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ABSTRACT

Title of Document: Interactive Relations of Racial Discrimination and Poverty Status and Sex with Blood Pressure among African Americans: HANDLS

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Racial discrimination has been linked to cardiovascular disease (CVD), but little is known about the pathways of this association due to limitations in previous literature. Specifically, more research is needed on the combined, interactive, and moderating influence of social statuses including socioeconomic status (SES) and sex on this association. The current study examined whether the association between racial discrimination and BP was moderated by poverty status and sex in African Americans. The sample (*N*=1,408) was drawn from the Healthy Aging in Neighborhoods of Diversity across the Life span (HANDLS) study (30-64 years; 43.8% male; 46% below the 125% poverty line). Multivariable and logistic regression analyses were used to examine interactive associations of racial discrimination, poverty status, and sex to resting blood pressure and hypertension status. The hypothesized interactions were not significant, and significant findings were found in the direction opposite of what was expected. The current study suggests that interactive relations of racial discrimination, poverty status, and sex are not consistently related to resting blood pressure and hypertension status in the present sample. This may be due to a true absence of association, or limitations related to the selected measures. Future research is suggested to account for these possibilities.

Interactive Relations of Racial Discrimination and Poverty Status and Sex with Blood Pressure among African Americans: HANDLS

By: Taylor M. Darden

Thesis submitted to the faculty of the Graduate School of the University of Maryland, Baltimore County in fulfillment of the requirements for the degree of Master of Arts, May 2019

Advisory Committee: Dr. Danielle L. Beatty Moody, Dr. Kenneth Maton, Dr. Shari Waldstein, Dr. Alan Zonderman

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Introduction

In the United States (U.S.), discrimination at the interpersonal level, defined as "behavioral manifestations of a negative attitude, judgment, or unfair treatment toward members of a group," is a well-documented and significant social determinant of health (Pascoe & Smart Richman, 2009; Williams, Neighbors, & Jackson, 2003). Racial and ethnic minorities who repeatedly experience discrimination, regardless of the type (e.g., racial) or level (e.g., interpersonal), can experience a wide array of adverse mental and physical health outcomes (Brondolo, ver Halen, Libby, & Pencille, 2011b; Paradies et al., 2015; Pascoe & Smart Richman, 2009; Williams, Yu, Jackson, & Anderson, 1997). The theoretical framing underscoring the majority of this research is the stress framework (Clark, Anderson, Clark, & Williams, 1999; Lazarus & Folkman, 1984). Within this framework, discrimination is conceptualized as a chronic stressor (Clark et al., 1999; Harrell, 2000), similar to various other ongoing and taxing events in day-to-day life. For instance, chronic stress arising from caregiver strain has been linked to a host of adverse outcomes such as decreased quality of life, increased mortality rates, and overall greater risk for physical health problems, including cardiovascular disease (CVD; Ames, Jones, Howe, & Brantley, 2001; Ohlin, Nilsson, Nilsson, & Berglund, 2004; Schulz & Beach, 1999; Vitaliano, Scanlan, Zhang, Savage, & Hirsch, 2002). Similarly, a growing literature demonstrates that discrimination, in its role as a chronic stressor, is associated with similar poor health endpoints (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011a; Brondolo, Rieppi, Kelly, & Gerin, 2003; Dolezsar, McGrath, Herzig, &, Miller, 2014; Paradies, 2006; Paradies et al., 2015; Pascoe & Richman, 2009). For instance, a recent report found that every one-point increase in self-reported discrimination was associated with a 3-12% increased risk of mortality in an older community-based adult sample (Barnes et al., 2008). It may be important to examine the

contribution of discrimination to the health of particular minority groups in the U.S. who may face a greater health burden associated with their increased exposure to these stressful experiences (Williams et al., 2003). In this regard, although historically marginalized statuses in the U.S. fall along the lines of sex, socioeconomic status (SES), and race, the preponderance of the work examining the health implications of discrimination has focused on African Americans.

Among African Americans, discrimination is a unique source of chronic stress that may act as a non-traditional risk factor for CVD (Clark et al., 1999). CVD encompasses disorders of the heart and blood vessels and is the leading cause of death in the United States (American Heart Association, 2017d; National Center for Health Statistics, 2011). CVD comprises ischemic stroke, coronary heart disease, arrhythmia, and particularly high blood pressure (BP) or hypertension (HTN; American Heart Association, 2017d). Several biological and behavioral determinants including family history, age, gender, obesity, and substance use have been established as traditional risk factors for CVD development (American Heart Association, 2017a, 2017b). However, these risk factors do not fully account for the CVD burden observed in the general population, especially among African Americans (Klonoff & Landrine, 1999). To this end, mounting research has demonstrated that discrimination may be adversely associated with clinical and subclinical CVD risk factors. For instance, across several systematic reviews (e.g., Brondolo et al., 2003; Dolezsar et al., 2014), emerging evidence demonstrates that discrimination may be adversely linked to CVD across multiple endpoints including, atherosclerosis, intima medial thickness, and elevated BP and HTN risk. Altogether, these growing findings demonstrate that discrimination may be implicated in the disproportionate CVD burden among African Americans. However, researchers (see studies reviewed in Brondolo et al., 2003; Dolezsar et al., 2014) continue to call for further exploration of the various pathways linking

these factors. It is widely posited that the chronic stress from discrimination experienced by African Americans substantially contributes to their disproportionate CVD burden for at least two reasons; 1) it may alter cardiovascular functioning via short-term increases in BP and ultimately through chronic physiological alterations (Clark et al., 1999) *and* 2) it can also increase BP indirectly through the psychological, emotional, and behavioral responses an individual may experience in response to the stressor (Bennett, Wolin, Robinson, Fowler, & Edwards, 2005; Klonoff & Landrine, 1996). Therefore, among African Americans, increased exposure to discrimination may be a unique and significant contributor to their disproportionate chronic stress burden, which in turn increases BP levels and subsequently leads to greater HTN risk and ultimately, CVD (Clark et al., 1999; Harrell, 2000). Given the need to reduce the elevated CVD risk among African Americans and calls by researchers to examine alternative pathways that may link discrimination to HTN and CVD, it is essential to further explore this association and any additional moderating factors that may allow greater understanding of this relationship.

SES is one such factor that may influence the linkage between discrimination and CVD. SES, which reflects an individual's access to resources and opportunities, has been conceptualized as a source of chronic stress, particularly for individuals with lower SES, because they are more likely to experience limited means (e.g., lower quality health care, employment, grocery stores, etc.) than individuals with higher SES (Collins et al., 2003; Williams & Collins, 1995). As a source of chronic stress, researchers have posited that SES is a non-traditional risk factor implicated in a host of health endpoints including CVD, contributing to the prevalence and incidence of BP and HTN across all races (Levenson, Skerrett, & Gaziano, 2002; Marmot & Wilkinson, 2006). Importantly, because African Americans are more likely to have a lower SES

(DeNavas-Walt & Proctor, 2014; Williams & Williams-Morris, 2000), it has been proposed that this adds to their disproportionate CVD burden (Frierson et al., 2008; Wyatt et al., 2003). Conversely, higher SES may also serve as a source of chronic stress for African Americans, as it may provide access to resources and opportunities that lead to increased interactions with other dominant racial groups, mainly Whites, which may be stressful and confer increased exposure to discrimination (Farmer & Ferraro, 2005). Thus, it is plausible that the type (e.g., racial), frequency (e.g., daily), and severity (e.g., police) of discrimination African Americans face may differ as a function of whether they have higher or lower SES (Kessler, Mickelson, & Williams, 1999; Sigelman & Welch, 1991; Williams, 1999). In this way, while the specific experiences of discrimination may differ, it is plausible that African Americans with lower and higher SES both experience chronic stress that can negatively impact their cardiovascular health. Considering that the pathways that delineate these experiences are different for these two groups of African Americans, the role of the interactive effects of both SES groups should be explored in relation to CVD outcomes and the overall CVD burden among African Americans. Indeed, studies have demonstrated that discrimination is linked to adverse health endpoints in African Americans at both higher and lower levels of SES (Fuller-Rowell, Doan, & Eccles, 2012; Williams, Priest, & Anderson, 2016). However, to date our understanding of these linkages with CVD is quite limited. For example, few studies explicitly examine whether SES influences the relationship between discrimination and BP (e.g., Krieger & Sidney, 1996), with most adjusting for the contribution of this sociodemographic factor (e.g., Ryan, Gee, & Laflamme, 2006). Thus, a central issue that remains unclear is how the relation of discrimination to BP varies as a function of SES among African Americans.

Sex may also serve as an understudied moderating pathway of the association between discrimination and BP among African Americans. Sex differences in elevated BP and HTN are well established. In the general population, men tend to have higher BP and a greater prevalence of HTN than women (Everett & Zajacova, 2015; Mozzaffarian et al., 2015; Yoon et al., 2015). Among African Americans however, women typically have higher BP levels and HTN rates than men (Mozzaffarian et al., 2015). Similarly, among African Americans, there are sex differences in the prevalence of experiences with discrimination (Borrell et al., 2006; Roberts, Vines, Kaufman, & James, 2008). For example, African American men tend to report greater racial discrimination compared with African American women (Kwate & Goodman, 2015; National Public Radio, Robert Wood Johnson Foundation, & Harvard T.H. Chan School of Public Health, 2017). However, sex differences in experiences of discrimination among African Americans are likely more nuanced than illustrated by prior research because the specific types of discrimination that they experience as a function of their sex has not been well characterized (Kwate & Goodman, 2015). Sex differences in the experiences of discrimination between African American men and women may also vary due to SES (Kwate & Goodman, 2015; Landry & Marsh, 2011). Altogether, there are various complex factors that may shape access to resources and experiences with discrimination, which collectively may contribute to the differences in BP and HTN among African American men and women. In this way, it is plausible that the synergistic interrelations among sex, SES, and discrimination may bear upon the patterning of BP and HTN. However, whether SES and sex concurrently impact the relation of discrimination to BP and HTN among African Americans remains unclear. To this end, the current study will examine whether SES and sex concurrently moderate the relation of discrimination to BP among African Americans.

The following section will provide a brief, but substantive overview of the empirical and conceptual literature examining the association between discrimination and CVD among African Americans, with a more extensive focus on the relation of racial discrimination to BP and HTN. In examining this relationship, the contributions of traditional and non-traditional CVD risk factors will be highlighted, with detailed attention given to SES and sex as potential moderators of the relationship between racial discrimination and BP and HTN among African Americans. Lastly, in this section, the aims and hypotheses of the proposed study will be presented. Finally, the methodology and data analytic procedures will be outlined in the Methods section.

Literature Review

Racial Disparities in Cardiovascular Disease

African Americans in the U.S. have the highest rates of HTN in the world, including Blacks throughout the diaspora (Mozzaffarian et al., 2015). HTN is defined as systolic blood pressure (SBP) > 130mmHg and/or diastolic blood pressure (DBP) > 80mmHg, which reflect the force and contraction of blood flowing through blood vessels, respectively (American Heart Association, 2017c). A recent American Heart Association report (Benjamin et al., 2017) indicated that in the U.S., the overall rate of HTN is 34% (85.7 million), however by race, rates are 45.7%, 33.4%, 29.8%, and 27.3% in African Americans, Whites, Hispanics, and Asians, respectively. Further, when compared by sociodemographic factors such as gender and age, African Americans have higher rates of HTN compared with their White counterparts (Will & Yoon, 2013). Altogether, it is evident that African Americans are disproportionately affected by HTN.

The disproportionate incidence and prevalence of HTN among African Americans is of critical concern and poses a significant threat to public health. According to the American Heart

Association, HTN is a central risk factor for CVD, which is the leading cause of death globally (Mozaffarian et al., 2015). CVD includes heart failure, coronary heart disease (CHD), stroke, and ischemic heart disease (American Heart Association, 2017d). Over the past few decades, African Americans have consistently had high HTN rates that have led to adverse health consequences related to CVD. For example, since 1998, racial disparities in hospitalizations due to chronic cardiovascular conditions, such as uncontrollable HTN, have increased resulting in over 430,000 HTN hospitalizations among African Americans compared with Whites (Doshi, Aseltine Jr., Sabina, & Graham, 2016). The overrepresentation of African Americans in the population of individuals with HTN has contributed to racial disparities in CVD incidence, as almost 50% of African Americans have some form of CVD (Benjamin et al., 2017). Altogether, these disparities in HTN and subsequent CVD onset ultimately contribute to racial inequities in CVD mortality. One-third of the disparity in the number of potential years of life lost between African Americans and Whites is attributable to CVD (Wong, Shapiro, Boscardin, & Ettner, 2002). Similarly, a 2016 report from the National Center for Health Statistics—within the Centers for Disease Control and Prevention (CDC)—on long-term health trends in the U.S. found comparable results in national data. The total years of potential life lost before age 75 (per 100,000 population under age 75) due to CVD was 1,637.9 for African Americans and was 939.2 for Whites. Indeed, these data demonstrate protracted racial differences in the burden of CVD (National Center for Health Statistics, 2017). A preponderance of research has sought to elucidate factors which may explain these disparate outcomes, with a primary emphasis on what are considered to be *traditional risk factors*.

Traditional Cardiovascular Disease Risk Factors

It is essential to identify the various traditional risk factors that typically contribute to HTN when exploring the pathways that delineate racial disparities in CVD. The term traditional risk factors refer to factors that are commonly associated with increased CVD risk. These risk factors were originally established in the Framingham Heart Study. This study was initiated in 1948 and remains an ongoing prospective study of CVD etiology. This study contributed to the shift in the earlier emphasis on CVD treatment to the current focus on CVD prevention (Framingham Heart Study, 2017). The study sought to identify those with the highest risk for future cardiovascular events in a sample of participants who had not yet developed any overt CVD symptoms or experienced any cardiovascular events such as a heart attack or stroke (Framingham Heart Study, 2017). As a result, eventually the term "risk factor" was coined and popularized with regard to the study of CVD in the seminal work, *Factors of Risk in the Development of Coronary Heart Disease* (Kannel, Dawber, Kagan, Revotskie, & Stokes, 1961). Traditional CVD factors, including HTN, include individual-level factors such as obesity, substance use (e.g., cigarette and alcohol usage), sex, and age (Framingham Heart Study, 2017). Extensive amounts of literature have found smoking and alcohol use to be predictors of HTN and CVD (Arboix, 2015; Burns, 2003). Specifically, smoking is associated with increased HTN and CVD risk because it leads to the stiffening of arterial vessels, atherosclerotic plaque progression, thickening of the blood, and the decrease of high-density lipoprotein (HDL) cholesterol, all of which increase BP levels. Heavy alcohol consumption is also linked to increased HTN and CVD risk, as it can raise BP and increase the risk for a host of different CVD outcomes including stroke, heart failure, and arrhythmia (American Heart Association, 2017a). Being overweight or obese, which is reflected by a body mass index (BMI; weight in kilograms divided by the square of height in meters) of $\geq$ 25 kg/m2, is also associated with an increased risk for HTN and several

CVD endpoints (American Heart Association, 2017a; Hubert, Feinleib McNamara, & Castelli, 1983; Mokad et al., 2001). This is because increased body fat causes excess strain on the heart and circulatory system, leading to increased blood flow, widening of blood vessels, and cardiac output (Re, 2009). Moreover, a longitudinal study (Saydah et al., 2014) of over 10,000 participants in the National Health and Nutrition Examination Survey (NHANES) found that HTN prevalence rates were highest among obese individuals (35.7%), followed by those who were overweight (26.4%) Lastly, age is the strongest non-modifiable factor for CVD because as individuals advance in chronological age, there is an increase in the chance of developing high BP due to decreased elasticity in blood vessels, reflecting physiological aging (American Heart Association, 2017a). Altogether, these variables reflect traditional CVD risk factors because they are established markers of both HTN and CVD risk and for many years, were the main focus of research on CVD risk factors.

Despite the abundance of literature conceptualizing traditional CVD risk factors as significant contributors to HTN/CVD risk, solely investigating these factors when analyzing these cardiovascular endpoints may not fully reflect risk factors unique to certain groups. Essentially, traditional risk factors may not fully explain racial disparities in CVD because the incidence and prevalence rates of HTN and CVD in African Americans persist even when controlling for the aforementioned traditional risk factors, as well as other more non-traditional risk factors such as SES related factors, and individual and community level factors (e.g., proportion of families in poverty, index of racial isolation, and index of dissimilarity; data separated and measured by zip code and county level) and characteristics (Finkelstein, Khavjou, Mobley, Haney, & Will, 2004). For example, traditional HTN and CVD risk factors explain >80% of the excess population risk for CHD (Cobb, Kraus, Root, & Allen, 2003). Thus, there is

speculation as to what other factors may explain the unaccounted variance in the disparate CVD rates in the African American population. As a result, researchers have suggested the consideration of more unique *non-traditional risk factors* to better understand the potential pathways that may explain racial disparities in CVD and HTN (Cushman et al., 2008).

Non-Traditional Risk Factors: Social Determinants and Cardiovascular Disease

Protracted racial health disparities have been instrumental in the development of scientifically-based national objectives that explicitly seek to identify and ameliorate factors that precipitate and maintain these disparities. For instance, *Healthy People 2020* was set forth by the Office of Disease Prevention and Health Promotion in the U.S. Department of Health and Human Services (HHS) as a platform to improve the health of all Americans via, "the establishment of benchmarks and monitored progress over time to empower individuals to make informed health decisions, encourage collaboration across communities, and measure the impact of prevention activities" (Office of Disease Prevention and Health Promotion, 2018a). Specific to cardiovascular outcomes, *Healthy People 2020* aims to improve cardiovascular health through better prevention, detection, and treatment of risk factors for heart attack and stroke. These aims are highlighted within the *Healthy People 2020* topic of Heart Disease and Stroke. This topic has 25 objectives including the overall increase of CVD health in the U.S. (objective 1), reduction of the proportion of persons in the population with HTN (objective 5), and the reduction of CHD mortality (objective 2) by 20% (Office of Disease Prevention and Health Promotion, 2018b).

Although *Healthy People 2020* extends upon the goals established in *Healthy People 2010* and overall improvement has been observed, changes have not been sufficient to ameliorate or offset the disproportionate burden of HTN and CVD that remains among African Americans. For example, while there were overall improvements in several Heart Disease and Stroke

objectives (objectives 1, 7, 6a, and 9) for *Healthy People 2010*, African Americans had significantly worse prevalence and death rates than other racial/ethnic groups, with a rate more than twice the best group rate in all three objectives except one (National Center for Health Statistics, 2012). In this regard, *Healthy People 2020* recognizes that the racial disparities in CVD may stem from determinants of health that reflect non-traditional risk factors, which may be of particular relevance for African Americans. Determinants of health outlined by *Healthy People* fall under the broad categories of policymaking, health services, individual behavior, biology, genetics, and most importantly for African Americans—social factors. Within the category of social factors, *Healthy People 2020* included both physical determinants and social determinants of health, the latter of which includes discrimination.

Discrimination as a Social Determinant

Racism has been described as, "a phenomenon that results in avoidable and unfair inequalities in power, resources and opportunities across racial or ethnic groups" (Paradies et al., 2015). In this way, racism can be expressed through beliefs, stereotypes, prejudices, or discrimination. Thus, discrimination is a behavioral expression of racism. On the interpersonal level, the term discrimination is used to refer to differential or unfair treatment based on actual or perceived membership in any disadvantaged group that can manifest in numerous behaviors such as open threats or insults (Berman & Paradies, 2010; Williams, Lavizzo-Mourey, & Warren, 1994). This can also occur institutionally when these beliefs or discriminatory behaviors become deeply embedded in social systems and structures (Berman & Paradies, 2010). When either of these types of discriminatory actions occurs based on phenotypic characteristics or ethnic group affiliation, this constitutes racial discrimination. Since racism is a key determinant of health

(Mays, Cochran, Barnes, & 2007; Paradies, 2006; Williams & Mohammed, 2009), it is plausible that discrimination functions similarly.

A growing literature indicates that discrimination is a well-established social determinant of health. Social determinants of health reflect social (including economic) factors and physical conditions of the environment individuals are immersed in that have important effects on health, whether direct or indirect (Braveman, Egerter, & Williams, 2011), and impact a wide range of health, functioning, and quality-of-life outcomes (Office of Disease Prevention and Health Promotion, 2018a). Discrimination is considered a social determinant because this experience can have a direct or indirect effect on health. In fact, both general and racial discrimination (Steffen, McNeilly, Anderson, & Sherwood, 2003) have been found to impact health via their links to stress (Clark et al., 1999; Kessler et al., 1999; Paradies, 2006; Williams & Mohammed, 2009). In addition to studies where African Americans subjectively reported racial discrimination as a stressor (Brown et al., 2000; Krieger, Smith, Naishadham, Hartman, & Barbeau, 2005), laboratory experiments have demonstrated that discriminatory experiences related to race are associated with physiological stress responses and cardiovascular activity (Bowen-Reid & Harrell, 2002; Guyll, Matthews, & Bromberger, 2001; Jones, Harrell, Morris-Prather, Thomas, & Omowale, 1996; Morris-Prather et al., 1996). Thus, racial discrimination is a chronic stressor that may lead to increased risk of HTN and CVD.

Prevalence of Racial Discrimination among African Americans

In comparison to other racial groups, African Americans consistently and disproportionately experience overall discrimination and racial discrimination. For example, a study using data from the MacArthur Foundation Midlife Development in the United States (MIDUS) survey—a large national general population survey of over 3,000 participants—found

that 33.5% of respondents reported exposure to major lifetime discrimination and 60.9% reported exposure to daily discrimination (Kessler et al., 1999). Across both measures of discrimination, African Americans reported more exposure than Whites. Another study (Barnes et al., 2004) examined experiences of discrimination among approximately 2,000 African Americans and 1,600 Whites and found that African Americans scored higher than their White counterparts on the discrimination subscales of unfair treatment and personal rejection. Regarding racial discrimination specifically, Klonoff and Landrine (1999) found that 96% of African Americans experienced some form of racial discrimination in the past year, and 98% reported experiencing it at some point in their life. Studies have also found that African Americans experience more racial discrimination than Whites and other racial minorities (Chou, Asnaani, & Hofmann, 2012; Rosependa, Richman, & Shannon, 2009). For example, Krieger et al. (2005) utilized the Experiences of Discrimination in a sample of African Americans, Whites, and Latinos. Results were that scores on the measure were highest for African Americans, lower for Latinos, and the lowest for Whites. Thus, for African Americans, the literature has clearly established a pattern of prevalent and disparate experiences of racial discrimination.

In addition to increased prevalence rates of discriminatory experiences, African Americans encounter racial discrimination across various settings and domains. A recent report on a survey of 3,453 racially diverse adults in the U.S., 802 of which were African American, examined discriminatory experiences (National Public Radio, Robert Wood Johnson Foundation, & Harvard T.H. Chan School of Public Health, 2017). Results indicated that the majority of African Americans had experienced racial discrimination across both interpersonal and institutional domains. Within the interpersonal domain, a substantial number of African Americans reported having been called racial slurs (51%), being threatened or harassed due to

their race (35%), hearing insensitive or offensive comments about their race (52%), experiencing racial violence (42%), and being feared by others due to their race (40%). Furthermore, within the institutional domain, African Americans reported experiencing racial discrimination in interactions with police officers (50%) and in the court system (45%), medical settings (32%), and political settings (19%), or when securing resources such as housing (45%) and jobs (56%). Importantly, approximately 57% reported experiencing racial discrimination in their workplace (e.g., equal pay or consideration for promotion). Overall, as shown by these data, African Americans frequently experience racial discrimination across various life domains which are vital to their livelihoods and key for access to resources, but typical to traverse on a daily basis. Further, it must be noted that although African Americans report substantially more experiences of discrimination in comparison to Whites and other racial groups, as well as high levels of different forms of discrimination across different settings, great within-group variability exists among African Americans. Studies suggest that there are many sociodemographic variations in the experience of discrimination among African Americans. Regarding sociodemographic variations that can delineate experiences of racial discrimination—which will be further extrapolated upon later—studies reported differences in discrimination dependent on sex (Fischer & Shaw, 1997; McCord & Ensminger, 1997), SES (Sanders-Thompson, 1996), and age (Peters, 2004; Steffen et al., 2003). Thus, in this way racial discrimination may be a ubiquitous source of chronic stress for African Americans. To understand this pathway linking discrimination to BP, HTN, and CVD, one must understand how stress can lead to these cardiovascular outcomes.

Physiological Pathways Linking Stress to Blood Pressure, Hypertension, and Cardiovascular Disease

Lazarus and Folkman (1984) created the stress and coping theoretical framework, which attempts to elucidate the process of how stress can lead to adverse health outcomes such as HTN and CVD. According to this framework, the stress process begins when an individual perceives a stimulus in the environment as taxing or exceeding their resources. If it is determined that the stimulus cannot be managed effectively, then it may be deemed as a possible source of harm or loss, or *threat* (Clark et al., 1999; Lazarus & Folkman, 1984). Although popular, this framework is not comprehensive, as it does not fully explain the link between stress and health.

The Clark et al. (1999) model can be used to conceptualize this stress process as it pertains to racial discrimination. In this way, a racist stimulus can be perceived as threatening if it exceeds one's resources. This perception of racial discrimination as threatening depends on the various aforementioned moderating (constitutional, sociodemographic, psychological, & behavioral factors) and mediating (coping responses) factors. These factors are of import because each can individually or jointly influence one's perception of an event as racist, as well as their availability of resources or capacity to cope. The psychological process of identifying an event as a stressor, then determining it as a threat, altogether sets the tone for a physiological response. Importantly, this perceptual determination is posited to set off a cascade of physiological responses that may ultimately jeopardize health. Additionally, threats in the context of social interactions can transpire across a continuum that may reflect stimuli that are of more salience to the target than the perceiver.

With regard to the physiological aspect of the stress process, primarily via the hypothalamic pituitary adrenal axis (HPA) and the sympathetic nervous system, a dynamic cascade is initiated. Specifically, a hormonal response including the release of catecholamines and cortisol is initiated, that in turn, contributes to venoconstriction (constriction of veins) and

vasoconstriction (constriction of blood vessels), which increases heart rate and the force of blood flowing through vessels, which impedes blood flow and increases BP as well (Clark et al., 1999; Herd, 1991). The magnitude and duration of these cardiovascular responses to stress partly depend upon the frequency of exposure to the related stressor. Of note, this physiological activation is optimally suited to the management of acute stressors. However, it is posited that the experience of chronic stressors may lead to dysregulation of the HPA and related systems, including cardiovascular function by way of overactivation or chronic activation (Guilliams & Edwards, 2010). Therefore, chronic stress arising from racial discrimination can be a direct pathway to poor cardiovascular health outcomes, such as elevated BP and HTN, as a result of the body's physiological and cardiovascular responses to these stressful events.

Psychosocial and Behavioral Pathways Linking Discrimination to Blood Pressure, Hypertension, and Cardiovascular Disease

Although stress is a pathway that links racial discrimination to BP and HTN, there are other pathways as well. One such pathway is psychosocial, as several psychosocial factors have been linked to discrimination and BP and HTN. One such factor is mental health, which includes distress and depression. Two meta-analyses (Pascoe & Richman, 2009; Schmitt, Branscombe, Postmes, & Garcia, 2014) found that racism and discrimination were significantly associated with psychological distress, life satisfaction, and depression. Depression has also been linked to elevated BP levels (Duncan, James, & Griffiths, 2011; Hare, Toukhsati, Johansson, & Jaarsma, 2014; Mensah & Brown, 2007). In fact, out of 70 articles reviewed that explored the link between racism and discrimination and health, Paradies et al. (2015) found that depression was the most frequently reported mental health outcome (reported in 37.2% of articles).

Another pathway is behavioral, as several behavioral factors have been linked to both racial discrimination and BP and HTN, including BMI. Although this variable can be considered biological, there are several behavioral factors (e.g., diet, physical activity, etc.) that can also contribute to BMI. Out of the 70 articles, Paradies et al. (2015) reviewed that explored the link between racism and discrimination and health, overweight-related outcomes (BMI of overweight or obese, waist circumference, waist-hip ratio, etc.) was the second-highest reported physical health outcome (5.1%), which was preceded only by BP and HTN (reported in 7.2% of articles). The latter finding is consistent with other research that has consistently linked BMI to BP and CVD across the lifespan (Hubert et al., 1983; Mensah & Brown, 2007).

Frameworks Explicating How Racial Discrimination Functions as a Chronic Stressor

At the interpersonal level, the primary pathway through which racial discrimination is posited to impact health is through its role as a chronic stressor (Clark et al., 1999; Harrell, 2000). Stressors are defined as, "environmental demands that tax or exceed the adaptive capacity of an organism, resulting in psychological and biological changes that may place persons at risk for disease" (Cohen, Kessler, & Gordon, 1997, p. 3). Chronic stressors entail repeated or prolonged difficulties (Macarthur Research Network on SES & Health, 2000). They are unique from other types of stressors (e.g., acute) in that they encompass any environmental demands that are ongoing in daily interactions across various settings such as school or work. Due to the prolonged duration of chronic stress, they are more predictive of adverse health outcomes than acute stressors because the pervasive nature of chronic stressors can lead to a heightened state of stress response that is taxing to body systems (Avison & Turner, 1988; Cohen et al., 1997).

For African Americans, racial discrimination functions as a chronic stressor (Clark et al., 1999; Harrell, 2000) which is theoretically and empirically linked to the disproportionate burden

of poor health. With regard to the theoretical framing of racial discrimination as a chronic stressor with a deleterious health impact among African Americans, two theoretical and conceptual works have helped to elucidate these linkages. In 1999, Clark et al. produced their seminal work, "Racism as a Chronic Stressor: The Biopsychosocial Model," that draws upon the biopsychosocial model and the stress and coping framework, which are both central aspects of the fields of Health Psychology and Behavioral Medicine. This work set the groundwork for research linking racial discrimination among African Americans to health over the last 19 years through at least two critical aspects outlined in this work. First, the authors stated that while studies have examined the link between racism and health, this has not yet been done in a comprehensive manner or within a biopsychosocial model. They advocated for using this model because it had been used in prior literature to conceptualize biopsychosocial factors as predictors of health (Anderson, Kiecolt-Glaser, & Glaser, 1994; Jorgensen, Johnson, Kolodziej, & Scheer, 1996). Further, the biopsychosocial model extends upon the stress and coping theoretical framework introduced by Lazarus and Folkman (1984). Thus, since works on racism and health lacked this perspective Clark et al. provided an overview of the contextual biopsychosocial model and made a case for why it should be used to explore the effects of racism. They postulated that this exploration might 1) provide insight into how racial disparities in health are developed and maintained through the negative biopsychosocial effects of chronic stress from racial discrimination, 2) help explain the observed intragroup differences in health outcomes among African Americans, and 3) provide a basis for, or help inform, approaches to health intervention and prevention that emphasize combatting racism to reduce racial health disparities.

Secondly, Clark et al. (1999) outlined the proposed contextual model for the examination of the biopsychosocial effects of racism among African Americans. The model begins with an

individual being exposed to a stimulus in the environment that they experience as racial discrimination. Drawing on the stress and coping model (Lazarus & Folkman, 1984), this would be referred to as *perceived* racial discrimination. However, in this context, we simply utilize the term racial discrimination, as use of the word perceived can discredit the experience as real by allowing the interpretation that the act was misunderstood or unintended (Banks, 2014). Once the environmental demand is deemed as a threat, psychological and physiological arousal and stress responses occur, which are subject to individual and sociocultural characteristics. The occurrence and intensity of these responses depend on constitutional (e.g., family history of HTN or skin tone), sociodemographic (e.g., age, SES, or gender), psychological (e.g., obsessive compulsive disorder or perceived control), and behavioral factors (e.g., or anger expression or Type A behavior), as well as coping responses. Constitutional (Keith & Herring, 1991), sociodemographic (Forman, Williams, & Jackson, 1997; Williams, Yu, & Jackson, 1997), psychological, and behavioral factors (Adams & Dressler, 1988; Pearlin, 1989), are considered to moderate (or influence the strength of) the relationship between racism and health because they either influence the stress process or the perceptions of racism. Coping responses are considered to mediate (or account for) the relationship between racism and health because the intensity and duration of stress responses depend on an individual's available coping resources. (Burchfield, 1979; Clark & Harrell, 1982).

The second work elucidating the linkage between racial discrimination and stress was authored by Harrell in 2000. This work also drew on Lazarus and Folkman's definition of psychological stress, but centered on conceptualizing racial discrimination as a stressor, coining the term 'racism-related stress' (Harrell, 2000). Racism-related stress is defined as, "the race-related transactions between individuals or groups and their environment that emerge from the

dynamics of racism, and that are perceived to tax or exceed existing individual and collective resources or threaten well-being" (Harrell, 2000, p. 44). Further, Harrell defines racism as "a system of oppression based on racial/ethnic group designations in which pervasive ideology of racial superiority and inferiority provides the foundation for structural inequalities, intergroup conflict, prejudice, and discrimination" (Harrell & Sloan-Pena, 2006, p. 1). Based on this definition, Harrell and Sloan-Pena (2006) extend upon the concept of racism to define racial discrimination as, "differential treatment and behavior based on race." They also posited that racial discrimination is the "building block and product of racism." Essentially, racial discrimination can be a chronic stressor for racial minorities because their life stressors can include unique person-environment interactions that involve race, a phenotypic characteristic that cannot be masked.

Harrell (2000) also asserted that racial discrimination could impact well-being across five different domains. These include physical, psychological, social, functional, and spiritual domains. The relationship between racial discrimination and well-being can be influenced by antecedent variables (e.g., age, race, gender, SES, etc.) and internal (e.g., self-esteem or coping style) and external mediators (e.g., social support). Overall, if an individual deems an environmental stimulus as stressful, depending on psychosocial factors, stress responses occur which, over time, can negatively impact health and maintain health disparities. Clark et al. (1999) called for future studies to investigate the relationship between racism and health in a comprehensive, cohesive, and methodologically sound manner, such as their proposed conceptual model. Further, based on the proposed model, mediators and moderators (e.g., gender and SES) should be examined in relation to this association to enhance understanding of how these variables influence the relationship between racism to health.

Since the seminal work by Clark et al. (1999), other researchers have continued to expand their work by further conceptualizing racial discrimination as a chronic stressor that can impact health. For instance, researchers have found that African Americans report experiences of racial discrimination to be stressful (Klonoff & Landrine, 1996; Utsey, 1999). Numerous other studies and reviews have found associations between racial discrimination and stress (Brondolo et al., 2011b; Sellers, Copeland-Linder, Martin, & Lewis, 2006; Smart Richman & Jonassaint, 2008; Williams & Mohammed, 2013), psychological distress (Broman, Mavaddat, & Hsu, 2000; Kwate, Valdimarsdottir, Guevarra, & Bovbjerg, 2003). Studies have also explored (or emphasized the importance of exploring) the various pathways and moderators that can influence this relationship (Harrell et al., 2011; Krieger, Rowley, Herman, & Avery, 1993; Pascoe & Smart Richman, 2009; Williams & Mohammed, 2013). These studies support the notion that the stress stemming from racial discrimination impacts health, and that this association may be influenced by several other factors. The literature also suggests that these linkages may contribute to racial disparities in general health outcomes. Therefore, for African Americans, it is important to examine the link between racial discrimination and CVD, as well as other potential factors that may influence this relationship. Investigations of this nature may help further understanding of racial disparities in CVD among African Americans and mitigate the deleterious effects.

Racial Discrimination and Cardiovascular Disease

Interpersonal-level racial discrimination has been linked to cardiovascular endpoints among African Americans, including several reviews and meta-analyses that explore this relationship either directly or indirectly (e.g., Brondolo et al., 2003; Brondolo et al. 2011a; Dolezsar et al., 2014; Williams & Neighbors, 2001). Literature supporting this link suggests various ways in which this relationship influences CVD, with stress as one such pathway. For

example, Wyatt et al. (2003) reviewed the evidence on the different pathways linking racial discrimination to CVD. The review provides a summary of 19 population-based studies linking racism to CVD outcomes such as HTN, heart attack, intimal-medial thickness (IMT), and angina. The authors suggest that stress influences the pathway through which experiences of interpersonal racial discrimination impact cardiovascular health because these experiences can lead to increased physiological responses (i.e., cardiovascular reactivity) that can increase CVD risk (Clark et al., 1999; Jones, 2001; Manuck, Kasprowicz, & Muldoon, 1990). Furthermore, findings from this review suggest that there are several potential mediating and moderating factors (e.g., SES) influencing the link between racial discrimination and CVD that future studies should further investigate.

More recent studies have linked racial discrimination to CVD. Racial discrimination has been found to be associated with a history of CVD (Chae, Lincoln, Adler, & Syme, 2010), as well as a greater likelihood of arteriosclerosis, minor heart conditions (e.g., angina pectoris and tachycardia), and myocardial infarction (Udo & Grilo, 2017). On the other hand, some studies have found a conditional association between racial discrimination and CVD (e.g., Chae, Nuru-Jeter, Lincoln, & Jacobs, 2012), and no significant relationship between racial discrimination and CVD (e.g., Krieger et al., 2013). More generally, this literature is lacking because many studies adjusted for social factors (e.g., SES and/or sex), despite past literature suggesting exploration of them because they may influence the relationship between racial discrimination and CVD.

Racial discrimination has also been linked to subclinical CVD. Studies have linked racial discrimination to outcomes such as inflammation (as measured by interleukin-6 [IL-6] and C-reactive protein; Cunningham et al., 2012; Kershaw et al., 2016), a higher plaque score (defined as 50% greater thickness in carotid arteries; Troxel, Matthews, Bromberger, & Sutton-Tyrell,

2003), and an increased risk for coronary artery obstruction (Ayotte, Hausmann, Whittle, & Kressin, 2012). It is important to note, however, almost all of these findings were conditional upon the participant's gender, with three of the studies reporting significant findings only in African American women and one reporting them in only African American men. While these studies explored gender differences in the link between racial discrimination and subclinical CVD, they still did not explore the moderating effects of other important social statuses like SES, with some even controlling for this essential factor.

Together, the aforementioned studies support the linkage of racial discrimination to both clinical and subclinical CVD. These studies also highlight how sociodemographic characteristics like sex and SES can influence this link. Further studies should examine these variables in relation to the association between racial discrimination and clinical and subclinical CVD. Since elevated BP and HTN are risk factors for CVD, it is also essential for future studies to explore sex and SES as potential moderating pathways that influence the association between racial discrimination and BP and/or HTN.

Racial Discrimination, Blood Pressure, and Hypertension

The seminal work by Krieger and Sidney that linked racial discrimination to BP was reported in 1996 and utilized data from the Coronary Artery Risk Development in Young Adults (CARDIA) study. The ongoing CARDIA study was initiated in 1984 as a prospective study with the central goal of investigating the determinants and development of clinical and subclinical CVD and CVD risk factors in 5,115 African Americans and Whites in early adulthood (baseline age 25-30 years old; Friedman et al., 1988). Krieger and Sidney (1996) examined the relationship between racial discrimination and BP with the goal of elucidating racial disparities in elevated BP and HTN rates. To assess interpersonal-level experiences of racial discrimination,

CARDIA included a 7-item measure which surveyed exposure to racial discrimination across seven domains, including at school, work, and from the police or courts. In a subsample including 1,974 African Americans, 80% reported experiencing racial discrimination. The African American sample for analysis purposes was stratified by SES (working class vs. professional class) and gender. Analysis of the relationship between racial discrimination and BP within each of the four stratification subgroups revealed different patterns of association between racial discrimination and BP. For example, among African American women characterized as working-class (i.e., non-business owner and/or subordinate employment positions), those who reported experiencing no racial discrimination in any of the seven domains had higher SBP levels compared to other working-class African American women who reported experiencing racial discrimination in one or two of the domains. A similar pattern emerged for working-class African American men, as those who reported experiencing no racial discrimination had higher SBP than working-class African American men who reported experiencing racial discrimination. In sum, these findings suggest that working-class African Americans who reported experiencing no racial discrimination had higher SBP than their counterparts who reported experiencing racial discrimination.

Although these findings are contrary to what has been theoretically posited, Krieger and Sidney (1996) note two plausible explanations. First, some individuals who experience racial discrimination may not acknowledge it. This is consistent with research prior to Krieger and Sidney (1996) that has demonstrated that individuals who belong to groups that experience discrimination are more likely to report that their peers experience discrimination instead of they themselves (Essed, 1991; Gardner, 1980; Mays, 1994). This tendency may be caused by "internalized oppression," or the idea that members of groups that are discriminated against

believe that it is deserved, therefore no longer consider it discriminatory (Krieger & Sidney, 1996). Conversely, those who reported that they did not experience racial discrimination may have in fact experienced it but been unaware due to denial or misattribution to another marginalized social status (e.g., sex). Second, individuals who feel ill-equipped to challenge discrimination they have faced may find it difficult and painful to acknowledge these experiences. This tendency may be especially salient for African Americans, as they may have concerns related to re-enforcing stereotypes of being overly emotional.

Although this seminal work importantly demonstrated the contributions of SES and sex in elucidating the association between racial discrimination and BP, important questions about these linkages remain. For instance, Krieger and Sidney (1996) stratified by SES and sex but did not statistically test whether the variations in the association of racial discrimination to BP empirically differed by SES and sex. Further, no studies have simultaneously assessed these two sociodemographic factors – SES and sex – in relation to racial discrimination and BP. Additionally, they (Krieger & Sidney, 1996) focused on occupational status as a measure of SES, but other indices of SES as a multidimensional construct may provide additional insight. Due to these factors, future research further elucidating the simultaneous roles of sociodemographic factors—including SES and sex—on the relation of racial discrimination to BP is warranted.

In the 22 years since the CARDIA report (Krieger & Sidney, 1996) linked racial discrimination to BP in African Americans, there has been substantial growth in the empirical research in this area. Three comprehensive reviews have explicitly focused on the relation of racial discrimination to BP and/or HTN among African Americans (Brondolo et al., 2011a; Brondolo et al., 2003; Dolezsar et al., 2014). A review of a subset of 26 individual studies examined in these reviews most relevant to the current research (e.g., populations age 18 and

older; studies that included self-report measures of racial discrimination), plus several other studies identified, revealed inconsistent findings. Specifically four demonstrated a direct, positive association with either resting DBP or resting SBP (Clark, 2000; Guyll et al., 2001; Hill, Kobayashi, & Hughes, 2007; Lewis et al., 2009), whereas seven found a null association of racial discrimination with both resting DBP and resting SBP (Barksdale, Farrug, & Harkness, 2009; Beatty Moody et al., 2016; Brown, Matthews, Bromberger, & Chang, 2006; Peters, 2006; Pointer, Livingston, Yancey, McClelland, & Bukoski, 2008; Poston et al., 2001; Salomon & Jagusztyn, 2008). Four additional studies also did not find a direct association between racial discrimination and resting BP but did demonstrate a conditional association (interacting with coping-related or personality characteristics; Clark, 2003; Clark, 2006; Clark & Adams, 2004; Clark & Gochett, 2006).

Three studies found a positive association of racial discrimination with ambulatory blood pressure (ABP; Brondolo et al., 2008; Singleton, Robertson, Robinson, Austin, & Edochie, 2008; Steffen et al., 2003). A fourth study did not find a direct relationship between racial discrimination and ABP but demonstrated a conditional association (interacting with age; Beatty Moody et al., 2016).

Two studies have reported a direct, positive association between racial discrimination and HTN (Din-Dzietham, Nembhard, Collins, & Davis, 2004; Sims et al., 2012), three reported no relationship (Broman, 1996; Brown et al., 2006; Dressler, 1996), and two additional studies did not find a direct association (interacting with country and neighborhood-type where born, Cozier et al., 2006; interaction with level of education, Krieger et al., 2010). Relatedly, other forms of interpersonal-level discrimination not exclusively due to race have also been positively linked to BP in African Americans. At least three studies have found a positive association between

everyday discrimination (also referred to as unfair treatment) and BP (Krieger et al., 2008; Smart Richman, Pek, Pascoe, & Bauer, 2010; Tomfohr et al., 2010).

Doleszar et al.'s (2014) metanalysis of the literature reveals that there is a positive association between racial discrimination and BP/HTN. However, the effect size is quite small, and, as noted above, a number of studies have not found an association between racial discrimination and BP/HTN. Clearly, much work remains to be done in this area. To guide future research, at least two themes emerge from the existing literature should be considered. First, there were methodological challenges in assessment of clinic resting BP/HTN across studies. For example, although racial discrimination influences HTN risk, it may depend on the type of BP being measured (Brondolo et al., 2003; Brondolo et al., 2011a). Additionally, some of the aforementioned studies (e.g., Broman, 1996) noted limitations related to generalizability due to the method used to assess HTN. For generalizability, it may be important to assess both BP levels and HTN by a standardized clinical protocol administered by a trained professional.

Second, there is some empirical evidence in support of the theorized influence of various factors, including sociodemographic, moderating the relation of racial discrimination to BP and HTN. In particular, in terms of sociodemographic factors, age, SES, and sex appear potentially important (Beatty Moody et al., 2016; Dolezsar et al., 2014; Krieger et al., 2010; Krieger & Sidney, 1996; Roberts et al., 2008 Sims et al., 2012). Many studies have controlled for sociodemographic variables, hoping to establish that the relationship between racial discrimination and BP/HTN is independent of sociodemographics. While controlling for these variables is important, it appears equally important to focus on whether different sociodemographic subgroups of African Americans are particularly vulnerable to the influence of racial discrimination through the examination of moderating effects. Indeed, controlling these

variables may limit our understanding of the more complex or nuanced associations between racial discrimination and BP/HTN that involve important sociodemographic factors.

The current research will address the above two concerns, respectively, through the use of a standardized assessment protocol by trained medical staff person to assess BP and HTN, and by examining the moderating role of two key sociographic factors—SES and sex.

Sociodemographic Factors, Racial Discrimination, Elevated Blood Pressure, and Hypertension

Meta-analytical reviews of the literature suggest that experiences of discrimination (Pascoe & Smart-Richman, 2009) including experiences of racial discrimination (Paradies et al., 2015) are negatively associated with a diverse set of physical and mental health outcomes in the general population. Furthermore, the meta-analytic review by Dolezsar et al. (2014) found that racial discrimination was negatively related to elevated BP and/or HTN both in the general population, and for African American samples. However, effect sizes were low in magnitude, and as reported above, there are inconsistent findings across studies. Thus, many researchers have called for examination of psychosocial and sociodemographic moderators of the racial discrimination-BP/HTN relationship. The CDC (2001) suggested that two of the most well-established risk factors for poor cardiovascular health are SES and sex, and research suggests that these sociodemographic risk factors may influence the association of racial discrimination to BP and HTN among African Americans (Dolezsar et al., 2014; Krieger & Sidney, 1996; Roberts et al., 2008). Literature related to the potential moderating role of SES and sex among African Americans is presented below.

Socioeconomic Status as Moderator. There was only one study located that directly tested the moderating effects of SES (i.e., racial discrimination x SES interaction on

hypertension) among African Americans (Roberts et al., 2008), and this study did not find a significant racial discrimination x SES interaction. The authors suggest that the failure to find a moderating effect might be due to the lack of SES diversity in the sample. One additional study stratified the sample by educational attainment level and found that African Americans who reported the highest levels of racial discrimination had the highest levels of HTN (with those reporting no racial discrimination having an intermediate level of HTN), whereas there was no relationship between racial discrimination and HTN for those with a college degree (Krieger et al., 2010). More generally, the Dolezsar et al. (2014) metanalytic review comparing effect sizes across studies examining direct relations between racial discrimination and BP/HTN found evidence of a moderating influence of SES for African Americans. Specifically, they found that the larger the percentage of participants with high school education or less as their highest level of education in the study sample, the greater the effect size in terms of the relationship between racial discrimination and HTN. This analysis was based on ten studies (combined N=3,650). Of note, the same relationship was found for the general population—i.e., when all studies, not just those limited to African Americans, were included in the metanalysis.

The significant racial discrimination x SES finding for African Americans is consistent with the view that low SES African Americans have greater vulnerability to the impact of racial discrimination than higher SES African Americans, due both to greater exposure to chronic environmental stress linked to poverty (e.g., crime, hunger, substandard housing) , and fewer personal and environmental resources to cope with stress (e.g., Baum, Garofalo, & Yali, 1999; Williams & Collins, 1995; Winkleby, Jatulis, Frank, & Fortmann, 1992). More generally, this finding is consistent with the poorer health outcomes associated with lower SES (Calvin et al., 2003; Williams & Collins, 1995), including highest risk for HTN (Levenstein, Smith, & Kaplan,

2001) and CVD (Calvin et al., 2003), and highest rates of morbidity (Mirowsky & Hu, 1996) and mortality (Chapman & Hariharan, 1996; Feldman, Makuc, Kleinman, & Cornoni-Huntley, 1989).- It is important to note that those from low SES backgrounds are not at an increased risk for CVD merely because they are less likely to have access to adequate health care, as access to medical care explains less than 10% of the observed SES differences in CVD risk and development (Klonoff & Landrine, 1999). Indeed, the weathering hypothesis suggests that rapid aging and deteriorating health occur in response to a lifetime of socioeconomic disadvantage Geronimus, 1992; Geronimus, 1996).

The most commonly used measures of SES are education, occupation, and income, with the latter being the most widely used in literature (Macarthur Research Network on SES & Health, 2000). This is because income is considered to be the strongest and most robust predictor of health (McDonough, Duncan, Williams & House, 1997) and it partially mediates the impacts of other SES indicators (House & Williams, 2003). However, there are difficulties with using income as a measure of SES. For instance, participants may either be hesitant to share their income or cannot recall the exact amount. Therefore, other indicators of SES related to income, such as poverty may also be appropriate to use (Kington & Smith, 1997). The present study will utilize poverty as a measure of SES and examine the hypothesis that racial discrimination will be more strongly inversely related to BP and HTN among African Americans living in poverty than those not living in poverty. Given that working-class, middle-class and upper-class African Americans are included together in the non-poverty category in the data set used for this study, an alternative hypothesis suggested by several studies (e.g., Peters, 2004; Roberts et al., 2008) of a U-shaped or J-shaped relationship form of the racial discrimination x SES interaction cannot

be examined (where African Americans at both the lower and higher ends of the SES spectrum are more vulnerable to racial discrimination compared to those in the middle).

Sex as Moderator. Only two studies were located that directly tested the moderating effect of sex (i.e., racial discrimination x sex interaction) on BP/HTN among African Americans (Roberts el al., 2008; Sims et al., 2012). In both studies, the interaction was not significant (a third study reported having conducted a racial discrimination x sex interaction analysis as part of a preliminary set of analyses but did not report the finding). More generally, the Dolezsar et al. (2014) meta-analytic review comparing effect sizes across studies examining direct relations between racial discrimination and BP/HTN did find evidence of a moderating influence of sex for African Americans. Specifically, they found that the greater the percentage of male participants in a sample the larger the effect size in terms of the relationship between racial discrimination and DBP. This analysis was based on 21 studies (combined N=7,965). On the other hand, a null relationship was found for the general population—i.e., when all studies, not just those limited to African Americans, were included in the metanalysis.

Fewer health behaviors and coping resources for African American men than women may potentially provide at least a partial explanation for the Dolezsar et al. significant moderator effect findings for African Americans. African American men typically have poorer nutrition, fewer interpersonal relationships, and engage in fewer health-responsibility activities (e.g., exercise; Johnson, 2005; Walcott-McQuigg, Logan, & Smith, 1994). Furthermore, African American men generally report more experiences of racial discrimination across multiple settings such as with police, seeking housing, at work, getting a job, and voting (Borrell, et al., 2006; Kwate & Goodman, 2015; National Public Radio, Robert Wood Johnson Foundation, & Harvard T.H. Chan School of Public Health, 2017; Roberts et al., 2008). Greater vulnerability to

the negative impacts of racial discrimination for African American men than women may result from fewer personal resources available to cope with racial discrimination across the multiple settings in which racial discrimination is experienced.

On the other hand, African American women might plausibly be expected to be more vulnerable to the negative effects of racial discrimination due to sexed racism, a unique form of discrimination and oppression due to the simultaneous effects of being a woman and being African American (Essed, 1991). These unique experiences may explain why most of the national data and some empirical studies report African American women have poorer cardiovascular health outcomes than African American men, because they may experience more frequent and prolonged physiological stress responses as a result of this unique position. This is consistent with literature stating that African American women report more distress in response to stressful stimuli when compared to African American men (Morris-Prather et al., 1996).

Overall, there appears to be viable competing perspectives as to whether African American men or African American women might be expected to be more vulnerable to racial discrimination in terms of its impact on BP/HTN. Thus, in the current study examination of the racial discrimination x sex interaction will be exploratory, not directional.

SES and Sex as Joint Moderators. The literature reviewed thus far suggests that social statuses, such as SES and sex, influence experiences of racial discrimination and resulting cardiovascular health outcomes. Whether one experiences racial discrimination and subsequently deems it to be stressful appears to depend in part on the individual's SES and sex. Indeed, the seminal study by Krieger and Sidney (1996) suggested the possibility of complex, unexpected three-way interactions among SES, sex, and racial discrimination, although the authors did not statistically examine such possible effects. The synergistic influence of SES and sex might be

expected to significantly impact exposure to and experience of racial discrimination and resulting physiological responses and cardiovascular reactivity to stress. Thus, it is plausible that these social categories taken together uniquely impact the linkages of racial discrimination to BP/HTN and CVD beyond their respective individual contributions. Given the absence of evidence related to the expected form of the interaction, the current study will examine in an exploratory fashion the possibility of a three-way racial discrimination x SES x sex interaction.

Proposed Covariates and Control Variables

Since established CVD risk factors may potentially confound the possible interactive relations of racial discrimination, poverty status, and sex to SBP and DBP, statistical analyses will be adjusted for factors—age, BMI, depression, and substance abuse.

Age has been associated with BP levels, HTN, and exposure to racial discrimination. For example, BP levels typically increase with age across the entire population (American Heart Association, 2017a). Thus, age will serve as a covariate. Depression has also been linked to elevated BP levels (Duncan et al., 2011; Hare et al., 2014; Mensah & Brown, 2007) and will be included as a covariate in the analyses.

Additional factors including BMI and substance use will also serve as covariates due to their association with CVD. BMI has been consistently associated with resting BP and CVD across the lifespan (Hubert et al., 1983; Mensah & Brown, 2007). Substance use will also serve as a covariate due to its association with CVD outcomes. Specifically, smoking and alcohol use has been linked to elevated BP levels and BP and HTN (Burns, 2003; Davis, Vinci, Okwuosa, Chase, & Huang, 2007; Utsey, Ponterotto, Reynolds, & Cancelli, 2000).

Proposed Study

Statement of the Problem

There have been clearly defined and established traditional risk factors associated with the development of CVD including HTN. However, studies have found that half or more of CVD risk remains unexplained when these traditional risk factors have been accounted for, particularly among African Americans and those from low SES backgrounds. Thus, researchers have increasingly explored the role of social determinants in explaining CVD risk, especially among vulnerable sociodemographic subgroups. In that regard, racial discrimination has been identified as a potential social determinant of CVD risk, mainly via a pathway of stress. There is compelling evidence detailing how experiences of racial discrimination act as sources of chronic stress disproportionately affecting the health of African Americans. Further, the research demonstrates that these experiences of racial discrimination can ultimately lead to greater CVD risk in part through elevations in BP levels and HTN status. These linkages may partially account for the existing disparities in the elevated risk and development of CVD among African Americans. Importantly, experiences of racial discrimination may partially depend upon other social statuses occupied by African Americans including SES and sex. Due to the potential linkages of racial discrimination, SES, and sex to CVD, as well as the unique ways these variables can interact to influence one another, examination of this relationship is important. To date, a number of studies have explored the association of racial discrimination with elevated BP and HTN. However, only a handful have directly examined the moderating effects of SES and sex. Thus, there is limited understanding of how these social statuses independently and interactively influence the association of racial discrimination with BP and HTN in African Americans. This study seeks to address the gaps in existing knowledge and literature by investigating whether the relation of racial discrimination to BP and HTN is moderated by SES and sex among African Americans.

Proposed Hypotheses and Exploratory Analyses

The present study will be a secondary data analysis of the Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS) study (Evans et al., 2010). The HANDLS dataset will be used to address the following aims, hypothesis, and exploratory analyses:

Aim 1: Examine whether SES (as indicated by poverty status of below or above) moderates the association of racial discrimination with BP and HTN.

Hypothesis 1: There will be a significant two-way interaction of racial discrimination and poverty status with respect to SBP and DBP after adjustment for age, BMI, depression, substance use, and HTN medication. Racial discrimination will be more strongly related to SBP and DBP for individuals in poverty versus those with a non-poverty status.

Hypothesis 2: There will be a significant two-way interaction of racial discrimination and poverty status with respect to HTN after adjustment for age, BMI, depression, and substance use. Racial discrimination will be more strongly related to HTN for individuals in poverty versus those with a non-poverty status.

Aim 2: Examine whether sex moderates the association of racial discrimination to BP and HTN.

Exploratory Analysis 1: The two-way interaction of racial discrimination and sex with respect to SBP and DBP after adjustment for age, BMI, depression, substance use, and HTN medication will be examined in exploratory fashion.

Exploratory Analysis 2: The two-way interaction of racial discrimination and sex with respect to HTN after adjustment for age, BMI, depression, and substance use will be examined in exploratory fashion.

Aim 3: Examine whether SES (as indicated by poverty status of below or above) and sex moderate the association of racial discrimination to BP and HTN.

Exploratory Analysis 3: The three-way interaction of racial discrimination, poverty status, and sex with respect to SBP and DBP after adjustment for age, BMI, depression, substance use and HTN medication will be examined in exploratory fashion.

Exploratory Analysis 4: The three-way interaction of racial discrimination, poverty status, and sex with respect to HTN after adjustment for age, BMI, depression, and substance use will be examined in exploratory fashion.

Research Design and Methods

Parent Study Procedure

HANDLS is an ongoing, longitudinal study conducted by the Health Disparities Research Section of the National Institute on Aging (NIA) Intramural Research Program. The primary objective is to examine contributing factors to age-related disparities in health and disease attributable to race and SES. Chosen to represent adults from diverse socioeconomic backgrounds, participants were recruited for the baseline sample. They were selected from 13 neighborhoods in Baltimore, Maryland based on data from the 2000 Census. Field interviewers invited one to two participants from each identified residence to participate in the study. Out of the 32,959 households that recruiters visited, there were 14,799 potentially eligible individuals in 9,904 households.

In order to be eligible for HANDLS, participants had to identify as African American or White, be between 30-64 years old at the time of recruitment, be able to give informed consent, be able to complete at least five data measures (medical history, physical performance, cognitive testing, dietary recall, audio questionnaire, body composition, carotid Doppler, or pulse wave

velocity) on the medical research vehicle (MRV), and provide a valid photo identification and verifiable address. Individuals who were within six months of undergoing cancer treatment or had been previously diagnosed with AIDS were excluded. Participants who had uncontrollable HTN at the time of their MRV visit were excluded from examinations and recalled in subsequent waves. Of the 8,150 individuals that met initial screening criteria, 3,722 met all study inclusion criteria. This sample was 45% male and 59% African American, and 41% reported household incomes below the 125% poverty threshold.

Following the approval of the study protocol by the Institutional Review Board at the National Institute of Environmental Health Sciences, phase I was administered to individuals who consented to participate in HANDLS. They completed a household survey to obtain demographic information and psychosocial, dietary, and nutritional measures. Over the next six weeks, phase II was administered. This required participants to meet with a doctor or nurse practitioner who visited their neighborhoods on a mobile MRV. On the MRVs, participants provided information regarding their medical history, underwent a comprehensive physical examination, and completed biomedical, psychosocial, neuropsychology, and physical performance assessments. A total of 2,802 participants completed both phases of data collection during Wave 1. HANDLS data collection is ongoing and participants are reevaluated approximately every three to four years.

The present study will utilize cardiovascular and discrimination data from the baseline measurement (HANDLS Wave 1, August 2004 – March 2009). Specifically, participants in the proposed study will be a subsample of the HANDLS cohort who completed both phases of the HANDLS protocol. This subsample consists of 1,408 African American adults (mean age = 47.61, *SD* = 9.2; 44% male; 46% below the 125% poverty line) who completed discrimination

measures assessments during the first wave of data collection. To be included in the present data analyses, participants must have relevant racial discrimination, BP, and sociodemographic data from Wave 1 of data collection. However, data imputation was performed for all outcome variables that had <10% missing within each race, poverty status, sex subgroup. Multiple regression analysis (using sex, racial discrimination, and poverty status as predictors) was used for imputation for the purpose of replicability.

Measures

Racial Discrimination. Racial discrimination was assessed with a 6-item measure widely used in past epidemiological research (e.g., CARDIA study; Krieger, 1990), which examined experiences of racial discrimination across multiple settings. These settings include at school; getting hired or getting a job; at work; getting housing; getting medical care, and from the police or in the courts. To obtain these responses, the questionnaire prompts participants to answer the following question for each of the nine aforementioned settings, "Have you ever experienced discrimination, been prevented from doing something, or been hassled or made to feel inferior in any of the following situations because of your race, ethnicity, or color?" The dichotomous yes/no responses for each of these 6 items were added to obtain a total raw score, with higher scores indicating a greater number of experiences of racial discrimination.

Blood pressure. SBP and DBP levels were used to assess HTN risk. The standard brachial artery auscultation method was used, with the participant's arm at a 90-degree angle and their palm facing up. Two measures of SBP and two measures of DBP were obtained, one from each arm after participants rested for 5 minutes in a seated position with legs uncrossed. For the current analyses, the participants' two SBP and two DBP measures will be averaged to obtain a

mean SBP and mean DBP, respectively. These variables contain imputed values in the current study.

Hypertension and antihypertensive treatment. HTN was defined as average SBP ≥140mmHg or DBP ≥90mmHg or prior diagnosis of HTN (per self-reported medical history) or use of antihypertensive medication (per participants' pharmacy bottles). Treatment with antihypertensive medications will be included as a covariate in SBP and DBP analyses. Antihypertensive medication treatment will be dichotomized such that treatment = 1 and no treatment = 0.

Sociodemographics. The demographic variables that will be used in this study include self-reported age, sex, and SES. Age was measured continuously in years. Sex was dichotomized as men = 1 and women = 0. Poverty status, assessed in HANDLS via family income as a function of household size, was used as a measure of SES because pilot testing revealed that participants were better able to reliably report this status as opposed to a specific annual income. Poverty status was dichotomized using the 2004 Federal poverty guideline (e.g., $18,850 per year for a family of four; HHS, 2014). Poverty (coded as 1) was defined as family income below 125% of the poverty threshold, and non-poverty status (coded as 0) was defined as a family income above 125% of the poverty threshold.

Anthropometric measurement. Measures of height and weight were taken from calibrated equipment, with participants in an upright position wearing no shoes and a lightweight hospital gown. Body mass index (BMI) is a participant's weight in kg divided by his or her height in m^2. In the present study, this variable contains imputed values.

Depression. Depressive symptoms were assessed using the Center for Epidemiological Studies-Depression (CES-D; Radloff, 1997) scale, a 20-item self-report instrument that has been

widely used and validated in community-based epidemiological studies (Husaini, Neff, Harrington, Hughes, & Stone, 1980; Roberts, 1980; Weismann, Sholomskas, Pottenger, Prusoff, & Locke, 1977). Reliability analyses indicate that this measure has high internal consistency (alpha) with coefficients ranging from 0.63 to 0.93 (Devins et al., 1988). For the general population, it is estimated to be 0.85, and for within patient samples it is estimated to be 0.90 (Radloff, 1977). Furthermore, test re-test reliability from two weeks to 12 months is in the moderate range (.45-.70). In the present study, this variable contains imputed values.

Substance use. Data on smoking status and alcohol use were obtained from the medical history from Phase II of the study. History of smoking cigarettes and drinking alcohol were each dichotomized to indicate ever (coded as 1) or never (coded as 0) used. The "never" category encompassed "never tried" and "never used regularly." The "ever" category encompassed "formerly used" (> 6 months ago) and "currently use" (within the past 6 months).

Power Analysis

Power analysis was conducted using the G*Power 3.1.9.2 statistical software. The baseline sample of 1,408 participants with 9 predictors (including all interaction terms and covariates from the first model in each series of analyses) is powered (1-β = .99) to detect a small to medium Cohen's f^2 effect size of .030 at conventional levels of alpha (.05). Results from the power analysis predict that power will not attenuate at the smaller sample sizes and that detection of a small f^2 effect size is as likely as in the larger samples.

Data Analytic Plan

All data analyses will be conducted with Statistical Package for the Social Sciences (SPSS), version 25 (IBM, 2017). Participants will be excluded from the analyses if they indicate

a history of heart failure, stroke, dementia, HIV or AIDS, kidney dialysis, Parkinson's disease, or schizophrenia. All analyses will include adjustments for age, smoking status, alcohol use, BMI, and depression, with HTN medication serving as an additional covariate just for the SBP and DBP analyses.

For hypotheses 1 and 2, a series of multivariable regression analyses (linear for SBP and DBP; logistic for dichotomous HTN) will be conducted to assess potential independent and interactive associations of racial discrimination and poverty to BP and HTN, respectively. Specifically, the covariates (step 1), racial discrimination sum score and poverty status (step 2), and their interaction term (racial discrimination x SES; step 3) will be used to predict SBP, DBP, and HTN.

For the first two exploratory analyses, a series of multivariable regression analyses (linear for SBP and DBP; logistic for dichotomous HTN) will be conducted to assess potential independent and interactive associations of racial discrimination and sex to SBP and DBP and HTN respectively. The covariates (step 1), racial discrimination sum score and sex (step 2), and their interaction term (racial discrimination x sex; step 3) will be used to predict SBP and DBP and HTN respectively.

For the third and fourth exploratory analyses, a series of multivariable regression analyses (linear for SBP and DBP; logistic for dichotomous HTN) will be conducted to assess the three-way interaction of racial discrimination, poverty status, and sex predicting SBP and DBP, and HTN, respectively. The covariates, racial discrimination sum score, poverty status, and sex, their two-way interaction terms (racial discrimination x SES, racial discrimination x sex, SES x sex; step 3) and the three-way interaction term (racial discrimination x SES x sex) will be used to predict SBP, DBP, and HTN, respectively. The PROCESS macro for SPSS, Version 2.16

(Hayes, 2013), will be used to probe for the significant three-way interactions, as well as significant simple effects.

The two hypotheses and the four exploratory analyses will be evaluated using null hypothesis significance testing with alpha values at the .05 level. Predictors with a $p < .05$ will be judged significant, rejecting the relevant null hypothesis. Effect sizes (η^2_{partial}) will subsequently be calculated for all significant effects.

Results

Preliminary Analyses

Data screening. Data were screened to ensure that assumptions were met, and potential data entry or coding errors were addressed. Specifically, data were screened to identify possible outliers, non-normality, and non-linearity. The linearity (as well as homoscedasticity) of residuals was examined as part of the screening process for multivariate normality (Kline, 2011). The outcome variables of DBP and SBP have a symmetrical distribution with a skewness of .21 and .56, respectively. The predictor variable racial discrimination also had a symmetrical distribution with a skewness of .84. Preliminary data screening via visual inspection of histograms, normal Q-Q plots, and box plots revealed no significant violations of normality for the outcome variable distributions.

Descriptives. There were 1,408 participants (56.3% female, $n = 792$; 43.8% male, $n = 616$) who met inclusion criteria for the present study. Participants ranged in age from 30 to 64 years ($M = 47.6$, $SD = 9.26$), and the sample had a slight majority of individuals living above the poverty line (54%, $n = 761$). Approximately 46.4% ($n = 654$) of the sample had a diagnosis of hypertension, although only 29.7% ($n = 418$) reported having a prescription for antihypertensive medications. Additionally, 68.6% ($n = 966$) of the sample was considered overweight or obese

(BMI > 25). Table 1 presents sample characteristics for sociodemographic variables, predictors, and covariates.

Relations among study variables. Table 2 contains bivariate correlations among all study variables. All but one of the covariates (cigarette smoking status) were significantly related to one or more of the criterion variables, with age, BMI, and prescribed antihypertensive medication significantly related to all three criterion variables. Counter to expectation, neither racial discrimination nor poverty status were related to any of the criterion variables. However, sex was significantly related to two of the three. Specifically, men had higher levels of DBP (r = .09, p < .01) and lower levels of HTN (r = -.09, p < .01) relative to women.

Sex and racial discrimination were positively related to each other, with men reporting higher racial discrimination scores (r = .22, p < .01). As expected, the three criterion variables were positively and significantly related to each other, with correlations ranging from .37 (DBP and HTN) to .52 (SBP and HTN).

Primary Analyses

The present study had three primary aims. The first aim was to examine the 2-way interaction of racial discrimination and SES in predicting SBP, DBP, and HTN. The second aim was to examine the 2-way interaction of racial discrimination and sex in predicting SBP, DBP, and HTN. The third aim was to examine the 3-way interaction of racial discrimination, SES, and sex in predicting SBP, DBP, and HTN. Hierarchical multiple linear regression analyses were used when SBP and DPB were the criterion variables, whereas hierarchical logistic regression analyses were used when HTN was the criterion variable.

Systolic blood pressure. As shown in Tables 3-5, linear regression analyses with SPB as the criterion revealed one significant two-way interaction effect and no significant three-way

interaction effect. The two-way interaction effect of racial discrimination and poverty status on SBP (aim 1) was significant (Table 3). The two-way interaction effect of racial discrimination and sex on SBP (aim 2) was not significant (Table 4). Lastly, the three-way interaction effect of racial discrimination, poverty status, and sex on DBP (aim 3) was not significant (Table 5). Details of the relevant model for each aim is as follows.

Aim 1: Racial discrimination x poverty status. At step 1 of the analysis, age, depressive symptoms, BMI, prescribed anti-HTN medication, alcohol status, and cigarette status were entered, together accounting for a significant 14.8% (p = .00) of the variance in SBP. At step 2, racial discrimination and poverty status were added, together accounting for an additional, non-significant 0.3% (p = .07) of the variance in SBP. Poverty status (β =.06, p = .02) was significantly related to SBP, but racial discrimination (β = .00, p = .88) was not. At step 3 of the analysis, the two-way interaction term of racial discrimination x poverty status was entered, explaining an additional significant 0.3% (p = .04) of the variance in SBP (β =-.22, p = .04). The percentage of variance explained was near zero, not even reaching the level of a small effect size (1%).

Simple effect analysis revealed non-significant effects of discrimination on SBP for each SES group (see Figure 1). Analyses indicted that for African Americans living above the poverty line, SBP levels increased as they experienced more racial discrimination. Specifically, for each unit increase in racial discrimination, SBP increased by .46. Conversely, for African Americans living in poverty, SBP levels decreased as they experienced more racial discrimination. Specifically, for each unit increase in racial discrimination, SBP decreased by .44. The pattern is not consistent with my hypothesis that African Americans living in poverty would be more vulnerable to the BP effects of racial discrimination than those living above the poverty line. The

opposite occurred, as the significant interaction indicates that the group living above the poverty line who experienced more racial discrimination may be relatively more susceptible to elevated SBP than those living in poverty (whose SBP, in contrast, decreased with increasing racial discrimination).

The overall model significantly predicted resting DBP (R = .39 F(9, 1389) = 27.96, p = .00) and accounted for 15.3% of the variance in SBP. There were other significant effects in the final model, but they will not be detailed because the focus of the analysis—the interaction—was significant.

Aim 2: Racial discrimination x sex. At step 1 of the analysis, age, depressive symptoms, BMI, prescribed anti-HTN medication, alcohol status, and cigarette status were entered, together accounting for a significant 14.8% (p = .00) of the variance in SBP. At step 2, racial discrimination and sex were added, together accounting for an additional, non-significant 0.3% (p = .10) of the variance in SBP. Sex (β =.06, p = .03) was significantly related to SBP, but racial discrimination (β = -.01, p = .76) was not. At step 3 of the analysis, the two-way interaction term of racial discrimination x sex was entered, explaining an additional non-significant 0.1% (p = .22) of the variance in SBP. Thus, exploratory analysis revealed that there was not a significant racial discrimination x sex interaction (β =-.14, p = .22).

The overall model significantly predicted resting SBP (R = .39 F(9, 1389) = 27.50, p = .00) and accounted for 15% of the variance in SBP. There were three significant effects in this final model, all from covariates. Resting SBP was positively associated with (1) age, β = .28, p = .00, (2) BMI, β = .19, p = .00, and (3) prescribed anti-HTN medication, β =.11, p = .00.

Aim 3: Racial discrimination x SES x sex. At step 1 of the analysis, age, depressive symptoms, BMI, prescribed anti-HTN medication, alcohol status, and cigarette status were

entered, together accounting for a significant 14.8% (p = .00) of the variance in SBP. At step 2, racial discrimination, poverty status, and sex were added, together accounting for an additional, significant 0.7% (p =.01) of the variance in SBP. Poverty status (β = .06, p =.01) and sex (β = .06, p =.02) were significantly related to SBP, but racial discrimination was not (β = -.01, p =.72). At step 3 of the analysis, the two-way interactions terms of racial discrimination x poverty status, racial discrimination x sex, and poverty status x sex were entered, explaining an additional non-significant 0.4% (p = .10) of the variance in SBP. None of the individual two-way interaction terms were significant. At step 4, the three-way interaction term of racial discrimination x poverty status x sex was entered, explaining an additional non-significant 0% (p =.41) of the variance in SBP. Thus, the exploratory analysis revealed that there was not a significant racial discrimination x poverty status x sex interaction (β = -.15, p =.41).

The overall model significantly predicted resting SBP (R = .40 F(13, 1385) = 20.02, p = .00) and accounted for 15.8% of the variance in SBP. There were three significant effects in this final model, all from covariates. Resting SBP was positively associated with (1) age, β = .28, p = .00, (2) BMI, β = .20, p = .00, and (3) prescribed anti-HTN medication, β =.11, p = .00.

Diastolic blood pressure. As depicted in Tables 6-8, linear regression analyses with DPB as the criterion revealed no significant two-way interaction effects and one significant three-way interaction effect. The two-way interaction effect of racial discrimination and poverty status on DBP (aim 1) was not significant (Table 6). The two-way interaction effect of racial discrimination and sex on DBP (aim 2) was also not significant (Table 7). Lastly, the three-way interaction effect of racial discrimination, poverty status, and sex on DBP (aim 3) was significant (Table 8). Details for the relevant model for each aim is as follows.

Aim 1: Racial discrimination x poverty status. At step 1 of the analysis, age, depressive symptoms, BMI, prescribed anti-HTN medication, alcohol status, and cigarette status were entered, together accounting for a significant 3.0% (p = .00) of the variance in DBP. At step 2, racial discrimination and poverty status were added, together accounting for an additional, non-significant 0.3% (p = .10) of the variance in DBP. Neither racial discrimination (β = .03, p =.27) nor poverty status (β = .05, p = .07) were significantly related to DBP. At step 3 of the analysis, the two-way interaction term of racial discrimination x poverty status was entered, explaining an additional non-significant 0.2% (p = .11) of the variance in DBP. Thus, contrary to the hypothesis, there was not a significant racial discrimination x poverty status interaction (β =-.18, p = .11) for resting DBP.

The overall model significantly predicted resting DBP (R = .19 F(9, 1389) = 5.56, p = .00) and accounted for 3.5% of the variance in DBP. There were three significant effects in this final model, one from a predictor and two from covariates. Resting DBP was positively associated with (1) poverty status, β = .22, p = .04, (2) prescribed anti-HTN medication, β = .11, p = .00, and (3) BMI, β =.08, p = .01.

Aim 2: Racial discrimination x sex. At step 1 of the analysis, age, depressive symptoms, BMI, prescribed anti-HTN medication, alcohol status, and cigarette status were entered, together accounting for a significant 3.0% (p = .00) of the variance in DBP. At step 2, racial discrimination and sex were added, together accounting for an additional, significant 1.8% (p = .00) of the variance in DBP. Sex (β =.14, p = .00) was significantly related to DBP, but racial discrimination (β = .00, p = 1) was not. At step 3 of the analysis, the two-way interaction term of racial discrimination x sex was entered, explaining an additional non-significant 0% (p = .57) of

the variance in DBP. Thus, the exploratory analysis revealed that there was not a significant racial discrimination x sex interaction (β =-.07, *p* = .57) for DBP.

The overall model significantly predicted resting DBP (R = .22 F(9, 1389) = 7.8, *p* = .00) and accounted for 4.8% of the variance in DBP. There were three significant effects in this final model, all from covariates. Resting DBP was positively associated with (1) BMI, β = .11, *p* = .00, (2) prescribed anti-HTN medication, β = .12, *p* = .00, and (3) depressive symptoms, β =.06, *p* = .03.

Aim 3: Racial discrimination x poverty status x sex. At step 1 of the analysis, age, depressive symptoms, BMI, prescribed anti-HTN medication, alcohol status, and cigarette status were entered, together accounting for a significant 3.0% (*p* = .00) of the variance in DBP. At step 2, racial discrimination, poverty status, and sex were added, together accounting for an additional, significant 2.1% (*p* =.00) of the variance in DBP. Poverty status (β = .06, *p* =.03) and sex (men; β = .15, *p* =.00) were positively and significantly related to DBP, but racial discrimination was not significantly related (β = -.00, *p* =.95). At step 3 of the analysis, the two-way interactions terms of racial discrimination x poverty status, racial discrimination x sex, and poverty status x sex were entered, explaining an additional non-significant 0.4% (*p* = .11) of the variance in DBP. None of the individual two-way interaction terms were significant. At step 4, the three-way interaction term of racial discrimination x poverty status x sex was entered, explaining an additional significant 0.3% (*p* =.04) of the variance in DBP. Thus, exploratory analysis revealed that there was a significant racial discrimination x poverty status x sex interaction (β = -.39, *p* =.04). The percentage of variance explained was near zero, not even reaching the level of a small effect size (1%).

Simple effect analysis revealed only one significant effect related to the three-way interaction: African American men with a poverty status who reported experiencing higher levels of racial discrimination had lower levels of resting DBP (see Figure 2). Specifically, for this group, for each unit increase in racial discrimination, DBP decreased by .64 (B = -.64, SE = .33, p = .05). A similar, but non-significant pattern of DBP decreasing as racial discrimination increased emerged for high SES African American women (B = -.07, SE = .30, p = .82). Conversely, a non-significant pattern suggested that for African American men living above the poverty line (B = .35, SE = .29, p = .23) and African American women with a poverty status (B = .19, SE = .30, p = .53), DBP levels increased as experiences of racial discrimination increased.

The overall model significantly predicted resting DBP (R = .24 F(13, 1385) = 6.59, p = .00) and accounted for 5.8% of the variance in resting DBP.

Hypertension status. As shown in Tables 9-11, logistic regression analyses revealed no interaction effects for HTN. Neither the two-way interaction effect of racial discrimination and poverty status on HTN (aim 1; Table 9), the two-way interaction effect of racial discrimination and sex on HTN (aim 2; Table 10), nor the three-way interaction effect of racial discrimination, poverty status, and sex on DBP (aim 3; Table 11) were significant. Details for the relevant model for each aim is as follows.

Aim 1: Racial discrimination x poverty status. In block 1 of the analysis, age, BMI, depression, and alcohol and cigarette status were entered into the logistic regression equation, together accounting for a significant 25.6%) of the variance (p = .00) in HTN. Both racial discrimination and poverty status were added in block 2, together accounting for an additional, non-significant 0.3% (p = .20) of the variance in HTN. Neither racial discrimination (B = -.02, p = .53) or poverty (B = .21, p = .09) were significantly related to HTN. In block 3 of the analysis,

the two-way interaction term of racial discrimination x poverty status was entered into the regression equation, explaining an additional, non-significant 0% (p = .81) of the variance in HTN. Thus, counter to my hypothesis, the analysis revealed that there was not a significant racial discrimination x poverty status interaction (B = .02, p = .81) for HTN.

The overall model significantly predicted HTN (χ^2 (8) = 299.17, p = .00, -2 log likelihood = 1621.68). There were three significant effects in the final model, all from covariates. HTN was associated with (1) increased age, B = .10, SE = .01, p = .00, (2) more depressive symptoms, B = .02, SE = .01, p = .00, and (3) higher BMI, B = .07, SE = .01, p = .00.

Aim 2: Racial discrimination x sex. In block 1 of the analysis, age, BMI, depression, and alcohol and cigarette status were entered into the regression equation, together accounting for a significant 25.6% of the variance (p = .00) in HTN. Both racial discrimination and sex were added in block 2, together accounting for an additional, non-significant 0.1% (p = .64) of the variance in HTN. Neither racial discrimination (B = -.01, p = .67) nor sex (B = -.09, p = .47) were significantly related to HTN. In block 3 of the analysis, the two-way interaction term of racial discrimination x sex was entered into the regression equation, explaining an additional, non-significant .01% (p = .27) of the variance in HTN. Thus, the exploratory analysis revealed that there was not a significant racial discrimination x sex interaction (B = .07, p = .27) for HTN.

The overall model significantly predicted HTN (χ^2 (8) = 297.98, p = .00, -2 log likelihood = 1622.87). There were three significant effects in the final model, all from covariates. HTN was associated with (1) increased age, B = .09, SE = .01, p = .00, (2) more depressive symptoms, B = .02, SE = .01, p = .00, and (3) higher BMI, B = .07, SE = .01, p = .00.

Aim 3: Racial discrimination x poverty status x sex. In block 1 of the analysis, age, BMI, depression, and alcohol and cigarette status were entered into the regression equation,

together accounting for a significant 25.6% of the variance (p = .00) in HTN. Racial discrimination, poverty status, and sex were all added in block 2, together accounting for an additional, non-significant 0.3% (p = .30) of the variance in HTN. Neither racial discrimination (B = -.02, p = .64), poverty status (B = .21, p = .10), nor sex (B = -.08, p = .54) were significantly related to HTN. In block 3, the interaction terms for racial discrimination x poverty status, racial discrimination x sex, and poverty status x sex were entered into the regression equation, together accounting for an additional, non-significant 0.2% (p = .38) of the variance in HTN. None of the three individual two-way interaction terms were significant. In block 4 of the analysis, the three-way interaction term of racial discrimination x poverty status x sex was entered into the regression equation, accounting for an additional, non-significant 0.1% (p = .29) of the variance in HTN. Thus, the exploratory analysis revealed that there was not a significant racial discrimination x poverty status x sex interaction (B = .14, p = .29) for HTN.

The overall model significantly predicted HTN (χ^2 (12) = 303.72, p = .00, -2 log likelihood = 1617.14). There were three significant effects in this model, all of which were from covariates. HTN was associated with (1) increased age, B = .10, SE = .01, p = .00, (2) more depressive symptoms, B = .02, SE = .01, p = .00, and (3) higher BMI, B = .07, SE = .01, p = .00.

Discussion

The purpose of the present study was to examine whether the relation of racial discrimination to cardiovascular risk factors is moderated by poverty status and sex among African Americans. The cardiovascular factors examined were the continuous variables of resting SBP and DBP, and the dichotomous variable of hypertension status. Results did not support the proposed two-way racial discrimination by poverty status interaction hypothesis: the only significant two-way interaction, for SBP, was contrary to hypothesis, in that African

Americans not living in poverty appear to be more vulnerable to the BP effects of racial discrimination than those living in poverty. None of the three exploratory racial discrimination by sex interaction analyses were significant. One of the three racial discrimination by poverty status by sex three-way interactions was significant, but the pattern of findings was unexpected. Specifically, simple effects analyses indicated there was a significant inverse relationship between racial discrimination and DBP for African American men living below the poverty line, and no significant relationship between the two variables for African American women living below the poverty line, or African American men or women living above the poverty line. The study findings are discussed below.

Aim 1: Racial Discrimination x Poverty Status on BP and HTN

Contrary to hypothesis, there was no evidence to support the expectation that the poverty status group would be more susceptible than the group living above the poverty line to elevated SBP, DBP or HTN as a result of increased exposure to racial discrimination. Theoretically, given that poverty status is widely viewed as a significant chronic stressor (Baum, Garofalo, & Yali, 1999; Williams & Collins, 1995; Winkleby, Jatulis, Frank, & Fortmann, 1992), individuals living in poverty were expected to be more vulnerable to the negative effects of racial discrimination than those not living in poverty. This was not found to be the case in the current study.

In fact, concerning SBP, the opposite finding was obtained: individuals living in poverty appeared to be less vulnerable to the effects of racial discrimination than those not living in poverty. Interestingly, this finding is consistent with several recent studies suggesting that higher SES is not a protective factor for adverse health outcomes for African Americans. For instance, Assari and Lankarani (2016) examined the effects of education and income on all-cause mortality based on race and place. They found that the protective effects of education on all-

cause mortality is moderated by race; as education levels increase, Blacks do not achieve comparable improvements in health as Whites. This is known as the "diminishing returns" hypothesis. Ferraro and Farmer (2005) first used this term and described this phenomenon as a situation in which minority persons do not experience the same returns as whites for higher SES achievement. Link and Phelan (1995) also contributed to the conceptualization of this idea, as they first posed the question of whether there are certain social conditions in which income and education might be stronger or weaker predictors of health care use. More recent studies (e.g., Assari, 2017; Assari et al., 2017) have continued to examine this phenomenon and found that the protective effect of various measures of SES (e.g., education, income, employment, etc.) on various adverse health-related outcomes (e.g., high BMI, insomnia, mortality, etc.) are absent for African Americans. Consistent with these studies, the current findings also do not reveal a protective effect of SES (non-poverty status) for African Americans.

Diminishing returns, however, does not directly explain why African Americans living above the poverty line would be relatively more vulnerable to the negative effects of racial discrimination than African Americans living below the poverty line, nor why there was a (non-significant) inverse relation between racial discrimination and blood pressure for African Americans living below the poverty line. One possible contributing factor for the latter may be that low-income African Americans are more likely to deny or suppress their reporting of racial discrimination. Prior research (e.g., Brondolo, 2003) has noted that exposure to racism is difficult to assess, in part due to the fact that several psychological factors (e.g., personality differences, association of victimization with loss of control, etc.) may lead to participants minimizing (or maximizing) reports of exposure to racism. As such, there may be characteristics associated with low SES status (e.g., John Henryism, Roberts et al., 2008) that make this group less likely to

report experiencing racial discrimination, either because they are intentionally not reporting it or because unconsciously they do not recognize that they are experiencing it. And it is possible these personal characteristics lead simultaneously to lower reporting of racial discrimination and to higher levels of blood pressure.

On the other hand, one possible explanation for why African Americans living above the poverty line may be more vulnerable to the negative effects of racial discrimination than African Americans living below the poverty line is that racial discrimination may be more impactful when you live and work among Whites than when you live and work among other African Americans. African Americans living above the poverty line are more likely to live and work in places that are predominately White, which has been found to be associated with an increased exposure to racial discrimination (Assari & Lankarani, 2018). This is consistent with a recent survey report (National Public Radio, Robert Wood Johnson Foundation, & Harvard T.H. Chan School of Public Health, 2017) which found that compared to their urban counterparts, African Americans who reside in suburban areas are more likely to report being threatened or harassed due to their race and to report unfair stops or mistreatment by the police. As such, it may be the case that these experiences of racial discrimination experienced by higher SES African Americans are more impactful than those experienced by lower SES African Americans, perhaps higher SES Africans had expected their SES status to protect them from such exposure, or because of the constancy of experiencing racial discrimination across settings, and/or because of lower levels of protective support from other African Americans in their higher SES settings. More generally, apart from diminishing returns per se, African Americans above the poverty line may be systematically impacted by structural and institutional racism in ways that negatively

impact cardiovascular outcomes (Assari, 2018). Future research is necessary to replicate the current finding and to examine these and other possible mechanisms of influence.

The way that SES was measured in the present study may have also impacted the findings. Prior literature (Krieger, Williams, & Moss, 1997) suggests that it is important for SES to be measured using multiple indices (e.g., education, occupation, and income) and on multiple levels (e.g., individual, family, and neighborhood). This comprehensive approach to measuring SES is ideal because SES is not equivalent across all groups, and because it allows for a better picture of the participants' available resources (Brondolo, 2003). However, the present study used poverty status, via family income as a function of household size, as the sole measure of SES. Furthermore, as previously mentioned, the present study dichotomized SES (0 = non-poverty, 125% above threshold; 1 = poverty, 125% below threshold) rather than using a continuous measure, thus reducing variability as a predictive factor, and also not allowing the ability to separately examine relations between racial discrimination and blood pressure across multiple categories of SES (e.g., working class, middle class, upper middle-class, upper class). As such, in the present study, the usage of poverty status as a measure of SES and dichotomizing this variable into only two categories may not have allowed for a valid testing of the moderating impact of SES. It should also be noted that the effect size of the obtained interaction was near zero (0.3% of variance explained), reflecting a very minor effect, not even reaching the level of a small effect size (1% of variance explained).

Concerning DBP, a significant racial discrimination by poverty status interaction was not found. Given that SBP and DBP are both indicators of BP, and not surprisingly were highly correlated in the current sample ($r = .68$) it is surprising that the findings were not consistent across the two measures. Although it is not fully clear why there were significant findings for

SBP and not DBP, this finding is consistent with other studies which have found significant findings for only one, rather than both, indicators of BP (e.g., Dolezsar et al., 2014). One possible explanation has to do with limitations in the way BP was measured in the current study. Although the procedures used to measure and analyze resting SBP and DBP were in alignment with a number of prior studies, they are not considered to be in alignment with the gold standard in the field of how blood pressure should be measured. A report (2004) from the Seventh Joint National Committee (JNC 7) outlined standards for how BP should be classified and measured, stating that BP should be assessed using the average of two or more readings for each arm, on two or more office visits. However, the present study assessed BP based on one measurement from each arm, taken in the same day. Thus, the methods used for assessing BP in the present study may not have been as robust as desired, which may help to account for the unexpected and inconsistent findings across BP indicators. The above notwithstanding, it should be noted that the non-significant racial discrimination simple main effects for those not living in poverty for SBP (beta =.44) and DPB (beta=.38) were quite similar.

There also was not a significant racial discrimination by poverty status interaction for HTN. This is especially surprising given the Dolezsar et al. (2014) meta-analysis finding that studies with a greater proportion of individuals whose highest level of education was high school or less (low SES) had significantly stronger effect sizes for the relation between racial discrimination and HTN than studies with a smaller proportion of individuals whose highest level of education was high school less. As noted above, possible explanations for the lack of a significant racial discrimination by SES interaction for HTN are the previously mentioned limitations of the measures of SES and blood pressure used in the present study. Further, as also previously mentioned above, personality characteristics of a subset of individuals in poverty may

leave simultaneously to denial of racial discrimination and high levels of blood pressure. These factors related to SES may help explain the lack of a racial discrimination by poverty status interaction for HTN.

Finally, the current study findings may be due in part to limitations in the measure of racial discrimination used in the present study. Brondolo (2003) suggested that studies that used a measure similar to the one used in the present study may be insensitive to nuanced, within-group differences in exposure. Brondolo suggested use of alternate self-report measures (e.g., The Schedule of Racist Events, the Perceived Racism Scale, etc.) that can provide a more sensitive and comprehensive measurement of exposure to racism across everyday situations due to measuring additional dimensions such as the frequency, intensity, and duration of the exposure, as well as both overt and subtle forms of discrimination.

Aim 2: Racial discrimination x Sex on BP and HTN

Exploratory analyses did not reveal significant racial discrimination x sex effects for SBP DBP, or HTN. Concerning HTN, these findings are consistent with Sim et al. (2012) who did not find a significant racial discrimination by sex interaction for HTN. On the other hand, concerning DBP, the findings differ from the Dolezsar et al. (2014) meta-analysis finding that studies with a greater proportion of males found significantly stronger effect sizes for the relation between racial discrimination and DBP than studies with a smaller proportion of males.

One possible explanation for the lack of findings for racial discrimination x sex for all outcomes relates to the limitations of the measures of racial discrimination and cardiovascular outcomes noted above. Alternately, it may in fact be the case, as the findings appear to indicate, that males and females are equally vulnerable to the effects of racial discrimination, though perhaps for somewhat different reasons. For instance, African American men report more

experiences of racial discrimination across more settings (Kwate & Goodman, 2015; Johnson Foundation, & Harvard T.H. Chan School of Public Health, 2017), and this constant exposure to discrimination may lead them to be more vulnerable to its effects. On the other hand, African American women may experience a unique form of discrimination due to their double minority status (Essed, 1991), which contributes in a unique way to their vulnerability to the effects of racism. Overall, there may not be a difference in the vulnerability of African American men and women to the effects of racial discrimination.

It is also possible that vulnerability to the effects of racism for males versus females may depend partly on other factors. For example, there may be differing effects of age on African American men and women in terms of cardiovascular outcomes. Specifically, Stamler, Stamler, Riedlinger, Algera, & Roberts (1976) found that African American men had higher SBP than African American women up to age 50, but in older age groups African American women had higher SBP than African American men. Similarly, Cutler et al. (2008) found that regardless of race and ethnicity, for ages 18-39, men had a higher prevalence of HTN than women. However, this sex difference disappeared for African Americans (and Mexican Americans) ages 50-69, and after the age of 70 women had higher HTN across all racial groups. This suggests that age may be a moderating factor that influences whether African American men or women are more vulnerable to the cardiovascular effects of racial discrimination. In fact, recent literature (e.g., Beatty Moody et al., 2017) has explored the moderating effect of age in the relation between lifetime discrimination and ambulatory BP among African Americans and Latinos. Beatty Moody et al. found there were significant interactions of age and lifetime discrimination on 24-hour and daytime DBP, with significant relations between discrimination and outcomes emerging for older participants only.

Aim 3: Racial discrimination x Poverty Status x Sex on BP and HTN

Exploratory analysis revealed no significant findings for racial discrimination x poverty x sex with respect to elevated SBP or HTN status, but there was a significant finding for DBP. Prior literature has not directly examined this interaction, but it has suggested that exploration of this three-way interaction might be important. For example, although not statistically examined, one seminal work (Krieger & Sidney, 1996) suggested the possibility of complex, unexpected three-way interactions between SES (characterized as "working class" and "executive, professional, and/or supervisory"), sex and racial discrimination among African Americans. This study found patterns of associations to BP outcomes that differed by SES and racial discrimination in general and for both sexes. Specifically, working-class African Americans that did not report experiencing racial discrimination had higher SBP levels than those who did report experiencing it. For professional African Americans, those that did not report experiencing racial discrimination had lower SBP levels than those who did report it. Working-class African American women that did not report experiencing racial discrimination had higher SBP levels than those who did report it. Working-class African American men who reported experiencing racial discrimination across more settings had higher levels of SBP than those who reported experiencing it across fewer settings. Given the complex findings, Krieger & Sidney (1996) suggested that future analyses should consider how SES and sex affect exposure to and the impact of racial discrimination.

Concerning the current study's findings of a three-way interaction for DBP, the only significant simple main effect was the inverse relationship between racial discrimination and DBP for African American men living below the poverty line. This same inverse relationship, though not achieving significance, was reported earlier for African Americans living below the

poverty line. Here, the inverse relationship is found to be present particularly for a subgroup of those living in poverty—African American men—and achieves statistical significance. As previously mentioned, poverty status is considered to be a chronic stress that negatively impacts health. Thus, it was expected that African American men with a poverty status who experienced higher levels of racial discrimination would have greater vulnerability and thus higher, not lower, levels of DBP.

One possible explanation for this contrary finding is that some African American men living in poverty may be particularly likely to deny experiencing racial discrimination, and that this process of denial may simultaneously lead to negative cardiovascular outcomes. This view is consistent with Brondolo (2003) who stated that there are several psychological factors that may inhibit someone from recognizing and/or reporting experiencing discrimination. Furthermore, Roberts et al. (2008) specifically noted that the inverse relations between discrimination and cardiovascular outcomes among African American men and women found in several studies (Karlsen & Nazroo, 2006; Krieger, 1990; Krieger & Sidney, 1996) along with the generally high frequency of reports of discrimination suggest that African Americans, especially men, may habitually and unconsciously deny racism as a means of coping with it (i.e., John Henryism). Others have suggested that individuals from lower social classes may be more likely to use denial as a coping mechanism when confronted with racial discrimination, and that denial may be linked to negative health outcomes (Karlsen & Nazroo, 2002). An additionally, it may be the case that some African American men living in poverty may deny experiencing racism because as members of two oppressed identities, they may feel the need to deny in order to cope and appear stronger. This is loosely related to a phenomenon known as “cool pose” in which African American men often adopt a persona of being “cool” because coolness as a strength may be

linked to pride, self-respect, and masculinity (Majors & Billson, 1992). As such, denying racism can be a "cool pose" in that some low-SES African American men—who may already feel a lack of strength or respect for being low SES— believe they can appear stronger because they do not experience this. Altogether, future research is necessary to replicate the findings of the current research and to examine possible mechanisms of influence. It should also be noted, though, that the effect size of the obtained interaction was near zero (0.3% of variance explained), reflecting a very minor effect, not even reaching the level of a small effect size (1% of variance explained).

In relation to the null findings for SBP and HTN, as noted previously, several methodological issues existed in relation to how the present study measured racial discrimination, poverty, BP, and HTN. There are also considerations related to the complex nature of sex differences in experiences of racial discrimination and its effects. These various factors may have limited the ability of these variables to fully and comprehensively capture the participants' experiences may have contributed to the null findings for racial discrimination x poverty x sex for SBP and HTN (it should be noted, though, that for SBP, that simple effect analysis indicated that African American men living in poverty showed an inverse relation between racial discrimination and SBP, beta=-1.05, though this was not significant).

More generally, it appears that the relations among poverty, sex and racial discrimination appear quite complex and nuanced, likely depending on numerous factors, including SES, type of discrimination experienced, and setting of the exposure (Borrell, et al., 2006; Kwate & Goodman, 2015; Roberts et al., 2008). Additionally, research suggests that African American men and women may have different physiological responses to the same exposure to a discriminatory experience (Morris-Prather et al., 1996). Thus, capturing the interactive influences of variables may be limited if there are issues in the measurements. The results of the

interaction between poverty, sex, and cardiovascular outcomes depend upon those variables being measured as accurately and comprehensively as possible, which is an important priority for future research. Further, as noted earlier, additional variables such as age and personality factors may need to be examined as potentially important moderators, as past studies (e.g., Beatty Moody et al., 2017) have found evidence that these factors moderate the associations between discrimination and BP.

Study Limitations and Strengths

In addition to the methodological limitations regarding study measures discussed above, the present study is limited by a few concerns. First, secondary data analyses were conducted, which is a limitation because the data were already collected, and additional measures and variables could not be included. Further, the analyses were cross-sectional. Cardiovascular risk factors usually develop over time, and the literature suggests that longitudinal analyses are the ideal way assess the link between racial discrimination and BP/HTN, as well as how other variables may moderate the relationship. Essentially, examining how these variables are related to one another over time may provide more insight and understanding than a cross-sectional design. Furthermore, based on our existing limits in understanding from prior theory and research, most of the analyses were designed as exploratory (two of the three aims). Proposing study hypotheses is generally more preferred than exploratory analyses because the former is in alignment with the scientific process of conducting analyses to confirm or deny hypotheses. Finally, although compared to many studies in this area, HANDLS has a more diverse sample, it was still limited to self-identified African American individuals in Baltimore City. As such, the generalizability of the findings to other racial or ethnic populations and more suburban or rural samples is limited.

Despite these limitations, the present study had several notable strengths. First, the HANDLS study was intentionally designed to sample demographically diverse groups of men and women. Second, the analyses adjusted for several variables that are relevant to BP and HTN, which helped increase the likelihood that the findings were due to the predictor and moderator variables of interest. Finally, this study extended the current literature, going beyond simply controlling demographic variables to directly examine the interactive relations of racial discrimination and the sociodemographic variables SES and gender in predicting BP and HTN among African Americans. This can help contribute to greater understanding of the nuances and complexities of within-group differences among African Americans in cardiovascular health disparities.

Conclusions, Implications, and Future Directions

Compared to Whites and other racial/ethnic groups, African Americans have greater rates of BP and HTN. These racial health disparities in BP and HTN are problematic because HTN is a central risk factor for CVD. Although there are several well-established traditional risk factors associated with CVD, these factors do not fully account for the CVD burden among African Americans. As such, researchers have examined other factors, mainly social determinants, that also contribute to this CVD burden and racial health disparities. One such factor is discrimination, as it has been conceptualized as a chronic stressor and linked to adverse health outcomes, including CVD. Since African Americans have high prevalence rates of both CVD and racial discrimination, research has called for the examination of pathways that may link discrimination to HTN and CVD among this population. Two factors that the literature suggests may moderate the relation between racial discrimination and cardiovascular outcomes are SES and sex. Although the findings from this study were complex and mixed in their agreement with

the literature, they indicate that in the current sample, poverty status and racial discrimination were interactively related to BP but not HTN, the interaction of sex and racial discrimination were not related to any of the outcomes, and the interaction of poverty status, sex and racial discrimination was related to BP but not HTN.

The current findings were not expected and need to be replicated in future research. More generally, in future research in this area, better assessment is needed for of each of the primary study variables. There were several issues related to the measurement of racial discrimination. As previously mentioned, the literature highlights concern and difficulties with how this variable is conceptualized and measured. Specifically, past literature (e.g., Brondolo, 2003) stated that racism itself is already a difficult concept to assess. As such, Brondolo suggested that measures of racism and racial discrimination need to be sensitive, comprehensive, and nuanced enough to capture more details (e.g., within group differences, type, frequency of exposure, etc.) about these experiences. Findings from other studies have supported the idea of more variation and nuance in measures of discrimination, specifically as it relates to type and timing. For example, there is evidence that institutional discrimination has a stronger association to HTN than interpersonal discrimination (Brondolo et al., 2011), and that assessment of racial discrimination in the past year is more sensitive than lifetime measures (Paradies, 2006). As such, future research should consider utilizing measures that simultaneously assess different forms of racial discrimination (e.g., interpersonal and institutional). Future research should also ideally assess racial discrimination that occurred over different time intervals (e.g., daily during the past week, past year, and lifetime) as well as examine generational cohort effects.

It is important as well to consider use of additional approaches to measure racial discrimination in a more nuanced and comprehensive manner. One such way is the use of

qualitative methods such as one-on-one interviews or focus groups. Using this method alone or using mixed methods by combining this method with quantitative measures will allow for more rich details from participants regarding their experiences of racial discrimination. Another way to measure discrimination more comprehensively is to broaden the focus to also include other types of discrimination (e.g., gender, socioeconomic, etc.) in addition to racial. This would provide more insight into experiences of discrimination, especially as it relates to intersectionality because those who experience racial discrimination often do not experience it in a vacuum outside of their other various identities.

Future research can also benefit from more comprehensive measures of SES. As previously mentioned, prior research (e.g., Brondolo, 2003; Krieger, Williams, & Moss, 1997) stated that due to the complex nature of SES and its variation among individuals, this variable should be measured using multiple indices on multiple levels. The present study may have been limited in its ability to explore how SES may moderate the relation of racial discrimination to BP and HTN because it used poverty status as a proxy for SES and dichotomized it. However, it is important to note that another limitation is specific to the way HANDLS used poverty status as a way to measure SES. Although it is not the ideal way to measure this construct, it was purposely used because participants either do not recall or do not wish to share complex information related to SES. Nevertheless, future research that includes a more comprehensive measure of SES would likely contribute to greater understanding of its impact.

Lastly, in regard to measurement, future research can strive for a more comprehensive assessment of BP that can capture and reflect more nuances in BP levels. Specifically, as stated above, one improvement would be to use the gold standard of BP measurement and expand the number of visits in which BP is taken as well as taking measurements from both arms. Future

research should also consider other ways to measure BP. For example, past literature indicates that ABP may be a more sensitive BP measure as opposed to SBP and DBP. Specifically, Brondolo et al. (2011) suggested that ABP is a more consistent measure of BP because it captures BP reactivity to daily events. Future research should consider ABP as a better way to measure cardiovascular reactivity.

Future research should also examine longitudinal relations among study variables, include other racial/ethnic groups, and consider examining other variables that may moderate the relationship between racial discrimination and BP and HTN. For example, as previously mentioned, the literature (e.g., Beatty Moody et al., 2017) suggests that age moderates the relationship between discrimination and BP. Specifically, this study found that this relationship was significant for older participants. Further, given the complexities surrounding the concept of racial discrimination, it is plausible that in addition to age, there may be several other factors or determinants that may moderate its association to BP and HTN. Future research seeking to explore the pathways linking racial discrimination to BP and HTN should consider the moderating effects of additional variables.

Although the relations among racial discrimination, moderating variables, and cardiovascular outcomes are complex and nuanced, further study remains a high priority, given the continued presence of racism in our society and the negative effects on the health and well-being of persons of color.

Table 1
Sample characteristics

Variable	
Age (years)	47.60 (9.26)
Cigarette status (% current users)	47.6
Alcohol use (% current users)	20.7
BMI	29.97 (7.71)
CES-D	14.48 (10.76)
Antihypertensive medication use (% prescribed)	29.7
Mean racial discrimination	7.65 (1.92)
Poverty status (%>125% 2004 federal poverty level)	54
Sex (% female)	56.3
Mean SBP	122.021 (17.24)
Mean DBP	73.42 (10.81)
Hypertension diagnosis (% yes)	46.4

Table 2.
Matrix of correlation coefficients (Pearon's r) among all variables

	1.	2.	3.	4.	5.	6.	7.	8.	9.	10.	11.	12.
1. Age	-											
2. Cigarette smoking status	.014	-										
3. Alcohol use	.080**	.296**	-									
4. BMI	.035	-.216	-.043	-								
5. CES-D	-.082**	.081**	-.029	-.004	-							
6. Prescribed antihypertensive medication	.362**	-.044	.098**	.246**	-.018	-						
7. Racial discrimination	.055*	.060*	.026	-.055*	.101**	.008	-					
8. Poverty status	.095**	.106**	-.171**	-.104**	.166**	-.050	.031	-				
9. Sex (men)	-.030	.185**	.134**	-.283**	-.071**	-.119**	.219**	-.032	-			
10. SBP	.313**	-.018	-.003	.205**	.014	.248**	.015	.019	-.021	-		
11. DBP	.071**	-.032	-.003	.110**	.041	.147**	.031	.036	.087**	.681**	-	
12. Hypertension status	.373**	-.015	.097**	.233**	.059*	.697**	.002	-.012	-.093**	.515**	.373**	-

Note. *$p < .05$; **$p < .01$; ***$p < .001$

Table 3
Racial Discrimination x Poverty Status Predicting SBP: Final Model

Variables Included	B	SE	β	*t*	*p*
Age	.52	.05	.28	10.42	.00
Depressive symptoms	.04	.04	.03	1.00	.32
BMI	.41	.06	.18	6.85	.00
Anti-HTN medication	4.06	1.04	.11	3.93	.00
Alcohol status	-1.25	1.08	-.03	-1.16	.25
Cigarette status	.57	.90	.02	.63	.53
Racial Discrimination (RD)	.47	.31	.05	1.52	.13
Poverty status	9.07	3.53	.26	2.57	.01
RD x Poverty status	-.92	.45	-.22	-2.06	.04

Table 4
Racial Discrimination x Sex Predicting SBP: Final Model

Variables Included	B	SE	β	*t*	*p*
Age	.52	.05	.28	10.32	.00
Depressive symptoms	.06	.04	.04	1.53	.13
BMI	.42	.06	.19	6.94	.00
Anti-HTN medication	4.29	1.04	.11	4.13	.00
Alcohol status	-1.41	1.07	-.03	-1.32	.19
Cigarette status	.73	.90	.02	.81	.42
Racial Discrimination (RD)	.20	.32	.02	.64	.52
Sex (men)	6.32	3.63	.18	1.74	.08
RD x Sex	-.56	.46	-.14	-1.24	.22

Table 5
Racial Discrimination x Poverty Status x Sex Predicting SBP: Final Model

Variables Included	B	SE	β	*t*	*p*
Age	.52	.05	.28	10.47	.00
Depressive symptoms	.05	.04	.03	1.20	.23
BMI	.43	.06	.20	7.13	.00
Anti-HTN medication	4.29	1.04	.11	4.13	.00
Alcohol status	-1.29	1.08	-.03	-1.20	.23
Cigarette status	.41	.91	.01	.45	.66
Racial Discrimination (RD)	.43	.45	.05	.96	.34
Poverty status	6.72	4.76	.20	1.41	.16
Sex (men)	4.36	4.93	.13	.89	.38
RD x Poverty status	-.52	.63	-.12	-.82	.41
RD x Sex	-.23	.63	-.06	-.37	.72
Poverty status x Sex	4.76	7.26	.11	.66	.51
RD x Poverty Status x Sex	-.75	.91	-.15	-.82	.41

Table 6
Racial Discrimination x Poverty Status Predicting DBP: Final Model

Variables Included	B	SE	β	*t*	*p*
Age	.04	.03	.03	1.10	.27
Depressive symptoms	.04	.03	.04	1.34	.18
BMI	.11	.04	.08	2.82	.01
Anti-HTN medication	2.63	.69	.11	3.79	.00
Alcohol status	-.75	.72	-.03	-1.05	.30
Cigarette status	-.85	.61	-.04	-1.41	.16
Racial Discrimination (RD)	.39	.21	.07	1.91	.06
Poverty status	4.75	2.36	.22	2.01	.04
RD x Poverty status	-.48	.30	-.18	-1.60	.11

Table 7
Racial Discrimination x Sex Predicting DBP: Final Model

Variables Included	B	SE	β	*t*	*p*
Age	.04	.03	.03	1.09	.28
Depressive symptoms	.06	.03	.06	2.17	.03
BMI	.15	.04	.11	3.76	.00
Anti-HTN medication	2.86	.69	.12	4.14	.00
Alcohol status	-.95	.71	-.04	-1.33	.18
Cigarette status	-.98	.60	-.05	-1.63	.10
Racial Discrimination (RD)	.08	.21	.02	.39	.70
Sex (men)	4.41	2.41	.20	1.83	.07
RD x Sex	-.17	.30	-.07	-.57	.57

Table 8
Racial Discrimination x Poverty Status x Sex Predicting DBP: Final Model

Variables Included	B	SE	β	*t*	*p*
Age	.04	.03	.04	1.24	.22
Depressive symptoms	.05	.03	.05	1.90	.06
BMI	.16	.04	.11	3.90	.00
Anti-HTN medication	2.87	.69	.12	4.17	.00
Alcohol status	-.84	.71	-.03	-1.17	.24
Cigarette status	-1.11	.60	-.05	-1.85	.07
Racial Discrimination (RD)	-.07	.30	-.01	-.22	.83
Poverty status	.56	3.16	.03	.18	.86
Sex (men)	.90	3.27	.04	.27	.78
RD x Poverty status	.24	.42	.09	.57	.57
RD x Sex	.42	.42	.17	1.02	.31
Poverty status x Sex	7.50	4.81	.28	1.56	.12
RD x Poverty Status x Sex	-1.24	.60	-.39	-2.06	.04

Table 9
Racial Discrimination x Poverty Status Predicting HTN: Final Model

Variables Included	B	SE	Wald	*df*	*p*
Age	.10	.01	169.84	1	.00
Depressive symptoms	.02	.01	10.54	1	.00
BMI	.07	.01	67.41	1	.00
Alcohol status	.28	.15	3.35	1	.07
Cigarette status	.02	.13	.03	1	.87
Racial Discrimination (RD)	-.03	.04	.39	1	.53
Poverty status	.10	.50	.04	1	.84
RD x Poverty status	.02	.06	.06	1	.81

Table 10
Racial Discrimination x Sex Predicting HTN: Final Model

Variables Included	B	SE	Wald	*df*	*p*
Age	.09	.01	167.51	1	.00
Depressive symptoms	.02	.01	11.89	1	.00
BMI	.07	.01	60.56	1	.00
Alcohol status	.25	.15	2.73	1	.10
Cigarette status	.05	.13	.18	1	.67
Racial Discrimination (RD)	-.05	.05	1.17	1	.28
Sex (men)	-.63	.51	1.56	1	.21
RD x Sex	.07	.06	1.21	1	.27

Table 11
Racial Discrimination x Poverty Status x Sex Predicting HTN: Final Model

Variables Included	B	SE	Wald	*df*	*p*
Age	.10	.01	168.79	1	.00
Depressive symptoms	.02	.01	10.21	1	.00
BMI	.07	.01	62.54	1	.00
Alcohol status	.27	.15	3.18	1	.08
Cigarette status	.02	.13	.01	1	.91
Racial Discrimination (RD)	-.02	.06	.06	1	.81
Poverty status	.57	.68	.72	1	.40
Sex (men)	-.27	.69	.15	1	.70
RD x Poverty status	-.07	.09	.60	1	.44
RD x Sex	.01	.09	.00	1	.96
Poverty status x Sex	-.72	1.02	.49	1	.49
RD x Poverty Status x Sex	.14	.13	1.12	1	.29

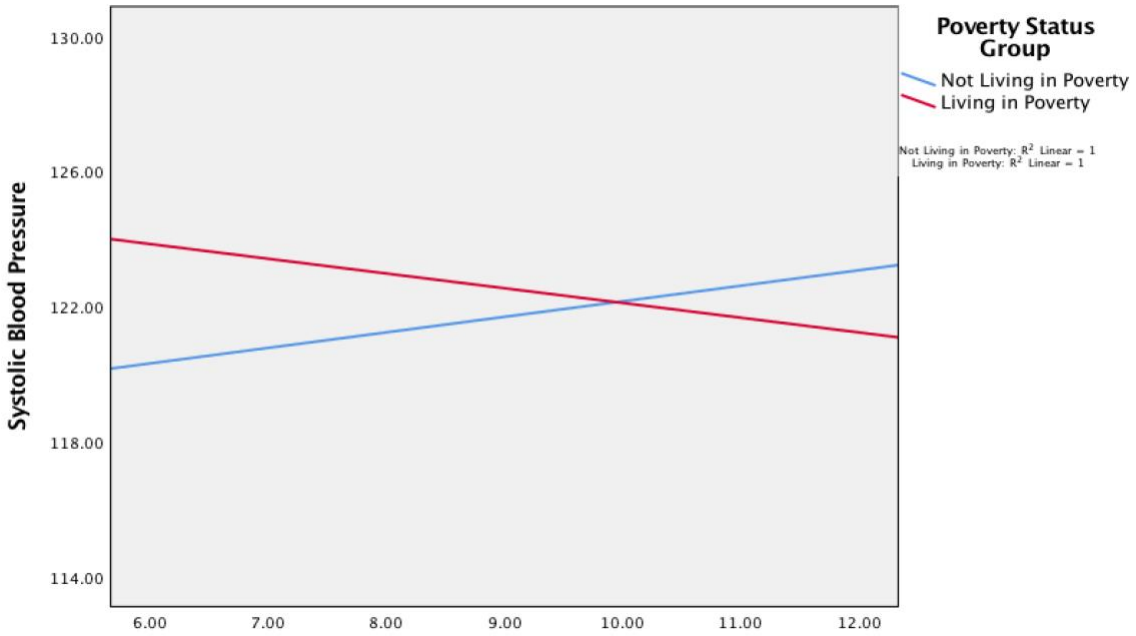

Figure 1. Simple effect analysis of racial discrimination on SBP for each poverty status group.

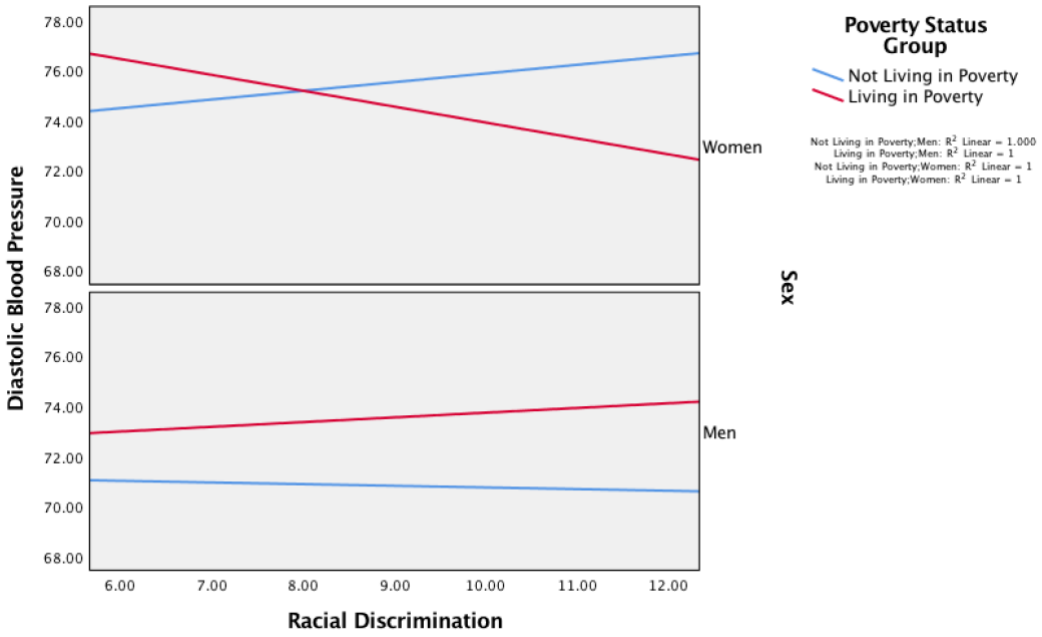

Figure 2. Simple effect analysis of racial discrimination on SBP for each poverty status and sex

References

Adams, J. P., & Dressler, W. W. (1988). Perceptions of Injustice in a Black Community: Dimensions and Variation. *Human Relations*, *41*(10), 753-767.

American Heart Association. (2017a, December 12). Know Your Risk Factors for High Blood Pressure. Retrieved from http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/UnderstandSymptomsRisks/Know-Your-Risk-Factors-for-High-Blood-Pressure_UCM_002052_Article.jsp#.WjQTNiPMyCQ

American Heart Association. (2017b, December 12). Understand Your Risks to Prevent a Heart Attack. Retrieved from http://www.heart.org/HEARTORG/Conditions/HeartAttack/UnderstandYourRiskstoPreventaHeartAttack/Understand-Your-Risks-to-Prevent-a-Heart-Attack_UCM_002040_Article.jsp#.WjQUDiPMyCQ

American Heart Association. (2017c, December 5). Understanding Blood Pressure Readings. Retrieved from http://www.heart.org/HEARTORG/Conditions/HighBloodPressure/KnowYourNumbers/Understanding-Blood-Pressure-Readings_UCM_301764_Article.jsp#.WjgA8CPMyb8

American Heart Association. (2017d, June 7). What is Cardiovascular Disease?. Retrieved from http://www.heart.org/HEARTORG/Support/What-is-Cardiovascular-Disease_UCM_301852_Article.jsp#.WjPwryPMyCQ

Ames, S. C., Jones, G. N., Howe, J. T., & Brantley, P. J. (2001). A prospective study of the impact of stress on quality of life: An investigation of low-income individuals with hypertension. *Annals of Behavioral Medicine*, *23*(2), 112-119. doi:10.1207/S15324796ABM2302_5

Andersen, B. L., Kiecolt-Glaser, J. K., & Glaser, R. (1994). A biobehavioral model of cancer

stress and disease course. *American Psychologist*, *49*(5), 389-404. doi:10.1037/0003-066X.49.5.389

Arboix, A. (2015). Cardiovascular risk factors for acute stroke: Risk profiles in the different subtypes of ischemic stroke. *World Journal of Clinical Cases*, *3*(5), 418-429. doi:10.12998/wjcc.v3.i5.418.

Assari, S. (2018). Health Disparities due to Diminished Return among Black Americans: Public Policy Solutions. *Social Issues & Policy Review*, *12*(1), 112–145. doi: 10.1111/sipr.12042

Assari, S. (2018). Life Expectancy Gain Due to Employment Status Depends on Race, Gender, Education, and Their Intersections. *Journal of Racial and Ethnic Health Disparities*, *5*(2), 375-386.

Assari, S., & Lankarani, M. M. (2016). Race and Urbanity Alter the Protective Effect of Education but not Income on Mortality. *Frontiers In Public Health*, *4*, 100. Doi: 10.3389/fpubh.2016.00100

Assari, S., & Lankarani, M. M. (2018). Workplace Racial Composition Explains High Perceived Discrimination of High Socioeconomic Status African American Men. *Brain Sciences (2076-3425)*, *8*(8), 139. Doi: 10.3390/brainsci8080139

Assari, S., Nikahd, A., Malekahmadi, M. R., Lankarani, M. M., & Zamanian, H. (2017). Race by Gender Group Differences in the Protective Effects of Socioeconomic Factors Against Sustained Health Problems Across Five Domains. *Journal of Racial and Ethnic Health Disparities*, *4*(5), 884-894.

Avison, W., & Turner, R. (1988). Stressful Life Events and Depressive Symptoms: Disaggregating the Effects of Acute Stressors and Chronic Strains. *Journal of Health and Social Behavior, 29*(3), 253-264. Retrieved from http://www.jstor.org/stable/2137036

Ayotte, B. J., Hausmann, L. R., Whittle, J., & Kressin, N. R. (2012). Clinical Investigation: The relationship between perceived discrimination and coronary artery obstruction. *American*

Heart Journal, *163*677-683. doi:10.1016/j.ahj.2012.01.006

Barksdale, D. J., Farrug, E. R., & Harkness, K. (2009). Racial discrimination and blood pressure: Perceptions, emotions, and behaviors of Black American adults. *Issues in Mental Health Nursing, 30,* 104–111. doi:10.1080/01612840802597879

Barnes, L. L., De Leon, C. M., Lewis, T. T., Bienias, J. L., Wilson, R. S., & Evans, D. A. (2008). Perceived discrimination and mortality in a population-based study of older adults. *American Journal of Public Health*, *98*(7), 1241-1247. doi:10.2105/AJPH.2007.114397

Baum, A., Garofalo, J. P., & Yali, A. M. (1999). Socioeconomic status and chronic stress: Does stress account for SES effects on health? *Annals of The New York Academy Of Sciences*, *896*(1), 131. doi:10.1111/j.1749-6632.1999.tb08111.x

Beatty Moody, D. L., Waldstein, S. R., Tobin, J. N., Cassells, A., Schwartz, J. C., & Brondolo, E. (2016). Lifetime racial/ethnic discrimination and ambulatory blood pressure: The moderating effect of age. *Health Psychology*, *35*(4), 333-342. doi:10.1037/hea0000270

Benjamin, E. J., Blaha, M. J., Chiuve, S. E., Cushman, M., Das, S. R., … Muntner, P. (2017). Heart disease and stroke statistics—2017 update: A report from the American Heart Association. *Circulation*, *135*(10), e146–e603. http://doi.org/10.1161/CIR.0000000000000485

Bennett, G. G., Wolin, K. Y., Robinson, E. L., Fowler, S., & Edwards, C. L. (2005). Perceived racial/ethnic harassment and tobacco use among African American young adults. *American Journal of Public Health*, *95*(2), 238-240.

Berman, G., & Paradies, Y. (2010). Racism, disadvantage and multiculturalism: Towards effective anti-racist praxis. *Ethnic & Racial Studies*, *33*(2), 214-232. doi:10.1080/01419870802302272

Borrell, L. N., Kiefe, C. I., Williams, D. R., Diez-Roux, A. V., & Gordon-Larsen, P. (2006). Self-reported health, perceived racial discrimination, and skin color in African Americans in the CARDIA study. *Social Science & Medicine*, *63*1415-1427.

doi:10.1016/j.socscimed.2006.04.008

Bowen-Reid, T. L., & Harrell, J. P. (2002). Racist experiences and health outcomes: An examination of spirituality as a buffer. *Journal of Black Psychology*, *28*(1), 18-36. doi:10.1177/0095798402028001002

Braveman, P., Egerter, S., & Williams, D. R. (2011). The social determinants of health: Coming of age. *Annual Review Of Public Health*, *32*381-398. doi:10.1146/annurev-publhealth-031210-101218

Broman, C. L. (1996). The health consequences of racial discrimination: A study of African Americans. *Ethnicity & Disease*, *6*(1-2), 148-153.

Broman, C. L., Mavaddat, R., & Shu-yao, H. (2000). The Experience and Consequences of Perceived Racial Discrimination: A Study of African Americans. *Journal of Black Psychology*, (2). 165.

Brondolo, E., Libby, D. J., Denton, E. G., Thompson, S., Beatty, D. L., Schwartz, J., Sweeney, M., Tobin, J. N., Cassells, A., Pickering, T. G., & Gerin, W. (2008). Racism and ambulatory blood pressure in a community sample.

Brondolo, E., Love, E. E., Pencille, M., Schoenthaler, A., & Ogedegbe, G. (2011a). Racism and hypertension: A review of the empirical evidence and implications for clinical practice. *American Journal of Hypertension*, *24*(5), 518-529. doi:10.1038/ajh.2011.9

Brondolo, E., Rieppi, R., Kelly, K. P., & Gerin, W. (2003). Perceived racism and blood pressure: A review of the literature and conceptual and methodological critique. *Annals of Behavioral Medicine*, *25*(1), 55-65. doi:10.1207/S15324796ABM2501_08

Brondolo, E., ver Halen, N. B., Libby, D., & Pencille, M. (2011b). Racism as a psychosocial stressor. In R. J. Contrada, A. Baum, R. J. Contrada, A. Baum (Eds.), *The handbook of stress science: Biology, psychology, and health* (pp. 167-184). New York, NY, US: Springer Publishing Co.

Brown, C., Matthews, K., Bromberger, J., & Chang, Y. (2006). The relation between perceived unfair treatment and blood pressure in a racially/ethnically diverse sample of women. *American Journal of Epidemiology*, *164*(3), 257-262.

Burchfield, S. R. (1979). The stress response: A new perspective. *Psychosomatic Medicine, 41*(8), 661-672. http://dx.doi.org/10.1097/00006842-197912000-00008

Burns, D. M. (2003). Epidemiology of smoking-induced cardiovascular disease. *Progress In Cardiovascular Diseases*, *46*(Issues on Smoking and Cardiovascular Disease, Part III), 11-29. doi:10.1016/S0033-0620(03)00079-3

Calvin, R., Winters, K., Wyatt, S. B., Williams, D. R., Henderson, F. C., & Walker, E. R. (2003). Symposium: Racism and cardiovascular disease in African Americans. *The American Journal of The Medical Sciences*, *325*315-331. doi:10.1097/00000441-200306000-00003

Chae, D. H., Lincoln, K. D., Adler, N. E., & Syme, S. L. (2010). Do experiences of racial discrimination predict cardiovascular disease among African American men? The moderating role of internalized negative racial group attitudes. *Social Science & Medicine*, *71*1182-1188. doi:10.1016/j.socscimed.2010.05.045

Chae, D. H., Nuru-Jeter, A. M., Lincoln, K. D., & Jacob Arriola, K. R. (2012). Racial Discrimination, Mood Disorders, and Cardiovascular Disease Among Black Americans. *Annals Of Epidemiology*, *22*104-111. doi:10.1016/j.annepidem.2011.10.009

Chapman, K. S., & Hariharan, G. (1996). Do Poor People Have a Stronger Relationship between Income and Mortality Than the Rich? Implications of Panel Data for Health-Health Analysis. *Journal of Risk And Uncertainty*, *12*(1), 51-63.

Chou, T., Asnaani, A., & Hofmann, S. G. (2012). Perception of racial discrimination and psychopathology across three U.S. ethnic minority groups. *Cultural Diversity and Ethnic Minority Psychology*, *18*(1), 74-81. doi:10.1037/a0025432

Clark, R. (2000). Perceptions of interethnic group racism predict increased vascular reactivity to

a laboratory challenge in college women. *Annals of Behavioral Medicine*, *22*(3), 214. doi:10.1007/BF02895116

Clark, R. (2003). Self-reported racism and social support predict blood pressure reactivity in Blacks. *Annals of Behavioral Medicine*, *25*(2), 127-136. doi:10.1207/S15324796ABM2502_09

Clark, R. (2006). Interactive but not direct effects of perceived racism and trait anger predict resting systolic and diastolic blood pressure in black adolescents. *Health Psychology*, *25*(5), 580-585. doi:10.1037/0278-6133.25.5.580

Clark, R., & Adams, J. H. (2004). Moderating effects of perceived racism on John Henryism and blood pressure reactivity in Black female college students. *Annals of Behavioral Medicine, 28,* 126–131. doi:10.1207/ s15324796abm2802_8

Clark, R., Anderson, N. B., Clark, V. R., & Williams, D. R. (1999). Racism as a stressor for African Americans: A biopsychosocial model. *American Psychologist*, *54*(10), 805-816. doi:10.1037/0003-066X.54.10.805

Clark, R., & Gochett, P. (2006). Interactive Effects of Perceived Racism and Coping Responses Predict a School-Based Assessment of Blood Pressure in Black Youth. *Annals Of Behavioral Medicine*, *32*(1), 1. doi:10.1207/s15324796abm3201_1

Clark, V. R., & Harrell, J. P. (1992). The relationship among Type A behavior, styles used in coping with racism, and blood pressure. In A. H. Burlew, W. C. Banks, H. P. McAdoo, D. y. Azibo, A. H. Burlew, W. C. Banks, ... D. y. Azibo (Eds.) , *African American psychology: Theory, research, and practice* (pp. 359-369). Thousand Oaks, CA, US: Sage Publications, Inc.

Cobb, F. R., Kraus, W. E., Root, M., & Allen, J. D. (2003). Assessing risk for coronary heart disease: Beyond Framingham. *American Heart Journal*, *146*(4), 572-580.

Cohen S., Kessler, R. C., & Gordon, L. U. (1997). *Measuring stress: A guide for health and*

social scientists. New York: Oxford University Press.

Cozier, Y., Palmer, J. R., Horton, N. J., Fredman, L., Wise, L. A., & Rosenberg, L. (2006). Racial discrimination and the incidence of hypertension in US Black women. *Annals of Epidemiology, 16,* 681–687. doi:10.1016/j.annepidem.2005.11.008

Cunningham, T. J., Seeman, T. E., Kawachi, I., Gortmaker, S. L., Jacobs, D. R., Kiefe, C. I., & Berkman, L. F. (2012). Racial/ethnic and gender differences in the association between self-reported experiences of racial/ethnic discrimination and inflammation in the Cardia cohort of 4 US communities. *Social Science & Medicine*, *75*(5), 922-931. doi:10.1016/j.socscimed.2012.04.027

Cushman, M., Cantrell, R. A., McClure, L. A., Howard, G., Prineas, R. J., Moy, C. S., & ... Howard, V. J. (2008). Estimated 10-year stroke risk by region and race in the United States: geographic and racial differences in stroke risk. *Annals of Neurology*, *64*(5), 507-513. doi:10.1002/ana.21493

Cutler, J. A., Sorlie, P. D., Wolz, M., Thom, T., Fields, L. E., & Roccella, E. J. (2008). Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988-1994 and 1999-2004. *Hypertension (Dallas, Tex.: 1979)*, *52*(5), 818–827.

Davis, A. M., Vinci, L. M., Okwuosa, T. M., Chase, A. R., & Huang, E. S. (2007). Cardiovascular health disparities. *Medical Care Research & Review*, *64*(5), 29s.

DeNavas-Walt, C., & Proctor, B. D. (2014). Income and poverty in the United States: 2013 current population reports. *Washington, DC: US Department of Commerce, US Census Bureau*.

Devins, G. M., Orme, C. M., Costello, C. G., Binik, Y. M., Frizzell, B., Stam, H. J., & Pullin, W. M. (1988). Measuring depressive symptoms in illness populations: Psychometric properties of the Center for Epidemiologic Studies Depression (CES-D) Scale. *Psychology & Health*, *2*(2), 139. doi:10.1080/08870448808400349

Din-Dzietham, R., Nembhard, W. N., Collins, R., & Davis, S. K. (2004). Perceived stress following race-based discrimination at work is associated with hypertension in African-Americans. The Metro Atlanta Heart Disease Study, 1999 –2001. *Social Science & Medicine, 58,* 449 – 461. doi:10.1016/S0277-9536(03)00211-9

Dolezsar, C. M., McGrath, J. J., Herzig, A. M., & Miller, S. B. (2014). Perceived racial discrimination and hypertension: A comprehensive systematic review. *Health Psychology*, *33*(1), 20-34. doi:10.1037/a0033718

Doshi, R. P., Aseltine, R. J., Sabina, A. B., & Graham, G. N. (2017). Racial and ethnic disparities in preventable hospitalizations for chronic disease: Prevalence and risk factors. *Journal of Racial and Ethnic Health Disparities*, *4*(6), 1100-1106. doi:10.1007/s40615-016-0315-z

Dressler, W. W. (1996). Social identity and arterial blood pressure in the African-American community. *Ethnicity & Disease*, *6*(1-2), 176-189.

Duncan, M. J., James, L., & Griffiths, L. (2011). The relationship between resting blood pressure, body mass index and lean body mass index in British children. *Annals of Human Biology*, *38*(3), 324-329. doi:10.3109/03014460.2010.546811

Essed, P. (1991). *Understanding everyday racism: An interdisciplinary theory*. Thousand Oaks, CA, US: Sage Publications, Inc.

Evans, M. K., Lepkowski, J. M., Powe, N. R., LaVeist, T., Kuczmarski, M. F., Zonderman, A. B. (2010). Healthy Aging in Neighborhoods of Diversity across the Life Span (HANDLS): Overcoming barriers to implementing a longitudinal, epidemiologic, urban study of health, race, and socioeconomic status. *Ethnicity and Disease, 20*(3) 267-275.

Everett, B., & Zajacova, A. (2015). Gender differences in hypertension and hypertension awareness among young adults. *Biodemography & Social Biology*, *61*(1), 1-17. doi:10.1080/19485565.2014.929488

Farmer, M. M., & Ferraro, K. F. (2005). Are racial disparities in health conditional on socioeconomic status? *Social Science & Medicine*, *60*(1), 191-204. doi:10.1016/j.socscimed.2004.04.026

Feldman, J. J., Makuc, D. M., Kleinman, J. C., & Cornoni-Huntley, J. (1989). National trends in educational differentials in mortality. *American Journal of Epidemiology*, *129*(5), 919-933.

Finkelstein, E. A., Khavjou, O. A., Mobley, L. R., Haney, D. M., & Will, J. C. (2004). Racial/ethnic disparities in coronary heart disease risk factors among WISEWOMAN Enrollees. *Journal of Women's Health (15409996)*, *13*(5), 503-518.

Forman, T. A., Williams, D. R., & Jackson, J. S. (1997). Race, place, and discrimination. *Perspectives on Social Problems*, *9*, 231-261.

Framingham Heart Study: A Project of the National Heart, Lung, and Blood Institute and Boston University. (2017). History of the Framingham Heart Study. Retrieved from https://www.framinghamheartstudy.org/about-fhs/history.php

Fuller-Rowell, T. E., Doan, S. N., & Eccles, J. S. (2012). Differential effects of perceived discrimination on the diurnal cortisol rhythm of African Americans and Whites. *Psychoneuroendocrinology*, *37*107-118. doi:10.1016/j.psyneuen.2011.05.011

Geronimus, A. T. (1992). The weathering hypothesis and the health of African-American women and infants: Evidence and speculations. *Ethnicity & Disease*, *2*(3), 207-221.

Geronimus, A. T. (1996). General paper: Black/white differences in the relationship of maternal age to birthweight: A population-based test of the weathering hypothesis. *Social Science & Medicine*, *42*, 589-597. doi:10.1016/0277-9536(95)00159-X

Guyll, M., Matthews, K. A., Bromberger, J. T. (2001). Discrimination and unfair treatment: Relationship to cardiovascular reactivity among African American and European American women. *Health Psychology. 20:* 315–325.

Hare, D. L., Toukhsati, S. R., Johansson, P., & Jaarsma, T. (2014). Depression and

cardiovascular disease: A clinical review. *European Heart Journal*, *35*(21), 1365-1372. doi:10.1093/eurheartj/eht462

Harrell, S. P. (2000). A multidimensional conceptualization of racism-related stress: Implications for the well-being of people of color. *American Journal of Orthopsychiatry*, *70*(1), 42-57. doi:10.1037/h0087722

Harrell, C. P., Burford, T. I., Cage, B. N., Nelson, T. M., Shearon, S., Thompson, A., & Green, S. (2011). Multiple Pathways Linking Racism to Health Outcomes. *Du Bois Review: Social Science Research on Race*, *8*(1), 143-157.

Harrell, S. P., & Sloan-Pena, G. (2006). Racism and Discrimination. In Y. Jackson (Ed.), *Encyclopedia of multicultural psychology*. Thousand Oaks, CA: Sage Publications. doi:10.4135/9781412952668

Hayes, A.F. (2013). *Introduction to mediation, moderation, and conditional process analysis: A regression-based approach*. New York: Guilford Press.

Herd, J. A. (1991). Cardiovascular response to stress. *Physiological Reviews*, *71*(1), 305-330.

Hill, L. K., Kobayashi, I., & Hughes, J. W. (2007). Perceived racism and ambulatory blood pressure in African American college students. *Journal of Black Psychology, 33,* 404 – 421. doi:10.1177/0095798407307042

House, J. S., & Williams, D. R. (2003). Understanding and Reducing and Racial/Ethnic Disparities in Health. In R. Hofrichter (Ed.), *Health and social justice: Politics, ideology, and inequity in the distribution of disease*. San Francisco, CA: Jossey-Bass.

Hubert, H. B., Feinleib, M., McNamara, P. M., & Castelli, W. P. (1983). Obesity as an independent risk factor for cardiovascular disease: A 26-year follow-up of participants in the Framingham Heart Study. *Circulation*, *67*(5), 968-977.

Johnson, R. L. (2005). gender differences in health-promoting lifestyles of African Americans. *Public Health Nursing*, *22*(2), 130-137. doi:10.1111/j.0737-1209.2005.220206.x

Jones, D. R., Harrell, J. P., Morris-Prather, C. E., Thomas, J., & Omowale, N. (1996). Affective and physiological responses to racism: The roles of Afrocentrism and mode of presentation. *Ethnicity & Disease*, *6*(1-2), 109-122.

Jorgensen, R. S., Johnson, B. T., Kolodziej, M. E., & Schreer, G. E. (1996). Elevated blood pressure and personality: A meta-analytic review. *Psychological Bulletin*, *120*(2), 293-320. doi:10.1037/0033-2909.120.2.293

Keith, V. M. & Herring, C. (1991). Skin Tone and Stratification in the Black Community. *American Journal of Sociology*, (3), 760.

Kershaw, K. N., Lewis, T. T., Roux, A. D., Jenny, N. S., Liu, K., Penedo, F. J., & Carnethon, M. R. (2016). Self-reported experiences of discrimination and inflammation among men and women: The multi-ethnic study of atherosclerosis. *Health Psychology*, *35*(4), 343-350. doi:10.1037/hea0000331

Kessler, R. C., Mickelson, K. D., & Williams, D. R. (1999). The prevalence, distribution, and mental health correlates of perceived discrimination in the United States. *Journal of Health and Social Behavior*, *40*(3), 208-230.

Kington, R. S., & Smith, J. P. (1997). Socioeconomic status and racial and ethnic differences in functional status associated with chronic diseases. *American Journal of Public Health*, *87*(5), 805.

Klonoff, E. A., & Landrine, H. (1996). Acculturation and cigarette smoking among African American adults. *Journal of Behavioral Medicine*, *19*(5), 501-514. doi:10.1007/BF01857681

Klonoff, E. A., & Landrine, H. (1999). Cross-validation of the Schedule of Racist Events. *Journal of Black Psychology*, *25*(2), 231-254. doi:10.1177/0095798499025002006

Krieger, N. (1990). Racial and gender discrimination: risk factors for high blood pressure? *Social Science & Medicine (1982)*, *30*(12), 1273-1281.

Krieger, N., Carney, D., Lancaster, K., Waterman, P., Kosheleva, A., & Banaji, M. (2010). Combining explicit & implicit measures of racial discrimination in research. *American Journal of Public Health, 100,* 1485–1492. doi:10.2105/AJPH.2009.159517

Krieger, N., Chen, J. T., Waterman, P. D., Hartman, C., Stoddard, A. M., Quinn, M. M., & ... Barbeau, E. M. (2008). The inverse hazard law: blood pressure, sexual harassment, racial discrimination, workplace abuse and occupational exposures in US low-income black, white and Latino workers. *Social Science & Medicine (1982)*, *67*(12), 1970-1981. doi:10.1016/j.socscimed.2008.09.039

Krieger, N., Rowley, D. L., Herman, A. A., & Avery, B. (1993). Racism, sexism, and social class: Implications for studies of health, disease, and well-being. *American Journal of Preventive Medicine*, *9*(6, Suppl), 82-122.

Krieger, N., & Sidney, S. (1996). Racial Discrimination and Blood Pressure: The CARDIA Study of Young Black and White Adults. *American Journal of Public Health*, *86*(10), 1370-1378.

Krieger, N., Smith, K., Naishadham, D., Hartman, C., & Barbeau, E. M. (2005). Experiences of discrimination: Validity and reliability of a self-report measure for population health research on racism and health. *Social Science & Medicine*, *61*(Building Trust and Value in Health Systems in Low- and Middle- Income Countries), 1576-1596. doi:10.1016/j.socscimed.2005.03.006

Krieger, N., Waterman, P. D., Kosheleva, A., Chen, J. T., Smith, K. W., Carney, D. R., & ... Freeman, E. R. (2013). Racial discrimination & cardiovascular disease risk: my body my story study of 1005 US-born black and white community health center participants (US). *Plos One*, *8*(10), e77174. doi:10.1371/journal.pone.0077174

Kwate, N. O. A., & Goodman, M. S. (2015). Racism at the intersections: Gender and socioeconomic differences in the experience of racism among African

Americans. *American Journal of Orthopsychiatry, 85*(5), 397-408. http://dx.doi.org/10.1037/ort0000086

Kwate, N. A., Valdimarsdottir, H. B., Guevarra, J. S., & Bovbjerg, D. H. (2003). Experiences of racist events are associated with negative health consequences for African American women. *Journal of The National Medical Association*, *95*(6), 450-460.

Landry, B., & Marsh, K. (2011). The Evolution of the New Black Middle Class. *Annual Review Of Sociology*, 373.

Lazarus, R.S., Folkman, S. (1984). Stress, appraisal, and coping. New York: Springer.

Levenstein, S., Smith, M. W., & Kaplan, G. A. (2001). psychosocial predictors of hypertension in men and women. *Archives of Internal Medicine*, *161*(10), 1341.

Lewis, T. T., Barnes, L. L., Bienias, J. L., Lackland, D. T., Evans, D. A., & Mendes de Leon, C. F. (2009). Perceived discrimination and blood pressure in older African American and White adults. *The Journals of Gerontology: Series A: Biological Sciences and Medical Sciences, 64,* 1002–1008. doi:10.1093/gerona/glp062

MacArthur Research Network on Socioeconomic Status and Health. (2000, February). Measures of Psychological Stress. Retrieved from http://www.macses.ucsf.edu/research/psychosocial/stress.php

Manuck, S. B., Kasprowicz, A. L., & Muldoon, M. F. (1990). Behaviorally-evoked cardiovascular reactivity and hypertension: Conceptual issues and potential associations. *Annals of Behavioral Medicine*, *12*(1), 17-29. doi:10.1207/s15324796abm1201_2

McDonough, P., Duncan, G. J., Williams, D., & House, J. (1997). Income dynamics and adult mortality in the United States, 1972 through 1989. *American Journal of Public Health*, *87*(9), 1476-1483.

Mensah, G. A., & Brown, D. W. (2007). An overview of cardiovascular disease burden in the United States. *Health Affairs*, *26*(1), 38-48. doi:10.1377/hlthaff.26.1.38

Mirowsky, J., & Hu, P. N. (1996). Physical impairment and the diminishing effects of income. *Social Forces*, *74*(3), 1073-1096.

Mokdad, A. H., Ford, E. S., Bowman, B. A., Dietz, W. H., Vinicor, F., Bales, V. S., & Marks, J. S. (2003). Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *JAMA: Journal of The American Medical Association*, *289*(1), 76-79. doi:10.1001/jama.289.1.76

Morris-Prather, C. E., Harrell, J. P., Collins, R., Leonard, K. L., Boss, M., & Lee, J. W. (1996). Gender differences in mood and cardiovascular responses to socially stressful stimuli. *Ethnicity & Disease*, *6*(1-2), 123-131.

Mozaffarian D., Benjamin, E. J., Go, A. S., Arnett, D. K., Blaha, M. J., Cushman, M., …& Turner, M. B. (2015). Heart disease and stroke statistics—2016 update: A report from the American Heart Association. *Circulation*. doi: 10.1161/CIR.0000000000000152.

National Heart, Lung, and Blood Institute. (2004). The Seventh Report of the Joint National Committee on Prevention, Detect, Evaluation, and Treatment of High Blood Pressure. U.S. Department of Health and Human Services, NIH Publication No. 04-5230.

National Public Radio, Robert Wood Johnson Foundation, & Harvard T.H. Chan School of Public Health. (2017, October). Discrimination in America: Experiences and Views of African Americans. Retrieved from https://www.npr.org/assets/img/2017/10/23/discriminationpoll-african-americans.pdf

Paradies, Y. (2006). A systematic review of empirical research on self-reported racism and health. *International Journal of Epidemiology*, *35*(4), 888-901.

Paradies, Y., Ben, J., Denson, N., Elias, A., Priest, N., Pieterse, A., & ... Gee, G. (2015). Racism as a determinant of health: A systematic review and meta-analysis. *Plos ONE*, *10*(9), 1-48. doi:10.1371/journal.pone.0138511

Pascoe, E. A., & Smart Richman, L. (2009). Perceived discrimination and health: A meta-analytic review. *Psychological Bulletin*, *135*(4), 531-554. doi:10.1037/a0016059

Pearlin, L. I. (1989). The sociological study of stress. *Journal of Health & Social Behavior*, *30*(3), 241-256.

Peters, R. M. (2004). Racism and hypertension among African Americans. *Western Journal of Nursing Research*, *26*(6), 612-631. doi:10.1177/0193945904265816

Pointer, M. A., Livingston, J. N., Yancey, S., McClelland, M. K., & Bukoski, R.D. (2008). Psychosocial factors contribute to resting blood pressure in African Americans. *Ethnicity & Disease, 18(3*), 289–293.

Poston, W. C., Pavlik, V. N., Hyman, D. J., Ogbonnaya, K., Hanis, C. L., Haddock, C. K., & ... Foreyt, J. P. (2001). Genetic bottlenecks, perceived racism, and hypertension risk among African Americans and first-generation African immigrants. *Journal of Human Hypertension*, *15*341-352.

Priest, N., Paradies, Y., Trenerry, B., Truong, M., Karlsen, S., & Kelly, Y. (2013). A systematic review of studies examining the relationship between reported racism and health and wellbeing for children and young people. *Social Science & Medicine*, *95*(Social Determinants of Child Health), 115-127. doi:10.1016/j.socscimed.2012.11.031

Radloff, L. S. (1977). The CES-D Scale: A self-report depression scale for research in the general population. *Applied Psychological Measurement*, *1*(3), 385-401. doi:10.1177/014662167700100306

Re, R. N. (2009). Obesity-related hypertension. *The Ochsner Journal*, *9*(3), 133-136.

Roberts, C., Vines, A., Kaufman, J., & James, S. (2008). Cross-sectional association between perceived discrimination and hypertension in African-American men and women: the Pitt County Study. *American Journal of Epidemiology*, *167*(5), 624-632

Rospenda, K. M., Richman, J. A., & Shannon, C. A. (2009). Prevalence and mental health correlates of harassment and discrimination in the workplace: Results from a national study. Journal of Interpersonal Violence, 24(5), 819–843.

http://doi.org/10.1177/0886260508317182

Ryan, A. M., Gee, G. C., & Laflamme, D. F. (2006). The Association between self-reported discrimination, physical health and blood pressure: findings from African Americans, Black immigrants, and Latino immigrants in New Hampshire. *Journal of Health Care For The Poor And Underserved*, *17*(2 Suppl), 116-132.

Saydah, S., Bullard, K. M., Cheng, Y., Ali, M. K., Gregg, E. W., Geiss, L., & Imperatore, G. (2014). Trends in cardiovascular disease risk factors by obesity level in adults in the United States, NHANES 1999-2010. *Obesity (Silver Spring, Md.)*, *22*(8), 1888-1895. doi:10.1002/oby.20761

Sellers, R. M., Copeland-Linder, N., Martin, P. P., & Lewis, R. L. (2006). Racial Identity Matters: The Relationship between Racial Discrimination and Psychological Functioning in African American Adolescents. *Journal Of Research On Adolescence (Wiley-Blackwell)*, *16*(2), 187-216.

Sims, M., Diez-Roux, A. V., Dudley, A., Gebreab, S., Wyatt, S. B., Bruce, M. A., & ... Taylor, H. A. (2012). Perceived Discrimination and Hypertension Among African Americans in the Jackson Heart Study. *American Journal of Public Health*, *102*(S2), S258-S265. doi:10.2105/AJPH.2011.300523

Singleton, G. J., Robertson, J., Robinson, J. C., Austin, C., & Edochie, V. (2008). Perceived racism and coping: Joint predictors of blood pressure in Black Americans. *Negro Educational Review, 59,* 93–113.

Smart Richman, L., & Jonassaint, C. (2008). The Effects of Race-related Stress on Cortisol Reactivity in the Laboratory: Implications of the Duke Lacrosse Scandal. *Annals of Behavioral Medicine*, (1). 105.

Smart Richman, L., Pek, J., Pascoe, E., & Bauer, D. J. (2010). The effects of perceived discrimination on ambulatory blood pressure and affective responses to interpersonal

stress modeled over 24 hours. *Health Psy- chology, 29,* 403–411.

Stamler, J., Stamler, R., Riedlinger, W. F., Algera, G., & Roberts, R. H. (1976). Hypertension screening of 1 million Americans. Community Hypertension Evaluation Clinic (CHEC) program, 1973 through 1975. *JAMA*, *235*(21), 2299–2306.

Steffen, P. R., McNeilly, M., Anderson, N., & Sherwood, A. (2003). Effects of perceived racism and anger inhibition on ambulatory blood pressure in African Americans. *Psychosomatic Medicine*, *65*(5), 746-750. doi:10.1097/01.PSY.0000079380.95903.78

Tomfohr, L., Cooper, D. C., Mills, P. J., Nelesen, R. A., & Dimsdale, J. E. (2010). Everyday discrimination and nocturnal blood pressure dipping in Black and White Americans. *Psychosomatic Medicine, 72,* 266–272.

Udo, T., & Grilo, C. M. (2017). Cardiovascular disease and perceived weight, racial, and gender discrimination in U.S. adults. *Journal of Psychosomatic Research*, *100*83-88. doi:10.1016/j.jpsychores.2017.07.007

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. (2012). *Healthy People 2012 Final Review* (DHHS Publication No. 2012-1038). Retrieved from https://www.cdc.gov/nchs/data/hpdata2010/hp2010_final_review.pdf

U.S. Department of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics. (2017). *Health, United States, 2016: With Chartbook on Long-term Trends in Health* (DHHS Publication No. 2017-1232). Retrieved from https://www.cdc.gov/nchs/data/hus/hus16.pdf

U.S. Department of Health and Human Services, Office of Disease Prevention and Health Promotion, Healthy People. (2018, January 4a). About Healthy People. Retrieved from https://www.healthypeople.gov/2020/About-Healthy-People

U.S. Department of Health and Human Services, Office of Disease Prevention and Health

Promotion, Healthy People. (2018, January 4b). Heart Disease and Stroke. Retrieved from https://www.healthypeople.gov/2020/topics-objectives/topic/heart-disease-and-stroke/objectives

Utsey, S. O., Ponterotto, J. G., Reynolds, A. L., & Cancelli, A. A. (2000). Racial discrimination, coping, life satisfaction, and self-esteem among African Americans. *Journal of Counseling & Development*, *78*(1), 72-80.

Vitaliano, P. P., Scanlan, J. M., Zhang, J., Savage, M. V., & Hirsch, I. B. (2002). A path model of chronic stress, the metabolic syndrome, and coronary heart disease. *Psychosomatic Medicine*, *64*(3), 418-435. doi:10.1097/00006842-200205000-00006

Walcott-McQuigg, J., Logan, B., & Smith, E. (1994). Prevention health practices of African American women. *Journal of National Black Nurses' Association*, *7*(1), 25-35.

Will, J. C., & Yoon, P. W. (2013). Preventable hospitalizations for hypertension: Establishing a baseline for monitoring racial differences in rates. *Preventing Chronic Disease*, *10*1. doi:10.5888/pcd10.120165

Williams, D. R., & Collins, C. (1995). US socioeconomic and racial differences in health: Patterns and explanations. *Annual Review of Sociology*, *2,* 349-386.

Williams, D. R., Lavizzo-Mourey, R., & Warren, R. C. (1994). The concept of race and health status in America. *Public Health Reports*, *109*(1), 26–41.

Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine*, *32*(1), 20-47. doi:10.1007/s10865-008-9185-0

Williams, D. R., & Mohammed, S. A. (2013). Racism and Health I: Pathways and Scientific Evidence. *The American Behavioral Scientist*, *57*(8), doi:10.1177/0002764213487340

Williams, D. R., & Neighbors, H. (2001). Racism, discrimination and hypertension: evidence and needed research. *Ethnicity & Disease*, *11*(4), 800-816.

Williams, D. R., Neighbors, H. W., & Jackson, J. S. (2003). Racial/ethnic discrimination and health: Findings from community studies. *American Journal of Public Health*, *93*(2), 200-208.

Williams, D. R., & Williams-Morris, R. (2000). Racism and mental health: The African American experience. *Ethnicity & Health*, *5*(3/4), 243-268. doi:10.1080/135578500200009356

Williams, D. R., Yu, Y., Jackson, J. S., & Anderson, N. B. (1997). Racial differences in physical and mental health: Socio-economic status, stress and discrimination. *Journal of Health Psychology*, *2*(3), 335-351. doi:10.1177/135910539700200305

Winkleby, M. A., Jatulis, D. E., Frank, E., & Fortmann, S. P. (1992). Socioeconomic status and health: how education, income, and occupation contribute to risk factors for cardiovascular disease. *American Journal of Public Health*, *82*(6), 816-820.

Wong, M. D., Shapiro, M. F., Boscardin, W. J., & Ettner, S. L. (2002). Contribution of major diseases to disparities in mortality. *New England Journal of Medicine*, *347*(20), 1585- 1592. doi:10.1056/NEJMsa012979

Wyatt, S. B., Williams, D. R., Calvin, R., Henderson, F. C., Walker, E. R., & Winters, K. (2003). Racism and cardiovascular disease in African Americans. *The American Journal of The Medical Sciences*, *325*(6), 315-331.

Yoon, S., Gu, Q., Nwankwo, T., Wright, J., Hong, Y., & Burt, V. (2015). Trends in Blood Pressure Among Adults with Hypertension: United States, 2003 to 2012. *Hypertension*, (1), 54.