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Dispositional depression and hostility are associated with inflammatory markers of cardiovascular disease in African Americans

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ABSTRACT

Prior research has demonstrated that state depressive symptoms and hostility can modulate inflammatory immune responses and directly contribute to cardiovascular disease (CVD) onset and development. Previous studies have not considered the contribution of dispositional depressive symptoms to the inflammatory process. They have also largely excluded African Americans, despite their disproportionate risk for CVD. The first aim of the study was to examine the impact of state and dispositional depression and hostility on CVD-associated inflammatory biomarkers interleukin-6 (IL-6) and C-reactive protein (CRP) in an African American sample. The second aim was to examine synergistic influences of hostility and state and dispositional depression on IL-6 and CRP. The final aim was to examine whether the relations between state and dispositional depression, hostility, IL-6, and CRP varied as a function of gender and education. Anthropometric measures, blood serum samples, and psychosocial data were collected from 198 African Americans from the Washington, DC metropolitan area. Hierarchical and stepwise regression analyses indicated that (1) increased levels of hostility were associated with increased levels of CRP; (2) hostility and IL-6 were more strongly associated among participants with lower educational attainment; and (3) dispositional depression and CRP were more strongly associated among participants with greater hostility and lower educational attainment. Findings suggest that enduring personality dispositions, such as dispositional depression and hostility, are critical to a thorough assessment of cardiovascular profiles in African Americans. Future studies should investigate causal pathways that link depressive and hostile personality styles to inflammatory activity for African American men and women.

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1. Introduction

Cardiovascular disease (CVD) is the number one cause of morbidity and mortality in the United States and disproportionately burdens African Americans (Yancy, 2005; Nabel, 2003). In comparison to Caucasians, African Americans have higher rates of diabetes mellitus (DM), obesity, and hypertension, which are associated with increased susceptibility to atherosclerosis, myocardial infarction (MI), coronary heart disease (CHD), and stroke (Berenson et al., 1998; Yusuf et al., 2004; Blankstein et al., 2011; Sacco et al., 2001; Carter et al., 1996; Flegal et al., 2010; Borrell, 2009). Although biological risk factors contribute to disparities between African Americans and other ethnic groups, psychological risk factors, such as depression and hostility, also contribute to the pathogenesis and expression of CVD and poorer cardiovascular health (Ariyo et al., 2000; Musselman et al., 1998; Kop, 1999).

The association between depressive symptoms, hostility, and CVD risk is greater among African Americans, when compared to Caucasians (Lewis et al., 2009; Jonas and Lando, 2000; Jonas and Mossolino, 2000; Davidson et al., 2000). Depressive symptoms have been associated with CVD risk factors, such as hypertension, obesity, DM, and stroke (Wuslin & Singal, 2003; Davidson et al., 2000; Dixon et al., 2003; Palinkas et al., 2004; Everson et al., 1998). Hostility has also been recognized as an independent predictor of CVD, CHD, low-density lipoproteins (LDL), cardiac death, MI, and hypertension (Graham et al., 2006; Miller et al., 1996; Brindley et al., 1993; Shekelle et al., 1983; Yan et al., 2003).

Psychological stressors can activate the hypothalamic-pituitary-adrenocortical (HPA) axis and the sympathetic nervous system to influence the onset and development of CVD. HPA activity can increase cortisol, which is implicated in the development of atherosclerosis and hypertension (Ghiadoni et al., 2000; Hajat

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et al., 2010; Whitworth et al., 2005). Moreover, the HPA axis can bidirectionally regulate the proinflammatory cytokine interleukin-6 (IL-6) and the hepatocyte produced C-reactive protein (CRP). Chronic IL-6 production can lead to the destruction of the cardiovasculature (Hou et al., 2008; Lagathu et al., 2003; Memoli et al., 2007; Singh et al., 2005; Fredj et al., 2005). Interleukin-6 is involved in the initiation and progression of atherosclerotic cardiovascular disease and can precede the onset of DM (Andersen and Pedersen, 2008; Memoli et al., 2007). Furthermore, CRP is a hallmark of acute and chronic inflammation. High levels of CRP are associated with risk of stroke, MI, and poor cardiovascular events, even among healthy individuals (Ridker et al., 1997). Both IL-6 and CRP are associated with sub-clinical and clinical CVD (Cesari et al., 2003; Ridker, 2007; Willerson and Ridker, 2004; Ridker et al., 2000; Ridker et al., 2002). Thus, examination of these markers concurrently may be a better predictor of CVD risk than when examined independently.

Studies show that greater depressive symptomatology and hostility are associated with increased concentrations of IL-6 and CRP in the blood (Suarez, 2004; Suarez, 2003a,b). A meta-analysis of studies conducted between 1967 and 2008 demonstrated a consistent association between depression, IL-6 and CRP, in both clinical and population-based studies, and suggested that depressive symptoms increased CVD risk even among non-patient samples (Howren et al., 2009). Of note, these studies have primarily examined the relation between acute depressive symptoms and inflammation to explain CVD risk. There is recent evidence to suggest that sustained long-term personality dispositions promote a more detrimental cardiovascular profile, particularly in African–American men (Sims et al., 2010).

Previous research has examined the joint effects of hostility and severity of depressive symptoms on inflammation (Suarez, 2003a; Miller et al., 2003; Stewart et al., 2008; Brummett et al., 2010). Consistently across these studies, an interactive relationship between depressive symptoms, hostility, and inflammatory marker levels was found. Suarez (2003a) categorized a sample of 90 relatively healthy men into groups based on depressive symptom and hostility scores. Results indicated that the group with higher depressive symptoms and higher hostility had greater levels of IL-6 compared to the remaining groups. Similarly, depressive symptoms moderated the relationship between hostility and IL-6 and CRP in a sample of 316 older individuals aged 50–70 years (Stewart et al., 2008). In these studies, positive associations between hostility and inflammatory markers were observed in participants that endorsed higher depressive symptoms, but not lower. However, Miller et al. (2003) found, in a sample of 100 ethnically diverse individuals, higher hostility was associated with greater IL-6 among participants with lower depressive symptoms scores. Sample differences among the studies may account for discrepant findings, although this is unclear. For example, gender composition may impact findings. Studies examining the moderating effects of gender on relations among depressive symptoms, hostility and inflammatory markers have yielded mixed findings. Brummett et al. (2010) found depressive symptoms were related to inflammatory markers more strongly in hostile women, while other studies did not find significant interactions between depressive symptoms, hostility, gender, and inflammatory markers (Stewart et al., 2008; Miller et al., 2003). Further, few studies have examined the combined effects of more longstanding personality styles. In one study, Boyle and colleagues (2007) found that a composite of depressive, angry and hostile personality styles predicted 10-year increases in complement component 3, an important marker of inflammation that is activated by CRP.

To our knowledge, there are no studies that have explored the temporal distinction in depression and further examined whether they work in tandem with hostility to influence inflammatory marker levels in African Americans. The current study investigated the independent and synergistic influences of state depression, dispositional depression and hostility on IL-6 and CRP. We posited that each of these psychological constructs would be independently associated with higher levels of inflammatory markers IL-6 and CRP. We further hypothesized that state and dispositional depression would be more strongly associated with markers of inflammation in the presence of higher levels of hostility, suggesting that greater cardiovascular risk is associated with the interaction of both negative affective and dispositional attributes. Moreover, we aimed to examine within-group differences to determine whether gender and socio-economic status (SES) influence CVD risk, as found in prior research (Lewis et al., 2011; Winston et al., 2009; Winkleby et al., 1998).

2. Methods

The current study was conducted as part of the National Minority Organ Tissue Transplant Education Program (MOTTEP) at Howard University entitled *Psychoneuroimmunological Risk Factors in Renal Health and Disease* and was approved by the Howard University (HU) Institutional Review Board. The study examined stress and psychoneuroimmunological factors in renal health and disease in African Americans. It was conducted from 2004 to 2007 at the General Clinical Research Center (GCRC) at HU Hospital. Participants were recruited through flyers posted at HU Hospital and advertisement at local health fairs. Participants were screened by phone and eligibility was based on self-report. Participants provided informed consent prior to participation. Data collection took an average of four hours and each participant received monetary compensation.

2.1. Participants

The participants were a community-based sample of 198 African Americans (48% male), who resided in the Washington, DC metropolitan area. Individuals with current physical, emotional, or drug abuse, as well as a previous diagnosis of a psychiatric illness, were excluded. Demographic information and a full health history were collected. Sixteen participants were omitted from the total sample of 214 participants due to missing data. Demographic information is provided in Table 1.

2.2. Biological measures

Participants underwent a medical examination conducted by nursing staff at the GCRC. Weight and height measurements were used to calculate body mass index (BMI). A seated blood pressure measurement was taken via a sphygmomanometer on a single occasion. A second and third blood pressure reading were not added until the 93rd participant; therefore, only the first blood pressure reading was included in the analyses. Blood serum assays were collected using a venipuncture procedure to determine CRP and IL-6 levels. Non-fasting blood samples were drawn into four cryovials and frozen at −70 Celsius. After clotting, blood samples were centrifuged and aliquoted into six vials to be stored at the GCRC until collected by Quest Laboratories. Blood samples were detected for levels of IL-6 and CRP by enzyme-linked immunosorbent assays (ELISA) and quantified by a microplate spectrophotometer. Self-reported diabetes and self-reported hypertension were assessed with a health history questionnaire.

2.3. Psychological measures

Depressive symptoms were assessed using the Beck Depression Inventory-II (BDI-II) (Beck et al., 1996). The BDI-II is a well-vali-

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