & Mohammed, 2013).

A strong conceptual and empirical case exists for examining the HPA axis as a biological pathway by which RD deteriorates health among African Americans. RD experiences during adolescence and emerging adulthood have been linked to dampened awakening response, diurnal rhythm, and circulating levels of cortisol among African Americans (Adam et al., 2015; Bergus & Sarnyai, 2015; Lee et al., 2017). Although acute stressors have been associated with increases in circulating cortisol levels, scholars have pointed to the *attenuation hypothesi*s (see Susman, 2006) to explain why cortisol levels are attenuated within the context of RD (Lee et al., 2017). The attenuation hypothesis contends that the HPA axis downregulates cortisol production following chronic stress to limit the body's exposure to cortisol (Susman, 2006). Since RD experiences are chronic among African Americans (Lee et al., 2017), it comes as no surprise that scholars have largely documented blunted cortisol production in the context of RD exposure.

RD-induced alterations to cortisol production may be better understood when considered in conjunction with DHEA. DHEA, a hormone that is synchronously released with cortisol from the adrenal cortex, is known to antagonize the adverse physiological effects of cortisol (Kaminska, Harris, Gijsbers, & Dubrovsky, 2000). Scholars have traditionally theorized that elevated cortisol and attenuated DHEA production (i.e., higher cortisol/DHEA ratio) increases the body's exposure to unopposed cortisol, thereby increasing risk for physical health problems such as acute stroke (Blum et al., 2013). While higher cortisol/DHEA ratios have generally been observed among individuals experiencing chronic stress (e.g.,

Moriguchi Jeckel et al., 2010) and major depressive disorder (Sollberger & Ehlert, 2016), studies also found lower cortisol/DHEA ratios to be associated with diagnoses of depression and post-traumatic stress disorder (Gill, Vythilingam, & Page, 2008; Yehuda et al., 2006). Drawing from the attenuation hypothesis (Susman, 2006), Karmin and Kertes (2017) argued that the HPA axis may downregulate cortisol production in the context of prolonged stress but upregulate DHEA to better meet the physiological demands of the chronic stressor(s). As RD is a pervasive and prevalent stressor in the lives of African Americans, it may dysregulate the HPA axis by creating an imbalance in circulating levels of cortisol and DHEA that over time can contribute to poorer health.

Depressive Symptoms as a Mediating Pathway

RD invokes poorer psychological well-being and physiological health among African Americans (Berger & Sarnyai, 2015). Chae and colleagues (2011) suggested that RD can generate psychological risks, which can, in turn, promote biological vulnerabilities (e.g., dysregulation of the HPA axis). To date, a few researchers have elucidated psychological factors that mediate the effect of RD on biological outcomes such as telomere length (Chae et al., 2015; Lee et al., 2017) and cortisol concentration (Lee et al., 2017). For example, Lee and colleagues (2017) found that RD indirectly, but not directly, influenced cortisol concentrations through anxiety symptoms. Psychological reactions to RD, to this end, may influence the regulation of biological systems engaged in the stress response such as the HPA axis (Berger & Sarnyai, 2015).

Embracing this line of thinking, two separate bodies of research point to the role of depressive symptoms specifically as a mediator of the link between RD and the cortisol/DHEA ratio. First, cross-sectional and longitudinal studies have documented a positive association between RD and depressive symptoms among African Americans (Hurd et al., 2014). Second, depressive symptoms have been associated with an imbalance in the cortisol/DHEA ratio (Sollberger & Ehlert, 2016). Considered within Chae's socio-psychobiological model of racism and health (2011), these studies suggest that depressive symptoms may mediate the association between RD and an imbalance of the cortisol/DHEA ratio.

Organizational Religious Involvement as a Protective Factor

Although psychological pathways from RD to physiological outcomes have been documented (Lee et al., 2017), no study, to date, has examined the role of protective factors within these mechanisms. organizational religious involvement (ORI), defined as "…public, institutional forms of religious involvement [e.g., service attendance]," has been consistently documented as a psychological protective factor for African Americans contending with racism-related stress (Mattis & Watson, 2009 for review). Guided by the risk and resilience model (Begun, 1993), ORI (e.g.,service attendance) has been found to mitigate the psychological consequences of RD among African Americans (Bierman, 2006; Ellison, Musick, & Henderson, 2008; Ellison et al., 2017; Hope et al., 2017; Lee, Neblett, & Jackson, 2015). Ellison and colleagues (2017), for instance, found that service attendance buffered the adverse effects of RD on mental health outcomes and life satisfaction. Bierman (2006) similarly found that religious service attendance was psychologically protective for

African Americans, but not White Americans. Moreover, religious social support offered by fellow congregants reduced the influence of RD on psychiatric disorders, suggesting that ORI may promote resilience by extending religious social support.

Scholars have proposed multiple mechanisms through which ORI offers psychological protection within the context of RD. Most notably, researchers have identified service attendance and organized religious activities (e.g., choir, volunteerism) as psychologically salutary for African Americans by increasing opportunities for social support (Hope et al., 2017; Mattis & Watson, 2009). Given the centrality of religion in the lives of African Americans, predominantly African Americans faith-based communities are fertile ground for receiving social and emotional support within the context of RD (Hope et al., 2017). For example, African Americans who access predominantly Black religious communities may be uniquely positioned to receive empathy and emotional validation from leaders and fellow co-religionists (Mattis & Watons, 2009). In addition to emotional support, one might surmise that religious social support promotes social assets such as mentorship (Cook, 2000) and mental health counseling from a religious leader (Krause, 2003) that underlies the psychologically protective role of ORI in the context of RD. Mentorship, for example, has been associated with less mental health problems and risk behaviors (e.g., substance use, sexual risk behaviors) over time among Black youth (Hurd & Zimmerman, 2010). Relational support structures in predominantly Black faith-based communities, to this end, may buffer the mental health consequences of RD (Hope et al., 2017).

ORI may also increase exposure to religious teachings that emphasize liberationist theology (Mattis et al., 2003) and activities that promote volunteerism and civic engagement (Hope & Spencer), which, in turn, promotes psychological well-being within the context of RD. For instance, exposure to liberationist theology may reinforce beliefs that RD experiences are not without meaning and that the divine is able to exact social, political, and economic justice to members of oppressed groups (Mattis et al., 2003). This belief, in turn, may cultivate greater optimism and diminish the mental health consequences of RD (Lee et al., 2015). Hope and Spencer (2017) have also noted that African Americans are more likely to spend time in volunteer and civic engagement activities, which may promote positive mental health outcomes in the context of RD. Thus, ORI may both enhance mental health among African Americans burdened by RD and offset the psychological pathways linking RD to its physiological toll.

Although considerable evidence points to ORI as a psychologically protective factor in the presence of RD, research examining ORI as a physiologically protective factor is limited. In a few studies, ORI has been associated with better physiological functioning in the context of stress (Hill, Bradshaw, & Burdette, 2016; Hill, Rote, Ellison, & Burdette, 2014). For example, Hill and colleagues (2016) noted that religious involvement may increase social resources, promote health behaviors, improve psychological functioning, and decrease stress exposure to mitigate allostatic load – such as, neuroendocrine functioning – within the context of stress (e.g, RD stress). Researchers have not, however, tested whether ORI reduces the physiological consequences of RD. Thus, it is important to test whether ORI shields against physiological imbalance by first reducing the psychological toll of RD (i.e., indirect effect moderation). It is also plausible, although limited in evidence (Cooper et al.,

2013), that ORI may directly mitigate the physiological effect of RD (i.e., direct effect moderation; see Figure 1).

Current Study

To advance our understanding of risk and resilience in the context of RD, our first aim was to investigate whether depressive symptoms mediated the association between RD and the cortisol/DHEA ratio. We hypothesized that RD exposure would increase depressive symptoms, which, in turn, would associate with imbalances between cortisol and DHEA. Second, in light of past studies on ORI as a protective factor (e.g., Hope et al., 2017), we tested whether ORI directly or indirectly moderated the above mediational pathway (see Figure 1).

Method

Participants

The sample for this study included 188 African Americans from an ongoing longitudinal study of school-dropout and alcohol/substance use (Zimmerman & Schmeelk-Cone, 2003). The original sample consisted of 850 ninth graders enrolled in one of four public high schools in a large, Midwestern city. To be eligible for the original study, participants needed to have an 8th grade GPA of 3.0 or lower and no diagnosis of an emotional disorder or a developmental disability. Participants in the present study were assessed at the sixth wave (2001; M_{age} = 20.98, SD_{age} = 0.64) and were included if they had complete cortisol and DHEA data (see Table 1). Pregnant participants and participants with a salivary blood protein contamination level greater than or equal to 3 mg/dL were excluded (n = 10).

Procedure

Our study was approved by the University of Michigan Institutional Review Board. Participants completed face-to-face interviews for approximately 60 minutes by trained interviewers in the participants' home or in the community setting (e.g., public library). Following the interview, participants were self-administered a paper-and-pencil questionnaire that included questions about demographics, RD experiences, and health behaviors. Additional methodological details can be found elsewhere (Zimmerman & Schmeelk-Cone, 2003).

Measures

Cortisol and DHEA.—Saliva samples were collected at three designated time-points during the wave 6 interviews. Saliva was only collected from participants who reported not eating, drinking, or using tobacco in the hour prior to study onset. Following consent procedures, participants were instructed to rinse their mouth with water. For each saliva collection, participants pooled saliva in their mouth for one minute and then expectorated through a straw into a cryotube. Participants were interviewed between 7:10 AM and 4:35 PM (M_{time} = 1:43 PM, SD = 2 hours, 59 minutes) and saliva samples were collected approximately 10-, 32-, and 62-minutes following consent. Immediately following

collection, the saliva samples were placed on ice and refrigerated, then transported to a −80 degree Fahrenheit freezer for storage.

Cortisol and DHEA were determined by high sensitivity salivary cortisol and salivary DHEA enzyme immunoassays (Salimetrics, State College, PA). Saliva samples were thawed and centrifuged at 3,000 rpm for 15 minutes prior to assay, and the assays followed standard enzyme immunoassay procedures (see Granger, Cicchetti, Rogosch, Hibel, & Flores, 2007; Klimes-Dougan et al., 2001). Saliva samples were assayed for cortisol in duplicate and averaged for analyses. The lower limit of sensitivity of this cortisol assay is .007 ug/DL (Zimmerman & Schmeelk-Cone, 2003). Intra- and inter-assay coefficients of variability were below 8% and 7%, respectively. Saliva samples were assayed for DHEA in singlet and the test uses 50 μl of saliva and has a minimum detection limit of 10 pg/ml. Raw cortisol (ug/dl) and DHEA (pg/ml) concentrations assayed from sample 1 were used to calculate the cortisol/DHEA ratio. A natural log transformation was performed on the cortisol/DHEA ratio to account for positive skew.

Racial discrimination (RD).—The participants' perceived RD was measured using the 18-item Daily Life Experience scale (DLE; Harrell, 1997) at waves 5 and 6. The DLE assesses the frequency of racial microaggressions and overt discriminatory encounters experienced during the past 12 months on a 6-point Likert-type scale ranging from 0 (*never happened to me*) to 5 (*once a week or more*). Items were averaged, with higher scores reflecting higher levels of RD experience ($\alpha_{wave\ 5} = .96$; $\alpha_{wave\ 6} = .95$). Prior research has demonstrated the validity and reliability of the DLE among African American young adults (e.g., Lee et al., 2017). Sample items include "because of your race, how often were you observed or followed while in public places" or "because of your race, how often were you laughed at, made fun of, or taunted?"

Depressive Symptoms.—The participants' depressive symptoms were measured using 6 items from the Brief Symptom Inventory (BSI; Derogatis & Spencer, 1982). The BSI assesses the frequency of depressive symptoms (e.g., feeling lonely, feeling worthless) experienced during the past week. Response options ranged from 0 (*not at all*) to 4 (*extremely*). Items were averaged, with higher scores indicating higher levels of depressive symptoms ($\alpha = .85$).

Organized Religious Involvement (ORI).—ORI was measured using two items that assessed the frequency of service attendance and whether the participant was involved in other religious activities (e.g., choir; bible study). The frequency of service attendance was scaled from 0 (*never*) and 7 (*more than once a week*), and participation in other religious activities ranged from 0 to 3 activities. Standard scores for service attendance and other religious activities were calculated for each participant to account for differences in the Likert scales. Standard scores for service attendance and other religious activities was then averaged to approximate ORI.

Covariates.—Participants self-reported their sex at Wave 1 (i.e., 0 = male, 1 = female). The socioeconomic status (SES) of participants was estimated using their level of educational attainment (i.e., no high school degree, high school degree/GED, 1–4 years of

college) at wave 7. The participants also disclosed their age at wave 6. We also assessed the participant's alcohol and cigarette use in the past 30 days. Participant's responses ranged on an 8-point Likert type scale of 0 (*none*) to 7 (*40+ times*) for alcohol use and cigarette use, separately. Participants also self-reported various health behaviors on a Likert-type scale of 0 (*Almost Never*) to 4 (*Everyday*), including the frequency of exercise and consumption of fatty foods and fruits/vegetables in the past week. The participants' body-mass-index was calculated by using their self-reported weight and height at wave 6. Lifetime prevalence of chronic illnesses (i.e., asthma, chronic bronchitis/emphysema, diabetes, high blood pressure/ hypertension; coded as "yes" or "no" if ever diagnosed) was assessed at wave 6, and the number of doctor visits in the past year due to an illness. Lastly, interview time was included as a control variable to account for the diurnal decline in cortisol.

Analytic Approach

All study analyses were implemented in SPSS version 24 (SPSS, Inc., Chicago, IL, USA). Preliminary data analyses entailed descriptive statistics and inter-correlations between study variables. To assess depressive symptoms as a mediator in the association between RD and the cortisol/DHEA ratio (Aim 1), we used Hayes' (2013) PROCESS macro (i.e., model 4; see Hayes, 2013). The PROCESS macros use ordinary least squares estimation to compute model parameters for various path models (e.g., moderated-mediation). Bias-corrected bootstrapped confidence intervals (i.e., 5,000 bootstrapped samples) were used to estimate the indirect effect.

To assess the second aim of the study, ORI was included as a moderator to the mediation model in Aim 1. We implemented a moderated-mediation model using PROCESS macro (i.e., model 8; see Hayes, 2013). A mean-centered product between discrimination and ORI suggested a moderated-mediation effect. If the mean-centered product predicted depressive symptoms (i.e., indirect effect moderation), we probed the indirect effect of our original mediation model at the mean, and ±1 standard deviation at the mean, of the focal moderator. If the mean-centered product between RD and ORI predicted the cortisol/DHEA ratio (i.e., direct effect moderation), we probed the effect of RD and the cortisol/DHEA ratio at the mean, and ±1 standard deviation at the mean, of ORI. Non-significant mean-centered products were removed from the model, and a simpler moderated-mediation model was estimated (i.e., model 7; see Hayes, 2013). In all models, we controlled for sex, age, educational attainment, study onset time, tobacco use, alcohol use, exercise frequency, fruit consumption, fatty food consumption, chronic illness, doctor visits, BMI, perceived stress, and RD encounters from one year ago. The PROCESS macro requires complete data and implements listwise deletion to handle missing data (i.e., 21.3% ($n = 40$) had missing data). Lastly, we assessed if the cortisol/DHEA ratio mediated the association between RD and depressive symptoms to rule out an alternative mechanism linking RD, depressive symptoms, and the cortisol/DHEA ratio.

Results

Descriptive statistics and inter-correlations between the study variables can be found in Table 1. RD was associated with more depressive symptoms ($r = .20$), whereas ORI was

associated with fewer depressive symptoms ($r = -.15$). The cortisol/DHEA ratio was not correlated with RD or depressive symptoms.

Aim 1: Mediation Model from Racial discrimination to Cortisol/DHEA ratio

As shown in Table 2, an increase in RD was associated with more depressive symptoms ($b = .155$), while depressive symptoms predicted a lower cortisol/DHEA ratio ($b = -.114$). Furthermore, although the direct effect of RD on the cortisol/DHEA ratio was not significantly different from zero, RD had a significant indirect effect on the cortisol/DHEA ratio through depressive symptoms ($b = -.018$; 95% CI = $-.047, -.004$).

Aim 2: Moderated-Mediation Models with Organizational Religious Involvement

To assess whether ORI moderated the direct and indirect effect from RD to the cortisol/DHEA ratio, we estimated a moderated-mediation model (i.e., conditional process model; see Figure 1). ORI moderated the influence of RD on depressive symptoms ($b = -.094$), but not the direct effect of RD on the cortisol/DHEA ratio. As a result, we estimated an indirect effect moderation model without direct effect moderation (Table 3). In particular, RD had an indirect effect on the cortisol/DHEA ratio through depressive symptoms at low ($b = -.029$; 95% CI = $-.079, -.006$) and moderate levels of ORI ($b = -.019$; 95% CI = $-.051, -.005$), but not at a high level.

Sensitivity Analysis

We assessed whether cortisol/DHEA ratio mediated the association between RD and depressive symptoms. The indirect effect of RD on depressive symptom through the cortisol/DHEA ratio was not significant ($b = .018$; 95% CI = $-.002, .063$). Further, indirect and direct effect moderation were not observed in the moderated-mediation model (see Table 4).

Discussion

RD is a salient stressor that can increase susceptibility to poorer psychological well-being and physiological health among African Americans (see Lewis et al., 2015). Moreover, Chae and colleagues' (2011) socio-psychobiological model suggests that RD may indirectly exact physiologic tolls by generating greater psychological risk (e.g., depressive symptoms; Chae et al., 2011). Our findings are consistent with this model, suggesting that depressive symptoms mediate the association between RD and the imbalance of the cortisol/DHEA ratio. Specifically, in line with prior research, we found that RD was associated with higher levels of depressive symptoms (e.g., Hurd et al., 2014), and that symptoms of depression were associated with lower cortisol/DHEA ratios (e.g., Gill et al., 2008). Importantly, a direct effect of RD on the ratio of cortisol/DHEA was not observed. This may suggest that the mental health consequences of RD may precede the imbalance of stress reactive hormones. Alternatively, RD encounters may shape acute cortisol reactivity, but not regulatory (i.e., basal) levels of cortisol as assessed in our study (Korous, Causadias, & Casper, 2017). Together, our findings indicate that RD may invoke dysregulations in the neuroendocrine system by deteriorating mental health.

It is important to mention that our results are inconsistent with some other work that has documented greater cortisol and lower DHEA concentrations (i.e., a higher cortisol/DHEA ratio) among individuals with major depressive disorder and elevated depressive symptoms (see Sollberger & Ehlert, 2016). Two separate lines of research can inform our understanding of why depressive symptoms, in some cases, predict a lower cortisol and higher DHEA (i.e., a lower cortisol/DHEA ratio). First, scholars have found that depressive *symptoms* are associated with a blunted cortisol response to a stressor (Burke, Fernald, Gertler, & Adler, 2005), as well as a dampened cortisol awakening response and diurnal rhythm (Doane et al., 2013; Stetler & Miller, 2005). Second, researchers have found higher DHEA concentrations among *patients* diagnosed with major depressive disorder (Assies et al., 2004), as well as healthy individuals reporting higher levels of depressive symptoms (Morrison, Ten Have, Freeman, Sammel, & Grisso, 2001). Together, these findings may begin to explain why depressive symptoms, in some cases, may reduce cortisol levels, while elevating DHEA.

The persistence of mixed results between symptoms of psychopathology and the cortisol/ DHEA ratio hints that an imbalance between stress-reactive hormones, regardless of its direction, reflects higher allostatic load (Shirtcliff, Peres, Dismukes, Lee, & Phan, 2014). While some researchers have reported positive associations between psychopathology and cortisol/DHEA ratio (e.g., Markopoulou et al., 2009; Young et al., 2002), others have documented a negative association (e.g., Gill et al., 2008; Shirotsuki et al., 2009; Yehuda, 2006). Furthermore, low cortisol/DHEA ratios have been associated with poorer health outcomes such as pro-inflammatory immune response (Gill et al., 2008). Our study, to this end, is one of the first to suggest that, at the very least, the psychological consequences of RD may contribute to the imbalance of cortisol and DHEA concentrations.

With regard to ORI, we found that ORI buffered the indirect effect of RD on the cortisol/ DHEA ratio. Specifically, ORI mitigated the influence of RD on depressive symptoms, which, in turn, offset the indirect influence of RD on the cortisol/DHEA ratio. Furthermore, we did not find evidence of a direct effect between RD and the cortisol/DHEA ratio across different levels of ORI. Our results, when considered in tandem, suggest that ORI counteracts the influence of RD on biological stress regulation by offsetting the psychological consequences of RD. Various mechanisms may underlie the protective influence of ORI in the context of RD. Religious social support (Hope et al., 2016), religious teachings that emphasize liberation and hope (Mattis et al., 2003), and civic engagement opportunities (Hope & Spencer, 2017) may be important mechanisms by which ORI can buffer the psychological consequences of RD. In light of our findings, future investigations should examine the psychologically salutary assets and resources afforded by ORI to ameliorate the physiologically deleterious effects of RD.

Although our study advances our understanding of how RD, depressive symptoms, and ORI work in concert in relation to biological stress response, several limitations suggest a cautious interpretation of our results. First, while we were able to detect direct and indirect effects in our sample, it is possible that our standard errors are biased due to the limited sample size (MacKinnon et al., 2007). Thus, it is important to validate our findings in a larger sample of African Americans. Second, due to documented instabilities in level of

cortisol across days (e.g., Doane et al., 2015), our one day assessment of cortisol may not generalize to the participant's global experience. Accordingly, psychological pathways from RD to the cortisol/DHEA ratio after taking into account day-to-day variability in cortisol level should be examined. Third, it is possible that our limited measure of ORI broadly captures religiosity domains, which, in turn, would upwardly bias the protective effects of ORI. It is, therefore, important to measure religious involvement using a multidimensional measure to more accurately assess the unique contribution of ORI on psychophysiological outcomes. Fourth, religious affiliation was not assessed in the original study and it is possible that our assessment of ORI as a protective factor may not generalize to a non-Christian sample. Given the documented differences in ORI across religious groups (Pearce & Denton, 2011), it is important to investigate how ORI promotes resilience within various religious groups. A final potential limitation concerns the measurement of RD as a unidimensional construct. Our measure was limited to the frequency of racial hassles. Future studies can advance our understanding of RD and stress reactive hormones by examining other dimensions of RD such as institutional forms (e.g., racial profiling by law enforcement, residential segregation; Williams & Mohammed, 2013).

Notwithstanding these limitations, our results contribute to the growing body of evidence that indicates the socio-psychobiological pathways by which RD "gets under the skin" (Chae et al., 2011). By identifying depressive symptoms as a mediator of the association between RD and the imbalance of cortisol and DHEA, our findings suggest that that the psychological and physiological consequences of RD may work together to accelerate the deterioration of health. In particular, an individual's psychological response to RD may generate susceptibility to greater physiological risk and poorer health. Our findings also illustrate that the mental health benefits of ORI may ultimately benefit the physical health of African Americans in the context of racism-related stress. Our study represents a preliminary step towards determining how ORI can shield African Americans from the adverse physiological effects of RD. Future investigations should mechanistically examine how ORI and different types of religious involvement offer physiological protection to African Americans contending with RD. Mechanisms such as gratitude (Krause, 2006), forgiveness (Krause & Ellison, 2003), and religious coping (Pargament, 1997) are plausible pathways by which ORI might offer protection to African Americans within the context of RD. By testing these mechanisms, interventionists, researchers, and clinicians will be able to identify approaches to address racism-related stress within faith-based communities.

Acknowledgments

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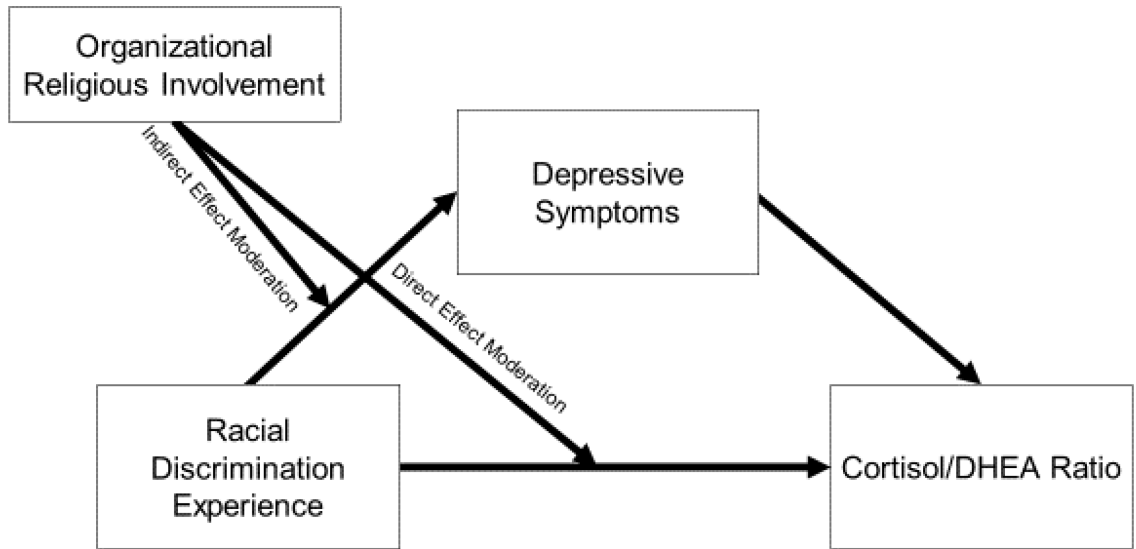
Figure 1.
Conceptual model bridging racial discrimination to the cortisol/DHEA ratio, with organizational religious involvement as protective factor

Table 1

Descriptive Statistics and Intercorrelations Between Study Variables

	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	% or M(SD)
1. Sex	--																			52.7% Male
2. Age	-.14 T	--																		20.98 (0.67)
3. Educational Attainment	.23 **	-.33 **	--																	4.78 (2.10)
4. Racial Discrimination (Wave 5)	-.07	-.01	-.04	--																0.85 (0.81)
4. Racial Discrimination (Wave 6)	-.15 *	.03	-.08	.40 **	--															0.90 (0.91)
5. Depressive Symptoms	.16 **	-.06	-.05	.18 *	.31 **	--														0.68 (0.69)
6. Alcohol Use	-.14 T	-.07	.01	.19 *	.20 **	.15 *	--													4.78 (3.78)
7. Cigarette Use	-.16 T	.05	-.25 **	.04	.02	-.08	.40 **	--												5.80 (2.82)
8. Exercise	-.19 **	-.01	-.05	-.07	-.04	.04	.07		--											2.55 (1.27)
9. Fruit Consumption	.00	-.04	.08	-.05	-.16 *	-.14 T	-.12		.21 **	--										2.70 (1.20)
10. Fat Food Consumption	-.02	-.02	-.04	.11	.03	.15 *	.05	.08	-.08		--									3.84 (1.15)
11. BMI	.19 *	-.02	.04	-.02	-.06	-.08	-.18 *	-.04	.18 *	-.07		--								26.59 (6.97)
12. Perceived Stress	.04	.06	-.16 *	.18 *	.56 *	.14 T	.16 *	.02	-.10	.06	-.04		--							2.43 (0.59)
13. Doctor Visits	.12	.08	-.02	.08	.07	-.08	-.05	.19 *	.17 *	.06	.02	.03	--							1.76 (2.02)
14. Chronic Illness	-.01	-.02	.08	-.02	.09	.03	-.03	.17 *	.15 *	-.03	.16 *	.03	.15 **	--						0.27 (0.54)
15. Organizational Religious Inv.	.03	.06	.13 T	-.01	-.14 *	-.31 **	-.22 **	.14 T	.29 **	-.01	.13 T	.11	.09	.04	--					.09 (0.92)
16. Saliva Collection Start Time	-.04	.05	-.01	.06	-.15 *	.01	.06	-.11	-.24 **	.06	-.10	-.07	-.06	.02	-.04	--				1:54PM (2.97 hrs)
17. Cortisol Concentration (ug/dl)	-.16 *	.01	.05	.02	.03	.00	.13 T	-.06	.06	-.07	.01	.16 *	-.13 T	-.09	.04	-.22 **	--			0.27 (0.23)
18. DHEA Concentration (pg/ml)	-.32 **	.06	-.01	.11	.02	.05	.14 *	-.01	.04	.06	-.04	.06	-.06	.04	-.04	.01	.41 **	--		127.75 (107.67)
19. Log(Cortisol/DHEA ratio)	.22 **	-.08	.03	.00	-.02	-.04	-.03	.05	.01	-.07	-.07	.07	.02	-.05	.09	-.16 *	.25 **	-.57 **	--	-0.74 (0.29)

Note.

T
p < .10.

*
p < .05.

**
p < .01.

BMI = Body Mass Index.

Table 2

Direct and Indirect Effects from Racial Discrimination to the Cortisol/DHEA Ratio

Outcome: Depressive Symptoms	b	s.e.	p	Biased Corrected 95% C.I.
Racial Discrimination (Wave 6)	.155	.061	.013	.034, .276
Racial Discrimination (Wave 5)	−.001	.065	.923	−.131, .128
Sex	.268	.106	.013	.058, .478
Age	−.059	.077	.443	−.211, .093
Educational Attainment	.014	.026	.577	−.036, .065
Alcohol Use	.026	.015	.083	−.003, .054
Cigarette Use	−.015	.019	.441	−.054, .024
Exercise	−.019	.041	.629	−.099, .060
Fruit Consumption	−.077	.045	.085	−.165, .011
Fatty Food Consumption	−.011	.044	.805	−.097, .075
BMI	−.006	.008	.431	−.022, .009
Perceived Stress	.515	.089	< .001	.339, .692
Doctor Visits	.022	.025	.374	−.027, .072
Chronic Illness	.126	.091	.167	−.053, .304
Organizational RI	−.061	.059	.299	−.177, .055
Saliva Collection Start Time	−.669	.43	.130	−1.537, .199
Constant	.812	1.731	.639	−2.617, 4.241

Outcome: Cortisol/DHEA Ratio	b	s.e.	p	95% C.I.
Depressive Symptoms	−.114	.049	.022	−.211, −.017
Racial Discrimination (Wave 6)	−.028	.033	.405	−.094, .038
Racial Discrimination (Wave 5)	.010	.035	.776	−.059, .078
Sex	.176	.058	.004	.062, .291
Age	−.046	.041	.260	−.127, .035
Educational Attainment	−.002	.014	.892	−.029, .025
Alcohol Use	.005	.008	.571	−.011, .020
Cigarette Use	.003	.010	.747	−.017, .024
Exercise	.021	.022	.341	−.022, .063

Outcome: Depressive Symptoms	b	s.e.	p	Biased Corrected 95% C.I.
Fruit Consumption	−.010	.024	.667	−.058, .037
Fatty Food Consumption	−.020	.023	.389	−.066, .026
BMI	−.006	.004	.169	−.014, .003
Perceived Stress	.134	.054	.014	.028, .240
Doctor Visits	.015	.013	.255	−.011, .042
Chronic Illness	−.019	.048	.698	−.114, .077
Saliva Collection Start Time	−.416	.235	.079	−.880, .049
Organizational RI	.022	.031	.474	−.039, .084
Constant	.134	.919	.885	−1.686, 1.953
Indirect Effect	−.018	.009	--	−.047, −.003

Note. BMI = Body Mass Index. C.I. = Confidence Interval.

Table 3

Organizational Religious Involvement as a Protective Factor (Indirect Effect Moderation)

Depressive Symptoms (Mediator)	b	s.e.	p	Bias Corrected 95% C.I.
Racial Discrimination (Wave 6)	.172	.061	.005	.052, .292
Racial Discrimination (Wave 5)	−.013	.064	.846	−.139, .114
Sex	.273	.105	.011	.065, .481
Age	−.046	.076	.543	−.196, .104
Educational Attainment	.022	.026	.401	−.029, .072
Alcohol Use	.021	.014	.157	−.008, .049
Cigarette Use	−.013	.019	.504	−.051, .025
Exercise	−.018	.039	.652	−.097, .061
Fruit Consumption	−.084	.044	.058	−.171, .003
Fatty Food Consumption	−.011	.043	.799	−.095, .074
BMI	−.007	.008	.373	−.022, .008
Perceived Stress	.519	.087	< .001	.345, .692
Doctor Visits	.022	.025	.374	−.027, .071
Chronic Illness	.102	.089	.252	−.074, .279
Organizational Religious Involvement	−.021	.021	.320	−.062, .020
Organizational Religious Involvement X Racial Discrimination	−.048	.020	.018	−.087, −.009
Saliva Collection Start Time	−.676	.430	.119	−1.528, .177
Constant	.698	1.718	.685	−2.704, 4.101

Cortisol/DHEA Ratio (Outcome)	b	s.e.	p	Bias Corrected 95% C.I.
Depressive Symptoms	−.117	.049	.018	−.214, −.021
Racial Discrimination (Wave 6)	−.026	.033	.435	−.092, .040
Racial Discrimination (Wave 5)	.010	.034	.766	−.058, .079
Sex	.173	.058	.003	.059, .287
Age	−.043	.040	.293	−.123, .037
Educational Attainment	−.004	.013	.974	−.027, .026
Alcohol Use	.003	.008	.672	−.012, .018
Cigarette Use	.003	.010	.807	−.018, .023

Depressive Symptoms (Mediator)	b	s.e.	p	Bias Corrected 95% C.I.
Exercise	.023	.021	.275	−.019, .065
Fruit Consumption	−.007	.024	.746	−.054, .039
Fatty Food Consumption	−.018	.023	.443	−.063, .028
BMI	−.006	.004	.192	−.014, .003
Perceived Stress	.135	.053	.013	.029, .241
Doctor Visits	.016	.013	.242	−.011, .042
Chronic Illness	−.022	.048	.650	−.117, .073
Saliva Collection Start Time	−.422	.234	.074	−.886, .041
Constant	.017	.911	.985	−1.787, 1.822
Indirect Effect: −1 SD ORI	−.035	.018	--	−.070, −.003
Indirect Effect: Mean of ORI	−.020	.011	--	−.042, −.001
Indirect Effect: +1 SD ORI	−.006	.008	--	−.022, .013

Note. ORI = Organizaitonal Religious Involvement. BMI = Body Mass Index.

Table 4

Sensitivity Analysis

Indirect Effect	b	s.e.	Bias Corrected 95% CI
Racial Discrimination → Cortisol/DHEA → Depressive Symptoms	.006	.011	−.013, .031
Conditional Indirect Effects			
−1 SD ORI	.019	.023	−.013, .080
Mean of ORI	.008	.012	−.011, .039
+1 SD ORI	−.003	.013	−.035, .017

Note. C.I. = Confidence Interval.