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Social context matters: Ethnicity, discrimination and stress reactivity

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ABSTRACT

Exposure to chronic discrimination is associated with increased morbidity and mortality. The study of biobehavioral pathways linking discrimination with health outcomes has mostly focused on the cardiovascular system, with fewer studies addressing the hypothalamus-pituitary-adrenal (HPA) axis. In this study we tested associations between Latino ethnicity, experiences of discrimination, and cortisol responses to an acute laboratory stressor. One hundred fifty eight individuals (92 female, 66 male) between the ages of 18 and 29 years participated in the study. Salivary cortisol was measured once before and eight times after administration of a laboratory stressor (the Trier Social Stress Test). Past experiences of discrimination were measured with the Experiences of Discrimination Scale. Findings from conditional process modeling suggest that Latino ethnicity predicted a) heightened cortisol reactivity and b) more pronounced cortisol recovery through discrimination experiences (mediator), and that this effect was further moderated by sex with a significant indirect effect only among males. The direct path from Latino ethnicity to cortisol reactivity or cortisol recovery was, however, not significant. In sum, findings suggest that Latino ethnicity and discrimination interact to predict cortisol dysregulation, which implies that an appropriate model for understanding minority health discrepancies must incorporate interactive processes and cannot simply rely on the effects of ethnicity or discrimination alone.

1. Social context matters: ethnicity, discrimination and stress reactivity

Discrimination is the unequal treatment of an individual or a group of individuals based on real or perceived differences, and it frequently occurs based on race, age, sex, sexual orientation, gender identity, nationality, religion or disability. Exposure to chronic discrimination has been associated with increased mortality and a wide range of negative physical and mental health outcomes (Paradies et al., 2015; Williams and Mohammed, 2009). Moreover, there is evidence that the stress resulting from discrimination may contribute to health disparities in ethnic minority populations that cannot be accounted for by sociodemographic variables alone (Williams, 1999; Williams and Collins, 1995).

In terms of biobehavioral pathways linking stress from discrimination with disease, the majority of studies have focused on discrimination-related cardiovascular dysregulations and cardiovascular health outcomes (Pascoe and Smart Richman, 2009). The relative lack of research on the role of the hypothalamus-pituitary-adrenal (HPA) system in this context is not just a theoretical omission. In their review of biobehavioral pathways linking discrimination-related stress with disease, Berger and Sarnyai (2015) proposed that stress-related cardiovascular dysregulations primarily result in adverse physical health outcomes, whereas stress-related dysregulations in the HPA system, via their structural and functional changes in brain structures relevant for stress regulation, also contribute to negative mental health outcomes. In fact, many decades of research support the idea that chronic dysregulations in HPA axis function are associated with physical and mental stress-related disease (Chrousos and Gold, 1992), making hormones of the HPA axis a highly relevant research target in the present context.

Briefly, when the HPA axis is activated, the hypothalamus releases corticotrophin-releasing hormone, which stimulates the pituitary gland to release adrenocorticotropic hormone, which then triggers cortisol release from the adrenal cortex; cortisol can bind to receptors on the pituitary, hypothalamus and higher order brain structures, regulating its own activity by a negative feedback system (Tsigos and Chrousos, 2002). The HPA axis is a highly dynamic system characterized by strong diurnal hormonal fluctuations and by its reactivity to acute environmental events, including food intake, exercise and, with particular relevance to the present study, acute stress. It has been argued that individual differences in stress reactivity are reflective of individuals' vulnerability to stress and may explain some of the variability in the link between stress and disease (Boyce et al., 1995; Cohen and Manuck,

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1995; Lovallo and Gerin, 2003).

The literature on discrimination and HPA axis responses to acute stressors, which we review in more detail elsewhere (Busse et al., 2017), is small and for the most part reports on changes in cortisol, the end-product of the HPA axis. Studies consistently point toward altered cortisol reactivity with discrimination experiences, but the direction of the effect has varied. One major observation in our review paper (Busse et al., 2017) was that studies inducing experiences of discrimination in the laboratory, typically by using stressors that include elements of discrimination, find more pronounced cortisol responses with discrimination (Hehman and Bugental, 2015; Townsend et al., 2014; Townsend et al., 2011). Conversely, studies comparing cortisol responses between individuals with and without a prior history of discrimination find more blunted responses with discrimination (Hatzenbuehler and McLaughlin, 2014; Jamieson et al., 2013; Richman and Jonassaint, 2008). Similarly, a more recent study showed flattened cortisol responses with internalized racism, a construct related to discrimination, but no association between cortisol reactivity and interpersonal racism was found (Berger et al., 2017).

A second observation concerned possible interactive effects. Some studies are suggestive of interactions between race and discrimination in predicting changes in cortisol reactivity, such that a link between cortisol reactivity and discrimination is found in one ethnicity but not in another (Huynh et al., 2016; Tse et al., 2012; Zeiders et al., 2014). Other studies have shown that discrimination can be associated with altered cortisol reactivity, independent of race (e.g., Skinner et al., 2011) and, conversely, that racial differences in HPA axis activity cannot be solely explained by discrimination experiences (e.g., Martin et al., 2012). At this time, the exact nature of these interactive processes remains poorly understood.

In the present study, we set out to investigate whether living in a social context that is characterized by a high degree of stigma is associated with more frequent experiences of discrimination and, in turn, with HPA axis dysregulation. We studied Latinos, a U.S. group whose members are frequently targets of discrimination. Latinos commonly report being discriminated against by their peers, their teachers, and others based on language skills, immigration status, socioeconomic status (SES) and skin color (Edwards and Romero, 2008). We hypothesized first, that being of Latino ethnicity and scoring high on experiences of discrimination would be independently associated with blunted cortisol reactivity; and second, that the association between Latino ethnicity and blunted cortisol reactivity would be mediated by experiences of discrimination.

2. Method

2.1. Participants

The complete study sample consisted of 158 individuals. Fifteen individuals were excluded, four because they reported being half Latino and half non-Latino, and 11 because of missing or insufficient cortisol data. The remaining 143 participants (80 female, 63 male) were between 18 and 29 years old (M = 20.49, SD = 2.08). Of these participants 57.3% reported being Latino, and 42.7% reported being non-Latino (White: 15.4%, Asian: 14.7%, Other: 12.6%). Participants came from various educational backgrounds (college degree, mothers: 25.2%, fathers: 35.7%; middle school or less, mothers: 24.5%; fathers: 21.0%). Mean years of education was 12.24 (SD = 5.38) for mothers and 13.21 for fathers (SD = 5.42). In terms of SES, most participants described their families as lower middle class (43.4%; skilled trade, steady employment), followed by upper middle class (22.4%; professionals, high earned income), upper working class (18.2%; skilled workers, steady employment), and lower working class (16.1%; unskilled workers, employed off-and-on). None of the participants identified their families as upper class (e.g., do not have to work for a living, inherited wealth).

Recruitment occurred among students at the University of California, Irvine and surrounding community colleges. Individuals were excluded from participation if they used medications known to affect cortisol, reported major medical conditions, speech or math phobia, alcohol and drug use, and tobacco use exceeding five cigarettes per day. Of note, none of the participants reported smoking regularly over the past six months.

2.2. Overall procedure

All procedures were approved by the Institutional Review Board of the University of California, Irvine, and all participants provided written informed consent before study procedures commenced. Study days were scheduled to begin at 2pm to control for the significant circadian variation in cortisol secretion.

After a 15-min rest period, a first saliva sample (–2 min) was collected, and participants were escorted to an adjacent room to complete the laboratory stressor (Trier Social Stress Test, TSST; Kirschbaum et al., 1993). The TSST consists of a 5-min mock job interview and a 5-min mental arithmetic task in front of two neutral, non-supportive expert evaluators of diverse ethnicities (e.g., Latino, European, East Asian, mixed background) and both sexes. Including the instruction and preparation period, the TSST lasted 15 min. Upon completion of the TSST, a second saliva sample was collected (+1 min). Participants then returned to the waiting room where additional saliva samples were collected at +10, 20, 30, 45, 60 and 90 min. During this time, participants also completed questionnaires. At the end of the session, participants were thanked, carefully debriefed and awarded their choice of course extra credit or $50.

2.3. Measures

2.3.1. Saliva sampling and cortisol assay

Saliva samples were collected with the Salivette sampling device (Sarstedt, Nümbrecht, Germany), stored at room temperature until completion of the session and then kept at −70 °C until assayed. After thawing, samples were centrifuged for 10 min at 2000 g and 4 °C. Free cortisol was determined in duplicate by an enzyme immunoassay (IBL-America, Minneapolis, Minnesota). The sensitivity of the assay is 0.033 nmol/L, and the dynamic range is 0–82.77 nmol/L. Inter-and intra-assay coefficients of variance are reported at 4.9% and 4.1%, respectively.

2.3.2. Discrimination

Discrimination experiences were assessed with the Experiences of Discrimination Scale (EOD, Krieger et al., 2005). The EOD contains 9 items about past incidences of discrimination in specific settings, including finding housing, a job, and medical care. Each item is rated based on the frequency of these occurrences over the lifetime (0 = never, 1 = once, 2.5 = two or three times, 5 = four or more times). Individuals are also asked to indicate what category (e.g., race, sex) they consider to be the major reason for the experience of discrimination. The EOD has shown acceptable internal consistency (α = 0.74).

2.3.3. Statistical methods

Summary scores were computed for the EOD, such that higher scores indicated more frequent discrimination experiences. To capture relevant aspects of the salivary cortisol response to the TSST, five summary measures were computed: Cortisol secreted throughout the entire assessment period (pre to +90 min samples) was computed using simple area under the curve computations using zero as a reference line (termed, *cortisol AUC*). To capture cortisol reactivity, we computed a) the *maximum cortisol increase* by a participant in response to the TSST by subtracting the pre-TSST cortisol value from the maximum cortisol value obtained at any time after the TSST (+1 min to +90 min samples), and b) the *mean cortisol increase* by subtracting the pre-TSST