Discrimination and Cardiovascular Disease in Blacks

Cynthia Dolezsar

A Thesis

In the Department

of

Psychology

Presented in Partial Fulfillment of the Requirements

For the Degree of

Doctor of Philosophy (Psychology) at

Concordia University

Montreal, Quebec, Canada

November 2014

CONCORDIA UNIVERSITY
SCHOOL OF GRADUATE STUDIES

This is to certify that the thesis prepared

By: Cynthia Dolezsar

Entitled: Discrimination and Cardiovascular Disease in Blacks

and submitted in partial fulfillment of the requirements for the degree of

Ph.D. Psychology

complies with the regulations of the University and meets the accepted standards with respect to originality and quality.

Signed by the final examining committee:

John Capobianco Chair

Ken Prkachin External Examiner

Robert Kilgour External to Program

Jim Pfaus Examiner

Mark Ellenbogen Examiner

Sydney Miller Thesis Supervisor

Approved by

Chair of Department or Graduate Program Director

Dean of Faculty

ABSTRACT

Discrimination and Cardiovascular Disease in Blacks

Cynthia Dolezsar, Ph.D.

Concordia University, 2014

This thesis is comprised of three studies conducted to further understand the role of racial discrimination in the increased risk of hypertension among individuals of African descent in North America. These studies extend previous research by testing the proposed link between racism and cardiovascular functioning in a laboratory setting and by quantitatively summarizing research findings. The pool of studies having estimated the association between perceived discrimination and blood pressure has yielded mixed results. As well, previous literature reviews have been qualitative and thus did not ascertain the magnitude of the relationship nor have they quantified the effect of moderator variables. To fill this gap in the literature, in the first study a meta-analysis of previous research examining the link between perceived racism and blood pressure was conducted. Results revealed a small relation between perceived racism and hypertension, but not with resting blood pressure. Blacks also experience a higher degree of stress than Whites due to racial discrimination and its consequences. Over time, sustained activation from stress has been posited to lead to structural changes in the vascular system resulting in disease. Exaggerated cardiovascular reactivity to stress has been found to be predictive of future cardiovascular disease (Chida & Steptoe, 2010). Several studies have demonstrated increased vascular reactivity to stress in Blacks as compared to Whites. This line of research, however, has exclusively been tested on American samples. The second study therefore sought to examine reactivity in a Canadian population, where the macro culture for Blacks differs from the U.S. in important ways. Black men were shown to exhibit greater vascular response to laboratory stress. A direct association between perceived racism and reactivity to stress was, however, not observed. Several researchers have proposed that other factors moderate the relation between discrimination and cardiovascular functioning. Anger has extensively been studied in relation to cardiovascular disease but few studies have looked at its relation to perceiving racism. The final study revealed that the influence of perceived racism on blood pressure reactivity depends on how anger is typically expressed. Taken together, these explain the increased risk of cardiovascular disease among Blacks.

Acknowledgements

I will forever be thankful to my dissertation supervisor Dr. Sydney Miller for having seen potential in me as an undergraduate student and supported my application for graduate work. I consider this education a great privilege. I would also like to thank Syd for his input in the development of this work, career advice, and gentle guidance over the years that fostered a sense of independence. My gratitude also extends to Dr. Jennifer McGrath for her extensive support during the development of the meta-analysis. Her unrelenting dedication to our profession is admirable. She is a powerhouse of knowledge which is evident when she mentors and really imparts this on her students. I would also like to thank previous lab members for their work in collecting data. I am also thankful to the members of my doctoral defense committee. I am also grateful for the financial support I have received, in the form of graduate awards from the Canadian Institute of Health Research, as well various sources of supplementary bursaries from Concordia University.

I deeply appreciate the training and guidance offered by my clinical supervisors: Drs. Allyson Harrison, Arunima Khanna, Sam Burstein, Debbie Sook, Hallie Frank, Stephanie Pantel, Wendy Wood, Nathalie Dinh, Suzanne Marcotte, Nathan Kuperstok, Lucie Bonneville, and Dina Giannopoulos. Their lessons have played an important role in shaping me as a practitioner. I would like to give special thanks to Dr. David Rynard who invested a tremendous amount to energy in my training in psychoeducational assessments during predoctoral internship. I am lucky to have him as a role model.

I thank all the professors, Shirley Black, and administrative staff at the Department of Psychology that run our graduate program. I also thank everyone at the Applied Psychology Centre. Together, you have made the graduate journey rich, challenging, and enjoyable. Thanks for all the work you put into us year after year.

I also thank my dearest friends, Gabrielle Ricard and Tamara Anber, for their support and friendship. Ever since my undergraduate work, Simon Meas has been an unwavering source of support for me. Thank you Simon for the countess hours you spent listening to me, encouraging me, and cheering me up when I needed it through the hard times. You were always there for me and I will forever be grateful.

I especially thank my mom, dad, and big sister. I am lucky to have the best family I could ever dream of. Thank you for supporting me in all my endeavors and for always believing in me. Thank you for pushing me to make sure I cross the finish line. Words cannot express how grateful I am for all that you do. My hard-working parents instilled in me a good work ethic, which is a necessary element to balances out my penchant for play and free-spirit. Mom, thank you as well for your work and dedication to AURA which has given me the financial freedom to pursue my doctoral degree. This would not have been possible without your contribution. Dad, thank you as well for all your care and help doing my taxes, car stuff, and fixing everything. Where would we be without you? Nana, I love you and thank you for your advice and support. For that, I dedicate this thesis to my family.

Contributions of Authors

As the first author on the following manuscripts, I took the lead role in the research design, analyses, data interpretation, and writing. Study One is co-authored by Dr. Jennifer McGrath who actively supervised the project and helped write part of the final manuscript. Study One was also co-authored by Alyssa Herzig who coded a subset of the articles to test inter-rater agreement. My supervisor, Dr. Sydney Miller, the final author on all three manuscripts, provided substantial contribution to the editing of the manuscripts. The findings of these manuscripts are original scholarship and distinct contributions to the scientific knowledge in field of Psychology.

Table of contents

List of Figures

Study 1

Study 2

Study 3

List of Tables

Study 1

Study 2

Study 3

List of Abbreviations

SES	Socioeconomic status
BP	Blood pressure
SBP	Systolic blood pressure
DBP	Diastolic blood pressure
TPR	Total peripheral resistance
FBF	Forearm blood flow
FVR	Forearm vascular resistance
MAP	Mean arterial pressure
HR	Heart rate
CO	Cardiac output
SV	Stroke volume

General Introduction

Racial discrimination is a potent and chronic psychosocial stressor characterized by social ostracism and blocked economic opportunities. Despite extensive changes in social policies over the last 60 years, Blacks continue to report racist experiences (Thomas, Herring, & Horton, 1994; Blank, Dabady, & Citro, 2004). Racism has been posited as a contributing factor in racial disparities seen across a wide range of health outcomes (Paradies, 2006b; Williams & Williams-Morris, 2000). Research has show racial discrimination to be associated with poor health in developed countries (Borrell et al, 2007; Bowen-Reid & Harrell, 2002; Finch, Kolody, & Vega, 2000; Gee, Spencer, Chen, & Takeuchi, 2007; Karlsen & Nazroo, 2002; Schulz, Gravlee, & Williams, 2006). Racial discrimination, and its relationship to hypertension and cardiovascular disease, has also received a great deal of attention in the literature. Hypertension is markedly more prevalent in Black as compared to White individuals, has an earlier age of onset, and is associated with greater mortality and morbidity (American Heart Association, 2008; Kurian & Cardarelli, 2007; Lui et al., 2010; Malan et al., 2010).

The pathways through which racial discrimination impacts cardiovascular functioning are likely highly complex and as of yet, not well understood (Brondolo, Hausmann et al., 2011; Williams & Mohammed, 2009). A better understanding of the link between racism and cardiovascular functioning and the factors that buffer or exacerbate its impact is therefore crucial. Reviews of the literature have suggested that this relationship may be moderated by individual differences (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Harrell, Hall, & Taliaferro, 2003; Pascoe & Smart Richman, 2009). Anger expression was a variable of interest in the current work given anger's well-documented relation to hypertension and the limited research on anger expression as it relates to perceived racism and cardiovascular functioning.

The overarching aim of this work is to further the understanding of the role of racism in the racial disparity in hypertension. In this introduction, racial inequalities in cardiovascular disease and other health outcomes are presented along with a discussion of the posited causes driving these differences. Next, racial discrimination is discussed as a possible contributor of the increased risk of hypertension in Blacks followed by a review of the research exploring this proposed association. Finally, anger and the expression of anger are considered as possible

pathways through which racial discrimination affects cardiovascular functioning.

Racial disparities in hypertension

Hypertension is a primary risk factor in the development of cardiovascular disease (Burt et al., 1995; Fuster & Pearson, 1996). The disproportionate prevalence of hypertension among Blacks in Canada and the United States, as compared to Whites, has been well documented (e.g., Burt et al., 1995; Centers for Disease Control and Prevention, 2011; Fields, 2004; Hajjir & Kotchen, 2003). Hypertension occurs at a younger age, is more severe, as well as being associated with greater pressure related target-organ damage and higher cause-attributable mortality in Blacks as compared to Whites in North America (American Heart Association, 2008; Ferdinand & Armani, 2007; Kurian & Cardarelli, 2007; Lui et al., 2010; Malan et al., 2010).

The majority of research investigating the causes of these inequalities has focused on the influence of socioeconomic status (SES), health behaviors, and genetics (Dressler, Oths, & Gravlee, 2005). SES and access to health care have profound and well-documented impacts on cardiovascular health. Williams and Williams-Morris (2000) discuss the decreased socioeconomic mobility of Blacks in the U.S. due to residential segregation as well as limited and unequal access to services and resources, education, and employment opportunities. Blacks are twice more likely to be unemployed and three times more likely to be poor compared to Whites and more than half of Black households in the United States are categorized as economically vulnerable. Given the relationship between SES and cardiovascular disease, examining racial disparities without considering SES can attribute too much influence to race (Isaacs & Schroeder, 2004). Research has looked at the combined and independent effects of SES and race on health. Socioeconomic measures indeed account for much of the racial differences, but independent effects of race are also observed (Adler & Rehkopf, 2008). Furthermore, racial disparities in cardiovascular disease persist after statistically adjustment, even at "equal" levels of SES (Hayward, Crimmins, Miles, & Yang, 2000; Wyatt et al., 2003). SES indicators are not equivalent across race. Blacks earn less than Whites at comparable years of schooling and Blacks have less purchasing power at any given level of income (Pamuk et al., 1998). These findings suggest that the experience of SES is at "equal" level is different among racial groups and therefore when statistical controls for SES explain racial differences, there may

be some processes that are not captured. In addition, the prevalence of hypertension in disadvantaged Hispanics, Asian Americans, and Native Americans is similar to that of Whites and therefore does not fully explain the increased risk of hypertension in disadvantaged Blacks (Kaplan, 1994). Therefore, inequalities in hypertension cannot solely be attributed to economic disadvantage.

Secondly, Blacks also tend to present with higher risk profiles for hypertension than do Whites, including higher rates of obesity, physical inactivity, cigarette smoking, excess sodium, limited access to quality health care, and family history of cardiovascular disease (Crimmins, Kim, Alley, Karlamangla, & Seeman, 2007; Hayward et al., 2000). Crimmins et al. (2007) found that even after controlling for age, gender, low education, and poverty score, the risk for high blood pressure was higher in Blacks than in Whites. Interestingly, the risk remained higher for Blacks than White even after controlling for health behaviors (smoking, poor diet, physical activity), and access to health care as well. Redmond, Baer, and Hicks (2011) also found that racial disparities in blood pressure exists after adjusting for demographics, SES, clinical characteristics and health behaviors which included smoking, physical activity, energy intake, sodium, alcohol, and fiber consumption.

Thirdly, studies have investigated the potential genetic factors contributing to the increased prevalence of hypertension in Blacks. A recent review by Hall, Duprez, Barac, and Rich (2012) examined the genetic findings relating to arterial stiffness and blood pressure in African Americans. They concluded that the evidence linking genetics with blood pressure is at an early stage and little evidence supports a role of genetics in arterial stiffness in any racial group. Other studies also suggest that genetic factors do not appear to account for the racial disparity (Hermann et al., 2000; Hertz, Unger, Cornell, & Saunders, 2005; Province et al., 2000; Xie, 1999). Another line of research has compared the rates of hypertension in Blacks living in Africa, Caribbean, and the U.S. Cooper, et al. (2005) found that the rates of hypertension among Blacks living outside of the U.S. are not unusually high. The authors suggested that the increased prevalence of hypertension in the U.S. relative to Africa and the Caribbean were attributed to social and environmental factors. These findings further challenge the role of biological mechanisms as a plausible explanation (Cooper et al., 1997; 2005).

Taken together, the reasons for the markedly higher rates of hypertension and consequences are not entirely clear but likely involve the combined influences of SES, health behaviors, and some additional factors. These additional factors, namely environmental or social factors, are likely involved in the increased risk for hypertension in North American Blacks. In an effort to better understand these racial disparities, researchers are pursuing other lines of inquiry. Racial discrimination is increasingly receiving attention in the literature as a determinant of racial inequalities in health (Braveman, Egerter, & Williams, 2011; Brondolo, Brady ver Halen, Pencille, Beatty, & Contrada, 2009; Goodman, 2000; Mujahid, Diez Roux, Cooper, Shea, & Williams, 2011; White et al., 2011).

Racial discrimination

Racism refers to a system that categorizes the population into groups based on race (Bonilla-Silva 1996). The underlying belief driving this categorization is that some racial groups are inherently superior to others. In a seminal paper, Clark, Anderson, Clark and Williams (1999) defined racism as "the beliefs, attitudes, institutional arrangements, and acts that tend to denigrate individuals or groups because of phenotypic characteristics or ethnic group affiliations" (p.805). Racism can be deeply rooted social norms and institutions and manifests as negative stereotypes, negative emotions, or unfair treatment (Jones, 1997). Racism can occur at three levels: internalized (i.e. racist attitudes or beliefs in one's worldview), interpersonal (between individuals), and institutional racism (inequalities in access to labor, education, and other resources; Berman & Paradies, 2010; Paradies, 2006a). Racial discrimination leads to unfair inequalities in power, resources, and opportunities across racial groups.

Despite important changes in social policies in the last 60 years, Blacks report that discrimination persists in multiple contexts including housing, labor markets, justice, education, and in everyday interactions with others (Blank et al., 2004). Moreover, some forms of racism are difficult to perceive (Paradies, 2006b). Internalized racism, due to its nature, can go unnoticed by those suffering from it and institutional racism can be so pervasive that it is invisible. Also, study participants are more likely to underreport than over report experiences of racial discrimination (Kaiser & Major, 2006). Most of the research on discrimination targets the perception of interpersonal racism and often captures only a subset of the oppressed experience.

In the current paper, the terms racism, discrimination, and racial discrimination are used interchangeably.

The perception of racism is posited to have adverse effects on mental and physical health. Feelings of distress, frustration, anger, and powerlessness are common reactions following racist experiences (Kessler, Mickelson, & Williams, 1999; McNeilly et al., 1996). In a review paper, Paradies (2006b) reported self-reported racism was associated with several negative mental health outcomes including distress, depression, and anxiety. It was also found to be inversely associated with positive health outcomes such as self-esteem and satisfaction ratings across a number of studies. Discrimination has also been associated with a range of poor physical health outcomes in addition to cardiovascular disease, including respiratory conditions, diabetes (Karlsen & Nazroo, 2002), somatic complaints (Bowen-Reid & Harrell, 2002), chronic health conditions (Finch et al., 2000; Gee et al., 2007), and morbidity (Clark et al., 1999; Krieger 1999; Williams, Neighbors, & Jackson, 2003).

Considered more broadly, rejection, social ostracism, and isolation are powerful upsetting experiences which have been found to be associated with poor physical and mental health (Cacioppo, Hawkley, & Berntson, 2003; Williams, 2001). Evidence suggests that rejection due to race may be particularly damaging. Interpersonal rejection by someone that is of a different race has been found to be even more stressful than by someone of the same race (Jamieson, Koslov, Nock, & Berry Mendes, 2012). Furthermore, rejection by an out-group member engenders harmful attributions, emotions, and behaviors that are different than those elicited by in-group members (Crocker, Voelkl, Testa, & Major, 1991; Mendes, Major, McCoy, & Blascovich, 2008).

Mechanisms linking racism to hypertension

Racial discrimination can affect health through several pathways. First, discrimination can lead to negative mood states which in turn can adversely affect health. Mental health correlates of racism are the most researched health outcome (Krieger, 1999; Williams & Williams-Morris, 2000). As previously mentioned, self-reported racism was associated with several negative mental health outcomes including depression and anxiety (Paradies, 2006b). Research demonstrates that depression and anxiety are associated with increased levels of

mortality (Bush et al., 2001; Davidson et al., 2010; Dickens et al., 2008; Frasure-Smith & Lesperance, 2008; van Melle et al., 2004). Depressive disorders have been associated with future onset of cardiovascular disease (Lett et al., 2004), and a recent review by Fiedowrowicz (2014) also reported that depression is an independent risk factor for cardiovascular disease. Well powered studies consistently demonstrate a large association between depression and cardiovascular morbidity and mortality, after adjusting for confounding variables. Multiple mechanisms linking depression and anxiety to cardiovascular disease have been posited including serotonergic dysfunction, systemic inflammation and immune activation, hypothalamic-pituitary-adrenal axis and autonomic nervous system dysfunction, vascular changes, genetics, medical comorbidities, and behavioral factors (Khan, Kulaksizoglu, & Cilingiroglu, 2010; Sher, Lolak, & Maldonado, 2010; Summers, Martin, & Watson, 2010).

Second, differential access to social, educational, and material resources has been shown to have both direct and indirect effects on health status (Adler & Rehkopf, 2008; Adler & Snibbe, 2003; Gallo & Matthews, 2003). Differences in the distribution of basic resources such as nutrition, sanitary living conditions, and health care affect health. Moreover, discrimination can affect both the access and the quality of medical treatment. In segregated communities, medical facilities are more likely to close, pharmacies are less adequately stocked, and residents are more likely to be treated by lower-quality physicians less able to refer to specialty care (Williams & Jackson, 2005). Blacks also receive fewer medical procedures and poorer quality medical care than do Whites even after accounting for differences in health insurance, SES, severity of disease, co-morbidity, and type of medical facility (Smedley, Stith, & Nelson, 2003). Evidence suggests that internalized racism is a likely determinant of this pervasive bias in the delivery of care (van Ryn, 2002; Green et al., 2007).

Third, coping responses used to manage stress or reduced self-regulation abilities from racism can lead to adopting unhealthy behaviours and disengagement of healthy behaviours. Several studies have demonstrated a consistent relationship between racism and risky behaviours such as smoking and substance use (Borrell et al., 2007; Choi et al., 2006; Landrine and Klonoff, 2000). Others have found that racism relates to less use of preventative health care such as cholesterol testing or mammography (Hausmann et al., 2008; Trivedi and Ayanian, 2006), and less adherence to medication regimens (Casagrande et al., 2007; Thrasher et al., 2008).

Fourth, responses to acute and chronic stressors can lead to structural and functional changes in physiological systems (Brondolo, Brady, Libby, & Pencille, 2011; Brondolo, Hausmann, et al., 2011; Harrell et al., 2011; Paradies, 2006b; Pascoe & Smart Richman, 2009). Stam (2007) identified several physiological systems where severe stress has been shown to have a negative impact: the neuroendocrine system, the gastrointestinal system (gastrointestinal ulcers, irritable bowel syndrome), pain sensitivity, chronic pain, immune function (suppressed immunity), and the cardiovascular system (increased heart rate and blood pressure responses).

Reactivity Hypothesis

Much research conducted in this area is in line with this fourth pathway and conceptually grounded in the cardiovascular reactivity hypothesis where heightened reactivity to stress is posited to play a causal role in the development of hypertension (Krantz & Manuck, 1984). Perceptions of racial discrimination are stressful life experiences (Clark, Anderson, Clark, & Williams, 1999) that create emotional distress (Landrine, Klonoff, Corral, Fernandez, & Roesch, 2006) and serve as potent psychosocial stressors (Brondolo, Gallo, & Myers, 2009). When a person experiences stress, there is an activation of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis system. Catecholamines are then released which result in increased blood pressure and heart rate (Kubzansky & Kawachi, 2000). Exaggerated cardiovascular activity over the course of the lifespan has been posited to lead to structural changes in arterial walls that may then ensue in a sustained increase of vasculature resistance, and thus, high blood pressure (Anderson, McNeilly, & Myers, 1992; Krieger, 1990).

The last three decades have witnessed a growing body of research examining the predictive value of the reactivity hypothesis in linking exaggerated responses with an elevated risk for hypertension. Reactivity is usually measured by the change in cardiovascular functioning following an aversive or challenging laboratory task such as the cold pressor, mental arithmetic, or public speaking (Manuck, Kamark, Kasprowicz, & Waldstein, 1993). Cardiovascular reactivity is typically conceptualized as a trait characteristic or an individual difference. It is often discussed as a two-dimensional disposition where individuals vary according to dimensions of "vascular" (e.g. peripheral resistance) and "cardiac" (e.g. cardiac output) changes in response to stress (Kasprowicz, Manuck, Malkoff, & Krantz, 1990; Allen, Boquet, & Shelley, 1991). Several processes may contribute to individual differences in

reactivity including peripheral inputs (e.g. vascular morphology or receptor function) as well as central nervous system inputs (e.g. centrally mediated autonomic activation, appraisal; Lovallo & Gerin, 2003).

The theory that an exaggerated response to stress plays a causal role in the development of hypertension remains controversial. However, several prospective studies have found that increased cardiovascular responses to stress were predictive of future elevations in resting blood pressure and hypertension, even when accounting for traditional risk factors (Carroll, 2003; Chida & Steptoe, 2010; Matthews, 2003; 2004; Treiber, 2003). Exaggerated responses to stress may therefore serve as a risk marker for disease.

Research on racial differences in cardiovascular reactivity

Several studies have compared the cardiovascular response patterns of Black and White participants in an effort to test the idea that Blacks have heightened reactions to stress which may explain their increased risk of hypertension. Blacks have indeed been shown to have greater vascular responses to a variety of stressors as compared to Whites (Anderson, Lane, Muranaka, Williams, & Houseworth, 1988a; Dorr, Brosschot, Sollers, & Thayer, 2007; Kelsey, Patterson, Barnard, & Alpert, 2000; Light, Turner, Hinderliter, & Sherwood, 1993; Reimann et al., 2012; Saab et al., 1992; Thomas, Neleson, Ziegler, Natarajan, & Dimsdale, 2009; Treiber et al., 1990). Furthermore, Blacks exhibit similar or diminished cardiac responses to stressors as compared to Whites (Light, Turner, Hinderliter, & Sherwood, 1993; Saab et al., 1992; Saab et al., 1997). Racial differences in reactivity have not always been reported, though it is unclear why other studies have failed to observe these effects (Anderson, Lane, Monou, Williams, & Houseworth, 1988b; al'Absi et al., 2006; Delhanty, Dimsdale, & Mills, 1991; Dimsdale, Ziegler, Mills, Delehanty, & Berry, 1990; Saab et al., 1997).

Greater reactivity to stress found among Black participants, as compared to Whites, was largely mediated through an increase in peripheral vascular resistance (Anderson, McNeilly, Myers, 1991; Falkner, 1996; Calhoun, 1992; Murphy, Alpert, Walker, 1994; Ray & Monahan, 2002; Stein, Lang, Singh, He, & Wood, 2000). A recent review of racial differences in the functioning and mechanics of resistance in arteries reported that normotensive Blacks as compared to Whites exhibit increased vascular reactivity to sympathetic stimulation, attenuated

responses to vasodilators, and a relatively narrow vascular lumen (Taherzadeh, Brewster, van Montfrans, & VanBavel, 2010). Blacks are also more frequently treated with diuretics, which reduce blood volume, for hypertension as compared to Whites (Miller & Doshi, 2006). Furthermore, a vascular rather than a myocardial pattern of regulating blood pressure is more frequently observed in Blacks (Anderson, Lane, Monou, Williams, & Houseworth, 1988a; Anderson, Lane, Muranaka, Williams, & Houseworth, 1988b). Vascular patterns may be particularly harmful given the role of increased total peripheral resistance (TPR) in hypertension (Devereux, Savage, Sachs, & Laragh, 1983) and studies have linked elevations in TPR at rest and during stress with markers of disease risk (Goldberg et al., 1996; Sherwood, Johnson, Blumenthal, & Hinderliter, 1999; Treiber, et al., 1993). Thus, differential exposure to stress, in combination with divergent mechanism of blood pressure regulation and vascular functioning, are likely contributors to well-documented Black–White disparities in hypertension.

Racial differences in reactivity to stress have been more consistently found in men than women (Saab et al., 1997; Light, Turner, Hinderliter, & Sherwood, 1993; Kelsey, Patterson, Barnard, & Alpert, 2000). Saab et al. (1997) found that Black men differed in their response to laboratory challenges compared to black women, white men, and white women, who did not differ from each other. Light et al. (1993) found that Blacks showed higher total peripheral resistance, whereas Whites showed greater heart rate and cardiac output increases. Among women, the same racial-group differences were observed, but not as consistently across tasks then did men. Kelsey et al. (2000) also found that men showed consistency in their cardiac and vascular reactivity across tasks but not women. These findings suggest that black-white differences in physiological reactivity observed in men may have limited generalizability for women. Looking at the broader literature of sex differences irrespective of race, cardiovascular and sympathoadrenal responses to stress in men tend to be characterized by increased blood pressure and catecholamine responses (Frankenhaeuser et al., 1978, 1980; Matthews and Stoney, 1988; Lepore et al., 1993; Becker et al., 1996; Steptoe et al., 1996; Traustadó´ttir et al., 2003; Matthews et al., 2001). Women (after puberty and before menopause), on the other hand, tend to have more pronounced changes in heart rate responses (Matthews and Stoney, 1988; Tersman et al., 1991; Heponiemi et al., 2004; Kudielka et al., 2004).

An important problem with this area of research is that the overwhelming majority of studies comparing racial patterns of reactivity have been conducted on American participants. Of the few conducted on samples outside of the U.S., Huisman et al. (2013) found that that Black South African men showed higher resting total vascular resistance as well greater reactivity to a stressor (Stroop color word conflict), as compared to Caucasian South African men who showed a decrease in vascular resistance following a stressor. Somova (1992) reported that Blacks in Zimbabwe showed greater vascular response to the cold pressor whereas Whites showed greater increases in cardiac output. More studies are needed to further test the generalizability of the U.S. findings.

To date, no research is available on the cardiovascular response patterns of Canadian Blacks despite the increased risk of hypertension in Canadian Blacks (Lui et al., 2010). A Canadian sample may be particularly useful in testing the reactivity hypothesis given that Canada's Blacks are a visible minority group but have not been subjected historically to the same degree of institutionalized racism and the SES experience is likely much different than that of American Blacks. Institutionalized racism and systematic discrimination have not been a part of Canada's history as they have in American history (Levitt, 1997). Furthermore, the majority of American Blacks are descendants of immigrants who moved to the U.S. involuntarily through the slave trade, whereas Canadian Blacks are mostly self-selecting immigrants and their descendants (Attewell, Kasinitz, & Dunn, 2010). The majority (72%) of Canadian Blacks are of first generation whereas most American Blacks are of third generation or more (86%; Attewell, Kasinitz, & Dunn, 2010). Canadian Blacks still report experiences with racism (Etowa, Keddy, Egbeyemi, & Eghan, 2007), though no study has looked at how these reports compare to those of American Blacks. Given these important differences, examining the stress response of Canadian Blacks would be valuable in further testing the reactivity hypothesis in explaining racial disparities in hypertension. Testing racial differences in reactivity with a Canadian sample would also help to evaluate the contribution of race, more independently from SES and related factors. Black Canadians have not experienced neighbourhood racial segregation comparable to Blacks in the U.S. and as such, have not been exposed to a high concentration of poverty, crime, and lack of resources (Hou, 2003). Universal health care and lower costs of education in Canada likely decreases the influence of reduced access to resources in Canadian Blacks and make for a different SES experience than that of American Blacks.

Research linking discrimination and cardiovascular functioning

Studies have also used analogues of racist experiences to study the effects of racism in the laboratory. Jones, Harrell, Morris-Prather, Thomas, & Omowale (1996) found that Black women showed an increase in heart rate and digital blood flow in response to films depicting racism. They also reported that greater changes were observed during blatant racism condition than subtle racism. McNeilly et al. (1995) compared the effects of a debate about racist attitudes and another nonracist debate in a college sample of Black women. They found that the racist debate elicited greater cardiovascular reactivity and delayed recovery than did the nonracist debate. They also found that social support buffered the cardiovascular response to the racist debate. Likewise, Armstead and colleagues (1989) demonstrated that Black college students had greater increases in BP while watching film excerpts depicting racist situations than while watching films depicting anger-provoking, nonracist situations. In contrast, Sutherland and Harrell (1986) examined the response patterns in Black female undergraduate students to racially noxious, fearful, and neutral scenes and found that the racially noxious films produced similar increases in blood pressure to the fearful scenes. Similarly, a study using a biracial college sample found that both Black and White participants exhibited greater increases in diastolic blood pressure to both racist and anger-provoking films compared to the neutral film (Fang & Myers, 2001). The racist film did not produce greater increases than the anger-provoking film. Together, these findings suggest that individuals show increased reactivity to stressful stimuli, but it remains unclear whether racist analogues produce greater physiological responses than nonracist stressful stimuli. Regardless, these results are in line with the hypothesis that racial discrimination is a stressor that can negatively impact the cardiovascular health of Blacks through pathogenic processes associated with physiologic reactivity.

Other lines of inquiry have examined the link between racism, as measured by a self-report questionnaire, and reactivity. Clark (2000) found that Black women who reported more frequent experiences with racism had greater increases in diastolic blood pressure to a public speech stressor. He also demonstrated that perceived racism was associated with protracted sympathetic stimulation measured by poorer post-task recovery. Prolonged recovery, as well as exaggerated reactivity, have been posited to culminate in higher resting blood pressure levels (Treiber et al., 2003; Anderson, McNeilly, & Myers, 1991; Stewart & France, 2001). In a larger

sample of Black women and using a different measure of perceived racism, Clark (2006b) also found racism to be positively related to changes in systolic blood pressure during a speech stressor. Social support was reported to moderate the relationship between perceived racism and blood pressure changes. In 2003, Clark reported that perceived racism was not independently related to blood pressure changes to a subtraction stressor but did interact with social support to predict reactivity. Thomas, Nelesen, Malcarne, Ziegler, and Dimsdale (2006) used phenylephrine, a pharmacologic stimulus rather than a psychological stressor. Phenylephrine is an alpha-1 agonist that stimulates the same pressor receptors as norepinephrine and it has been demonstrated that it mimics the short-term effects of stress on blood pressure by increasing vasoconstriction (Ziegler et al., 1995; Elliott, Sumner, McLean, & Reid, 1982). They found that more frequent reports of discrimination were positively associated with greater diastolic blood pressure reactivity to phenylephrine. Similar increases in diastolic blood pressure were observed in both Black and White adults. Perceived racial discrimination mediated the relationship between race and responses to phenylephrine. Therefore, individuals who perceive more experiences with racism seem to have greater physiological increases in responses to a variety of stressors and this relationship may be influenced by social support. Though most studies in this area have used samples consisting of exclusively Black participants, Thomas et al. (2009) findings suggest that regardless of race, stress from perceived discrimination may lead to increased physiological responses.

Other studies have looked at the relation between self-reported perceived racism and resting blood pressure though findings are mixed. Several studies conducted with mostly Black participants reported a positive association between perceived racism and resting blood pressure (Guyll et al., 2001; Kaholokula et al., 2012; Smart Richman et al., 2010). Unexpectedly, other researchers have found a negative relation between perceived discrimination and resting blood pressure (Clark & Gochett, 2006; Clark, 2003; Moghaddama et al., 2002). Finally, others did not find the two to be significantly related (Krieger et al., 2008; Din-Dzietham et al., 2004). These studies have used a variety of measures to assess discrimination, drew from samples ranging in age and gender, as well as other methodological variables. This may explain why the results are mixed. McClure et al. (2010a) found perceived racism to be positively associated with resting systolic blood pressure in men whereas in women, a negative association was observed. Klimentidis et al. (2012) showed that discrimination was positively associated with resting

systolic blood pressure in Black participants but was not associated in Hispanic or White participants.

In another vein, studies have also examined self-reported perceived racism as it related to hypertension. While some studies have reported a positive association (Din-Dzietham et al., 2004; Krieger, 1990; Tull et al., 1999), Clark & Gochett (2006) reported a negative relation. Others still reported perceived racism to be unrelated to hypertension (Krieger et al., 2008; Todorova et al., 2010). Again here, these studies have used a variety of assessment measures to quantify perceived discrimination and hypertensive status, as well as using samples drawn from a range of age groups and mixed gender. Several studies have reported varied effects based on moderating variables. For instance, Krieger et al. (2010) found the relation to be stronger among participants with lower education. Mezuk et al. (2011) found a positive relation between discrimination and hypertension in Black and White participants, whereas a negative relation emerged among Hispanic respondents. Roberts et al. (2008) reported a negative relation among men and did find hypertension to be related to discrimination in women. Rahman et al. (2008) reported that the relation depended on location where an inverse association was observed in participants in Massachusetts and a positive association among Florida respondents.

A final line of research has used at ambulatory blood pressure monitoring to further understand this relationship. It has become a commonly used method of evaluating the lab-to-life generalizability of measuring cardiovascular reactivity. Several studies have found strong associations between ambulatory monitoring and cardiovascular reactivity (Cornish, Blanchard, & Jaccard, 1994; Light, Turner, Hinderliter, & Sherwood, 1993; Steptoe & Cropley, 2000). Ambulatory blood pressure monitoring has also been shown to be superior over clinic blood pressure measurement in predicting major cardiovascular events (Verdecchia, Angeli, Gattobigio, & Porcellati, 2003). Findings using ambulatory data seem to be more consistent in the expected direction. Perceived racism has been found to be positively associated with daytime and nighttime blood pressure in several studies (Brondolo et al., 2008; Hill et al., 2007; Singleton et al., 2008; Steffen et al., 2003).

Overall, perceived racism appears to have some relation to blood pressure functioning though results are not consistently found. Previous reviews have also noted the inconsistency in the literature and discussed the complexity of the relationship between racism and health.

Paradies (2006b) conducted a systematic review of the link between racism and a range of health and health-related outcomes. Most studies found no association between self-reported racism and cardiovascular outcomes including hypertension, heart diseases and increased heart rate. The strongest associations were found for mental health outcomes and health-related behaviors such as smoking and substance use. Paradies (2006b) found that a number of variables modified the association but these were discussed across all outcomes. They included a strong sense of racial identity, spirituality, hardiness, SES, age, John Henryism (the tendency to work hard on disproving stereotypes of laziness and inability), and coping style. Included among a range of health outcomes, Williams & Mohammed, 2009 reviewed the relationship between discrimination and blood pressure, as well as cardiovascular disease. They characterized the relation as complex and unclear. Furthermore, they noted that the association seemed more consistent among certain subgroups including those born outside of the U.S., at high levels of stress, with passive coping styles, and high on trait anger. Wyatt et al. (2003) concluded that the available research findings are inconsistent, but point to a plausible association between discrimination and cardiovascular disease. Brondolo, Rieppi, Kelly, and Gerin (2003) focused exclusively on cardiovascular health and described the results to be "mixed and inconsistent". Later, Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe (2011) described the relation as "weak" and noted that findings are more consistent with ambulatory blood pressure and institutional forms of racism.

Given the inconsistencies in the literature, a quantitative review of the literature would be important to summarize the pool of findings. As described in the above paragraph, few reviews have discussed the link with cardiovascular outcomes exclusively. Many discussed them in combination with other physical health, mental health, and health-related behaviors. Furthermore, all of the reviews discussed the relationship between perceived discrimination and health in qualitative terms or using "vote-count" methods to describe the association with the exception of the meta-analysis conducted by Pascoe and Smart Richman (2009) on multiple health outcomes. A quantitative review is necessary to better estimate the magnitude and direction of the relationship. Furthermore, these qualitative reviews were not able to quantify the contribution of moderator variables discussed. A meta-analytical review would be helpful in addressing these shortcomings and better evaluate the relation between racism and hypertension and moderating factors.

Within the stress literature, there is a broad recognition that the effects of a particular stressor work together with other factors to impact health. These can include previous experiences, social support, psychological and behavioral coping responses, helplessness and perceived control, family history of psychological disorders, and genetic vulnerability among others (Yehuda, Bryant, Marmar, &Zohar, 2005; Stam 2007; Baum, Cohen, & Hall, 1993; Carter 2007; Harrell, Hall, & Taliaferro, 2003; Pascoe & Smart Richman, 2009). These factors can either mediate the effects of the stressor or interact with it to mitigate or exacerbate its effects on health. Understanding the complex ways in which psychological stressors affect health requires an understanding of the large variation in individual factors that relate to how one responds to stress exposure. Research on racial discrimination and health also needs to attend to the individual and situational factors that might affect the underlying processes. Research on these individual factors is under-developed. A better understanding of these factors may also help reconcile the inconsistent findings in this literature.

Anger

Anger is one such factor that may be particularly important, and that has not been discussed in much detail in previous reviews. It may be assumed that members of social groups that are systematically treated unfairly or disrespected experience more anger. Blacks would be expected to experience more anger than Whites given the social ostracism and economic disadvantages of racial discrimination. Indeed, Blacks report higher levels of anger than Whites (Barefoot, Dahlstrom & Williams, 1983; Scherwitz, Perkins, Chesney & Hughes, 1991). Anger is one of the most frequently reported psychological effects of racism (Bullock & Housten, 1987; Landrine & Klonoff, 1996) and the primary reason why Blacks seek out psychotherapy (National Institute of Mental Health, 1983). Furthermore, Blacks are also more likely to perceive interracial hostility and tend to interpret ambiguous situations as more threatening than do Whites (Scherwitz, Perkins, Chesney & Hughes, 1991; Chen & Matthews, 2001). Therefore, everyday encounters perceived as racial discrimination, such as driving, in restaurants, at work, etc., fuel feelings of anger among many Blacks (Cose 1993; Feagin and McKinney 2003; Fields et al. 1998).

Anger-related emotions are conceptualized as consisting of three dimensions: emotional, cognitive, and behavioral (Martin, Watson, & Wan, 2000). The emotional dimension is anger

which is characterized by a "strong feeling of distress or displeasure in response to a specific provocation experienced" (Thomas, 1993). Anger typically relates to feelings of being treated unjustly and is accompanied by subjective arousal. Hostility can be understood as a cognitive process characterized by lasting negative, or cynical, attitudes towards others and the environment. The behavioral dimension is anger expression, which refers to how one copes with angry feelings (Spielberger et al., 1985). Anger can be expressed outwardly towards others by verbal or physical aggressive behaviour (anger-out) or it can be suppressed or withheld (anger-in). Some evidence suggests that these patterns consist of two dimensions rather than opposing ends on a continuum (Spielberger & Sydeman, 1994). A third form of expression is anger-control, which includes items such as "…controls my temper" but this measure has not been used frequently in research (Spielberger et al., 1985). Furthermore, acute feelings, thoughts and behaviors, which can be considered state manifestations of anger, should be distinguished from trait anger which is the chronic disposition to feel, think and act angrily (Mittleman, Maclure, & Sherwood, 1995).

The long term impact of anger-related emotions on cardiovascular health has been studied extensively in prospective studies. Chida and Steptoe (2009) conducted a meta-analysis examining the relation between anger and hostility and coronary heart disease in prospective studies. They reported a hazard ratio of 1.19 (95% CI: 1.05 to 1.35) indicating that a greater risk of coronary heart disease is associated with higher anger and hostility in prospective studies starting with healthy populations. A hazard ratio of 1.24 (95% CI: 1.08 to 1.42) for poorer prognosis (mortality or events) was found in studies using populations with known coronary heart disease. As well, anger-related emotions were more harmful in healthy males than females. However, in studies that fully controlled for possible behavioral covariates (i.e. smoking, physical activity, SES), no significant associations between anger and hostility and coronary heart disease remained in either the healthy or patient populations. In a prospective study comparing the effects of five hostility measures in a sample healthy sample on cardiovascular disease and ischemic events, higher cynicism and lower anger control predicted cardiovascular disease and ischemic events, controlling for age (Haukkala, Konttinen, & Laatkainen, 2010). When all hostility measures and other covariates were included, only low anger control (RR = .81) and low anger-out (RR = .67) predicted cardiovascular disease events. Another study found that observed hostility (obtained from a structured interview) predicted a 2-fold increase of

incident ischemic heart disease in ten-year follow-up, even after controlling for traditional risk factors and patient-reported hostility (Suls & Bunde, 2005). A meta-analysis by Compare et al. (2014) found that anger but not hostility (OR = 1.38, 95% confidence interval, CI = 1.12-2.31) predicted the presence of coronary artery plaque. Taken together, the evidence suggests that anger-related emotions contribute to cardiovascular disease and that the magnitude of the effect size decreases when other risk factors are considered. Furthermore, the specific dimensions of anger that are harmful remain unclear.

Anger expression has been linked to cardiovascular disease progression in several studies, although the preferred mode of anger expression remains unclear. A longstanding hypothesis in the field of behavioural medicine has been that the inhibition of emotion puts individuals at an increased risk for disease. Pennebaker (1995) proposed that emotion inhibition requires physiological "work" and sustained inhibition places cumulative stress on the body, resulting in an increased risk to diseases. Anger inhibition has been linked with increased reactivity to stress and cardiac risk (Clark, Anderson, Clark, & Williams, 1999; Harburg, Erfurt, Hauenstein, Chape, Schull, & Schork, 1973; Haynes, Feinleib, & Kennel, 1980; Suarez & William, 1990, Richter et al. 2011; Denollet, Gidron, Vrints, and Conraads, 2010).

In line with these findings, a meta-analysis of studies looking at ambulatory blood pressure reported that the outward expression of anger was inversely related to diastolic blood pressure (Schum et al., 2003). However, research has not consistently supported this hypothesis. Patients with hypertension or coronary artery disease were more likely to express their anger outwardly than those in control groups (Hosseini, Mokhberi, Mahammadpour, Mehrabianfard, and Lashak, 2011; Schmidt et al., 2013). Taken together, evidence is more consistently found supporting the harmful influence of anger suppression on cardiovascular health, compared to outwardly anger expression.

The influence of anger expression styles on cardiovascular outcomes seems to be particularly important in Black individuals. Harburg et al. (1973; 1979) reported that anger inhibition was more predictive of cardiovascular risk factors in Blacks than in Whites. Johnson, Schork & Spielberger (1987) demonstrated that resting blood pressure levels were higher at lower levels of anger-in among Black than White women. Poole et al. (2006) found that anger-in, but not anger-out, played a modulating role in predicting reactivity in Black but not in White

participants. Magai, Kerns, Gillespie, and Huang (2003) reported that anger inhibition was a significant predictor of circulatory disease for Black but not White older adults and the relation between anger and circulatory disease was mediated by anger inhibition. Brownley, Light, and Anderson (1996) found that low hostility was related to higher blood pressure in Black men who suppressed their anger. Finally, Dorr et al. (2007) found that Black, as compared to White participants, who expressed their anger recovered from the stress task more slowly.

Given that anger expression is especially influential among Black individuals, it may be a factor in the impact of racism on health. Since the magnitude of the response to discrimination varies as a function of individual differences and anger is a common feeling associated with racism, it is likely that anger expression style interacts with perceived racism to influence cardiovascular functioning. Most research however has examined the influence of perceived racism, anger, and anger expression independently. In the only study to date that has looked at anger expression together with racism, Steffen et al. (2003) found that perceived racism was positively related to anger inhibition but not to outwardly expressed anger in Black and White participants. They also found that greater perceived racism predicted higher daytime ambulatory blood pressure and those who reported holding their anger in had increased nighttime ambulatory blood pressure. Anger inhibition, however, did not account for the relationship between discrimination and blood pressure. In another study, Clark (2006a) reported that trait anger and perceived racism did not independently predict resting blood pressure in Black adolescents but that trait anger mediated the relationship between racism and blood pressure. Racism was inversely related to blood pressure, only among those who were low in trait anger. Conversely, Clark (2000) reported that perceived racism predicted diastolic blood pressure changes to a speech stressor, but that this relationship was not mitigated by anger in Black women.

Research is only beginning to examine the role of anger expression in the relation between racism and blood pressure functioning. A consistent pattern of findings has not emerged from the handful of studies having considered these variables together. More research examining the effects of anger expression and its influence on perceived racism is necessary to further understand this complex relationship on cardiovascular functioning. The next step would be to examine both anger-in and anger-out on hemodynamic changes to stress in the laboratory.

Purpose

The goal of the present work is to further understand the role of racial discrimination in the increased risk of hypertension in Blacks. Three studies were conducted to address important gaps in the literature. First, a systematic review was accomplished to quantitatively examine the association between racism and hypertension. Specifically, a meta-analysis was conducted to ascertain the size and direction of the effect between perceived discrimination and blood pressure functioning. Further, the roles of individual-level and methodological factors in moderating the associations were evaluated. Second, a study was conducted to examine racial differences in cardiovascular reactivity to acute stress in Black and White Canadians given that proposed racial differences have nearly exclusively been tested with U.S. participants. This was performed to evaluate the generalizability of U.S. data to other developed countries and to further evaluate racial patterns of reactivity. A last study was conducted to estimate the direct and interactive effects of anger expression and perceived racism in predicting cardiovascular reactivity to stress. This last study is among the few in the area of anger and racism, as they relate to physiological functioning. Together, this series of work will further the understanding of this complex research area and ultimately help in better identifying the vulnerability and resilience factors associated with the impact of the stress of racism.

Study One:

Perceived Racial Discrimination and Hypertension: A Comprehensive Systematic Review

Dolezsar, C., McGrath, J., Herzig, A., & Miller, S. (2014). Perceived Racial Discrimination and Hypertension: A Comprehensive Systematic Review. *Health Psychology, 33*, 1, 20-34. doi: 10.1037/a0033718.

Abstract

Objective: Discrimination is posited to underlie racial disparities in hypertension. Extant literature suggests a possible association between racial discrimination and blood pressure, although inconsistent findings have been reported. The aim of this comprehensive systematic review was to quantitatively evaluate the association between perceived racial discrimination with hypertensive status and systolic, diastolic, and ambulatory blood pressure.

Method: Electronic database search of PubMed and PsycINFO (keywords: blood pressure/hypertension /diastolic/systolic, racism/discrimination/prejudice/unfair treatment) was combined with descendancy and ascendancy approaches. Forty-four articles (N = 32,651) met inclusion criteria. Articles were coded for demographics, hypertensive diagnosis, blood pressure measurement, discrimination measure and constructs, study quality, and effect sizes.

Results: Random effects meta-analytic models were tested based on *Fisher's Z,* the derived common effect size metric. Overall, perceived racial discrimination was associated with hypertensive status, $Z_{hypertension}$=0.048, *95% CI* [.013, .087], but not with resting blood pressure, $Z_{systolic}$=0.011, *95% CI* [-.006, .031], $Z_{diastolic}$=.016, *95% CI* [-.006, .034]. Moderators that strengthened the relation included sex (male), race (Black), age (older), education (lower), and hypertensive status. Perceived discrimination was most strongly associated with nighttime ambulatory blood pressure, especially among Blacks.

Conclusion: Despite methodological limitations in the existing literature, there was a small, significant association between perceived discrimination and hypertension. Future studies should consider ambulatory nighttime blood pressure, which may more accurately capture daily variation attributable to experienced racial discrimination. Perceived discrimination may partly explain racial health disparities.

Perceived Racial Discrimination and Hypertension: A Comprehensive Systematic Review

Hypertension is markedly more prevalent among Blacks than Whites (42% vs. 28.8%), with racial health disparities documented for decades (e.g., Burt et al., 1995; CDC, 2011). Among Blacks, hypertension has an earlier age of onset, with greater severity, and is associated with greater pressure related target-organ damage and higher cause-attributable mortality, compared to Whites (AHA, 2005; Ferdinand & Armani, 2007). Blacks typically present with higher risk profiles, including obesity, physical inactivity, cigarette smoking, excess sodium, low socioeconomic status, limited access to quality health care, family history of cardiovascular disease, and stress (Crimmins, Kim, Alley, Karlamangla, & Seeman, 2007). However, these risk factors alone do not adequately account for observed racial differences (Cornoni-Huntley, LaCroix, & Havlik, 1989). Racial discrepancies persist even after controlling for socioeconomic status (Hayward, Crimmins, Miles, & Yang, 2000). Moreover, researchers have failed to demonstrate any risk factors that are biologically unique to Blacks, despite extensive focus on genetics (Hertz, Unger, Cornell, & Saunders, 2005). In fact, the Black-White disparity in hypertension is less consistent when considered internationally (Cooper et al., 2005), which further challenges the role of biological mechanisms as a plausible explanation. Together, these findings have led researchers to consider other psychosocial and environmental factors that may explain the observed hypertension disparities, namely racial discrimination and racial segregation.

Shifting from biological explanations, researchers have increasingly focused on social aspects of racism, including discrimination and segregation, that may better account for the racial disparities in hypertension observed among industrialized countries (Brondolo, Brady ver Halen, Pencille, Beatty, & Contrada, 2009; Goodman, 2000; Mujahid, Roux, Cooper, Shea, & Williams, 2011; White et al., 2011). Racial discrimination leads to inequitable access to social, educational, and material resources that have both direct and indirect effects on health status (Adler & Rehkopf, 2008; Adler & Snibbe, 2003; Gallo & Matthews, 2003). Racial discrimination has been associated with a range of poorer health outcomes including respiratory conditions, diabetes (Karlsen & Nazroo, 2002), somatic complaints (Bowen-Reid & Harrell, 2002), and chronic health conditions (Finch, Kolody, & Vega, 2000; Gee, Spencer, Chen, & Takeuchi, 2007). Perceived discrimination has been posited to be an underlying determinant of racial disparities in hypertension.

The predominant theoretical explanation linking perceived discrimination and hypertension is conceptually grounded in the cardiovascular reactivity hypothesis (Krantz & Manuck, 1984). Perceptions of racial discrimination are stressful life experiences (Clark, Anderson, Clark, & Williams, 1999) that create emotional distress (Landrine, Klonoff, Corral, Fernandez, & Roesch, 2006) and serve as a potent psychosocial stressor (Brondolo, Gallo, & Myers, 2009). Studies that experimentally simulate exposure to racism (e.g., films depicting racism; harassment by confederates) have found that racist stimuli provoke greater reactivity among Blacks, than non-racist stimuli (Fang & Myers, 2001; Guyll, Matthews, & Bromberger, 2001; McNeilly et al., 1995; Sutherland & Harrell, 1986). Blacks who report past experiences with racism also exhibit exaggerated blood pressure reactivity (Clark, 2000). These findings largely support the tenet of reactivity linking racism as a stressor with heightened blood pressure.

Previous literature reviews suggest a plausible relation between discrimination and health. While several reviews discuss the relation broadly across multiple health outcomes (e.g., physical health; Pascoe & Smart Richman, 2009, Williams & Mohammed, 2009), Brondolo and colleagues focus exclusively on blood pressure and hypertension. In 2003, Brondolo, Rieppi, Kelly, and Gerin found the extant literature was "mixed and inconsistent" and elucidated methodological limitations that obscure the relation between perceived discrimination and hypertension. More recently, Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe (2011) described the relation as "weak" and suggested findings are more consistent with ambulatory blood pressure and institutional forms of racism. Others have concluded the association is "complex and unclear" (Williams & Mohammed, 2009) or alluded to such (Paradies, 2006). However, these conclusions are predominantly based on pooled findings of hypertensive status and blood pressure measurement. Drawbacks of qualitative reviews include the focus on statistical significance and use of "vote-count" methods where an entire study is categorized as having a positive, negative, or null relation; rarely is the *magnitude* of the association considered nor is the contribution of the *moderator* quantified. Quantitative reviews, or meta-analyses, aim to address these shortcomings by examining the pattern (direction and magnitude) of evidence across studies to derive a cumulative effect, the consistency of effect sizes, and the contribution of moderators. Some previous reviews discussed moderators in the relation between perceived discrimination and hypertension, including coping style or social support (Harrell, Hall, & Taliaferro, 2003; Pascoe & Smart Richman, 2009); discrimination time-frame, type, number of

items, or psychometric quality (Paradies, 2006; Pascoe & Smart Richman, 2009); stress (Williams & Mohammed, 2009); obesity and smoking (Brondolo et al, 2011); and, socioeconomic status (Brondolo et al, 2003; 2011). However, the extent to which these moderators affect the relation between study features and effect sizes is unknown. In the only quantitative review, Pascoe and Smart Richman (2009) conducted a meta-analysis of perceived discrimination and multiple health outcomes. While blood pressure was included within the 36 physical health outcome studies, only 12 had blood pressure outcomes, including two that reported redundant results; thus, the review did not systematically include relevant findings from several other blood pressure and hypertension studies. Taken together, a comprehensive, quantitative review of current research remains to be completed.

The aim of the present systematic review was to quantitatively examine the association between racism and hypertension. Specifically, a meta-analysis was conducted to ascertain the size and direction of the effect between perceived discrimination and hypertensive status as well as resting systolic and diastolic blood pressure. Further, the roles of individual-level (e.g., age, sex, socioeconomic status, social support, body mass index, smoking status) and methodological factors (e.g., perceived discrimination measure, hypertensive diagnosis, and blood pressure assessment) in moderating the associations were evaluated.

Method

Selection of Studies

Studies were identified through literatures searches of PsycInfo and PubMed electronic databases for the years 1970 to March 2012 (see Figure 1) using keywords (*blood pressure*/*hypertension*/*diastolic* /*systolic* AND *racism/discrimination/prejudice/unfair treatment)*. For the 248 initially identified studies, all titles and abstracts were reviewed; the level of agreement between two independent coders was high ($\kappa = .91$); in cases of disagreement, the article was retained for further consideration. Next, ascendancy and descendancy approaches were used to identify additional articles. Finally, letters of solicitation were sent to authors who published two or more articles on the topic, requesting possible data from unpublished manuscripts, including non-significant findings. Researchers' suggestions did not pertain to any non-redundant data. A total of 145 potentially relevant studies were identified for full review.

Upon completed review, 44 articles were included in the meta-analysis. (See Figure 1 for literature search strategy.)

Article Coding

Sample size, demographic information (e.g., age, sex, income, education, marital status), physical health characteristics (e.g., smoking, BMI), methodology (e.g., recruitment, inclusion/exclusion criteria), blood pressure measurement, hypertensive status definition, discrimination measure, study quality, outcome measures, and effect sizes were coded by a single rater (CD). For each study, approximately fifty coding decisions were made. To ensure there was no coding drift, a random sample (10%) of the initially coded studies was blindly recoded after an 18 mos delay; intra-rater agreement was excellent, *ICC* = .99, *95% CI* [.996, .999]. A second coder (AH) independently coded another random sample (10%); inter-rater agreement was excellent, *ICC* = .985, *95% CI* [.956, .995].

Perceived Discrimination

Measure. Three standardized instruments are commonly used to assess discrimination. Perceived Racism Scale (McNeilly et al., 1996) measures interpersonal and institutional racism during the past year and lifetime. Everyday Discrimination Scale (Williams, Yu, Jackson, & Anderson, 1997) measures everyday experiences of interpersonal discrimination and includes an item about the nature of the discrimination. Experiences of Discrimination (Krieger & Sidney 1996; Krieger, 1990) measures interpersonal and institutional discrimination across seven domains (e.g., school, work, obtaining housing). “Other Standardized Measures” included less frequently used standardized measures including Racism and Life Experiences Scale (Harrell, 1997; Harrell, Merchant, & Young, 1997), Perceived Ethnic Discrimination Questionnaire-Community Version (Brondolo et al. 2005), Oppression Questionnaire (Victoroff, 2005), and Nadanolitization Scale (Taylor & Grundy, 1999); these were grouped for comparison purposes. Adaptations of these measures (i.e., reworded questions, changed response options) were coded with original measure. (See Bond et al., 2007 for psychometric properties of standardized discrimination measures.) Finally, “Researcher-Defined Questions” was coded for singular or limited questions (e.g., “Have you ever felt badly treated because of your race?”).

Constructs. Item-analysis of each study's discrimination measure, to accurately capture

nuanced modifications and adaptations in wording, was used to code three underlying constructs: nature, type, and time-frame. Nature of the discrimination measure was coded as either "Racism" (items specifically racially-oriented) or "Discrimination (non-specific)" (items more generally about discrimination, not directly attributable to race). Type was coded as either "Interpersonal" (items pertained to interactions between individuals) or "Institutional-setting" (items specifically referred to place or setting; e.g., hospital, workplace). Time-frame was coded either as "Past year" or "Lifetime".

Hypertension

Hypertensive Status. Hypertensive status was coded "Physician diagnosis/Medication" if diagnosis was confirmed by medical chart review, or participants reported a physician diagnosis or use of prescribed hypertension medications. Self-report of physician diagnosis has been found to be reliable against medical records (Alonso, Beunza, Delgado-Rodríguez, & Martínez-González, 2005). Alternatively, "Measured Blood Pressure" was coded if hypertension was defined as measured values exceeding 140/90 mmHg on a single clinic visit (no study reported taking readings on more than one occasion).

Resting Blood Pressure. Blood pressure readings taken in a laboratory or clinic, as well as daytime only ambulatory recordings, were coded for resting blood pressure. Number of readings and assessment method (oscillometric vs. ausculatory) were also coded.

Ambulatory Blood Pressure. For studies reporting ambulatory data, nighttime only, 24 hour, and nighttime dipping blood pressure were coded. Dipping refers to the reduction in blood pressure at night compared with daytime blood pressure. These effect sizes were treated separately and not included in the resting blood pressure analyses.

Effect Size Calculation

Effect size calculations were guided by previously reported procedures (Cooper & Hedges, 1994). Fisher's Z was designated as the common effect size metric across studies; values range from $-\infty$ to $+\infty$ and it is interpreted similar to a correlation. Fisher's Z is advantageous as data may be converted from almost any test statistic (Rosenthal, 1991). Bivariate correlations were converted using Fisher's variance stabilizing transformation (Cooper, 1998). Test statistics (e.g., t-test, F-statistic) were converted into r and then into Fisher's Z, using

formulas reported elsewhere (Cooper & Hedges, 1994). Unstandardized beta coefficients were converted to *t*-test statistics; standardized regression coefficients were appropriately weighted (Peterson & Brown, 2005). Zero-order correlations were coded; when partial correlations were coded, the number of covariates was also recorded. Dichotomized outcomes (e.g., Odds Ratio) were transformed into Cohen's *d*, and then converted to Fisher's *Z* (Chinn, 2000; Sánchez-Meca, Marín-Martínez, & Chacón-Moscoso, 2003). When test statistic data were not reported, effect sizes were derived from exact *p* values using distribution tables (Lindley & Scott, 1984). If a comparison was described as "nonsignificant", an effect size of zero was used as a conservative estimate. Finally, if no statistical values were provided and no information was given regarding significance, the comparison was not included in the analysis.

Data Reduction

Effect sizes were coded for all available and relevant data reported within each article, thus yielding multiple effect sizes per study. Of the 44 articles included, there were a total of 167 effect sizes, for an average of 3.80 effect sizes per study. Stochastic dependencies, which can influence effect size estimates and their precision, are attributable to different effect sizes calculated on the same participants using different measures (e.g., hypertension and resting blood pressure), different samples within the same study (e.g., Blacks, Hispanics, Whites), or several studies reported by the same researchers. Thus, two analytical strategies were employed. First, non-redundant effect sizes were parsed from each study as a conservative approach. If identical participants were incorporated in more than one effect size, the average of the redundant effect sizes was used. Second, because the first strategy has been criticized as being overly conservative and not maximizing power by retaining all available data, analyses were also conducted on the aggregate of all coded effect sizes (see Post Hoc Analyses below).

Analytic Strategy

Random-effects meta-analytic models were used to evaluate the association between perceived racial discrimination and hypertension. Random effects models assume the samples are drawn from populations with different effect sizes. The true effect may differ across studies because of different participant characteristics, measurement of discrimination, or outcome variables (blood pressure vs. hypertensive status). Random-effects models are generally regarded

as more accurate than fixed-effects models, which typically yield overly narrow confidence intervals (Schmidt, Oh, & Hayes, 2009).

An analysis of heterogeneity (Q_T) was conducted for each meta-analysis. This heterogeneity statistic is a measure of variation for included effect sizes; non-significant Q_T indicates the variability is less than expected from sampling error. Distributions found to be heterogeneous (significant Q_T) generally warrant additional moderator analyses. Separate analyses were conducted for all *a priori* specified moderator variables, including age, sex, ethnicity, marital status, SES, BMI, smoking status, hypertensive status, discrimination measure and constructs, and blood pressure assessment. Bootstrap methods (1000 samples) were used to produce nonparametric estimates of confidence intervals about each effect size. To address concerns about possible publication bias and the file drawer-problem, Orwin's (1983) fail-safe numbers were calculated to determine the number of non-significant, unpublished, or missing comparisons that would be needed to make the overall effect negligible or not different from zero. Analyses were conducted using MetaWin (Version 2; Rosenberg, Adams, & Gurevitch, 2000).

Results

Study and Participant Characteristics

Of the 44 included studies, there was an average of 742 participants (*SD* = 1,125) per study, permitting an adequately powered test of a small effect size. Participants were young adults (38.0 years), 46.7% married, and 32.8% male (See Table 1). Samples across all studies were 62.2% Black, 13.6% White, 11.8% Hispanic, and 12.4% Other (e.g., Aboriginal, Asian, Indian, Pakistani, Mixed). Samples were largely low SES ($26,889), 15.93% high school education or less, overweight (BMI_{avg} = 27.4), and 14.9% smokers. Few participants were hypertensive (24.2%) or used prescribed hypertensive medication (6.6%). Participants were largely recruited from community settings (72%) throughout the United States: Midwest (20%), South (20%), Northeast (15%), or West (10%; data not shown).

Resting blood pressure was typically measured using the oscillometric method (32.5%) in a lab or clinic (80%) by a trained nurse or research assistant (35%; data not shown). Average resting blood pressure values largely fell within the normal range (121/73 mmHg). Hypertensive

status was typically self-report of physician diagnosis or prescribed medication (65%). Perceived discrimination was assessed using Perceived Racism Scale (15%), Experiences of Discrimination (15%), Everyday Discrimination Scale (20%), Other Standardized Measures (20%) or Researcher-Defined Questions (20%; data not shown).

Hypertensive Status and Perceived Racial Discrimination

Hypertensive status and discrimination were reported in 18 studies with 24 non-redundant effect sizes; see Table 2. The average cumulative effect size indicated a small relation, as greater perceived discrimination was associated with hypertensive status. This cumulative effect size was heterogeneous, warranting further moderator analyses. The observed relation between discrimination and hypertensive status was significant and stronger for effect sizes based on older participants, higher percent males, Blacks only, higher percent Blacks, lower educational attainment, and more hypertensives. Studies using physician diagnosis/medication use, Everyday Discrimination Scale, and non-specific nature measures of discrimination also yielded significant, positive cumulative effect sizes. Notably, about half of the heterogeneity statistics for the above analyses were significant, suggesting additional moderators exist.

Systolic Blood Pressure and Perceived Racial Discrimination

The relation between resting systolic blood pressure and discrimination was reported in 30 studies and yielded 40 non-redundant effect sizes. The average cumulative effect size did not significantly differ from zero; see Table 3. *A priori* moderator analyses revealed that effect sizes based on samples with more smokers and institutional-setting type discrimination measures yielded significant, positive associations between resting blood pressure and discrimination. When ambulatory blood pressure was considered separately (i.e., not included in cumulative analyses), nighttime systolic blood pressure was significantly associated with discrimination; this was the largest effect size observed.

Diastolic Blood Pressure and Perceived Racial Discrimination

The relation between resting diastolic blood pressure and discrimination was reported in 29 studies, with 40 non-redundant effect sizes. The average cumulative effect size did not significantly differ from zero; see Table 4. *A priori* moderator analyses revealed that for effect

sizes based solely on Blacks and institutional-setting type discrimination measures, there were positive, significant relations between resting diastolic blood pressure and discrimination. There was a negative relation for effect sizes calculated solely on Whites. Select discrimination measures also yielded significant effects. When ambulatory blood pressure was considered separately, nighttime diastolic blood pressure and dipping were significantly associated with discrimination; these were the largest effect sizes observed.

Post-Hoc Analyses

Blacks only. Random effects meta-analytic models were re-run for effect sizes derived from Blacks only; see bottom rows of Tables 2 - 4. There was an overall association for hypertensive status and diastolic blood pressure, based on the cumulative effect sizes. For hypertensive status, moderator analyses revealed the relation was stronger in effect sizes based on samples with lower educational attainment, more smokers, physician diagnosis/medication use, Experiences of Discrimination measure, Other Standardized Measures, and past-year discrimination. For resting systolic blood pressure, significant moderators included smoking, Experiences of Discrimination measure, and institutional-setting type discrimination measures; nighttime ambulatory blood pressure was also significant. For resting diastolic blood pressure, significant moderators included more males, Other Standardized Measures, institutional-setting type discrimination measures, and lifetime timeframe discrimination; nighttime ambulatory blood pressure was also significant. All of these Q_T statistics were non-significant, suggesting the cumulative effect sizes were homogenous and do not warrant further parsing. Examination of the fail-safe numbers reveals at least 100 non-significant findings are necessary to reduce the observed significant findings to negligible. Across all meta-analytic models, the strongest associations between hypertension and discrimination were observed for nighttime ambulatory blood pressure among Blacks only.

Smoking and Socioeconomic Status. In addition to testing smoking and education level as moderators at the study sample level, we compared effect sizes that included these as covariates. The overall effect sizes did not differ when socioeconomic status was included as a covariate, for hypertensive status or resting blood pressure, $Z_{\text{covariate}} = .033$, *95% CI* [.014, .068] vs. $Z_{\text{zero\_order}} = .024$, *95% CI* [.002, .051]. When smoking was included as a covariate, resting systolic and diastolic blood pressure were more strongly related to perceived discrimination,

$Z_{covariate}$ = .063, *95% CI* [.053, .072] vs. Z_{zero_order} = .009 *95% CI* [-.007, .027]; however, very few studies controlled for smoking. There was no difference when smoking was included as a covariate for hypertensive status.

Study quality. Seven dimensions of study quality were rated dichotomously and summed to yield a quality rating. Dimensions included: (1) inclusion/exclusion criteria explicitly described (82%); (2) discrimination measure with established psychometric properties (82%); (3) hypertensive status defined by physician diagnosis/prescription medication use or resting blood pressure assessed by trained professional (68%); (4) sample size greater than 100 participants (77%); (5) statistically controlled for covariates in at least one analysis (70%); (6) test statistic estimates presented (80%); and (7) peer-reviewed journal with impact factor greater than 2 (66%). Effect size was not related to study quality, r = .064 p = .490.

Alternate effect size selection. The aforementioned analyses were based on non-redundant effect sizes, with selection preference for zero-order or bivariate correlations. Largely identical results were obtained when all analyses were re-run with (i) non-redundant effect sizes, selection preference for partial correlations controlling for covariates, (ii) redundant effect sizes, including all calculated effect sizes regardless of number contributed per study, and (iii) non-redundant effect sizes, averaged to yield only one effect size per study (results not shown for parsimony).

Discussion

Perceived discrimination has been posited to explain observed racial disparities in hypertension. The aim of this comprehensive systematic review was to quantitatively ascertain the strength of the association between perceived racial discrimination and hypertension. There was a significant, albeit small relation between perceived discrimination and hypertensive status. This relation was stronger among older participants, males, Blacks, lower educational attainment, hypertensives, and physician diagnosis of hypertensive status. The relation was not apparent for resting blood pressure. For resting systolic and diastolic blood pressure, the relation was significant only for institutional-setting type discrimination measures. The largest associations were observed for ambulatory nighttime blood pressure and dipping; this was especially true among Blacks. Overall, there was a small, significant relation between perceived discrimination and hypertensive status, which is more salient among certain subgroups or study

features.

Previous narrative reviews also suggest a small and complex relation between racial discrimination and hypertensive status (Brondolo et al., 2011), as determinants at both the individual- and contextual-level likely moderate this relation. Individual-level differences (e.g., demographics, lifestyle behaviors, social support) have been considered to be important moderators. In the present meta-analysis, age, sex, race, and education moderated at least one association between discrimination and hypertension. With the exception of race, these demographic variables have been underemphasized in prior reviews, with greater attention to other posited moderators. Lifestyle behaviors (e.g., smoking, fitness, alcohol) and weight status (i.e., obesity) have been previously observed as moderators (Brondolo et al., 2011). Smoking was found to moderate the association between perceived discrimination and resting systolic blood pressure only; further, post-hoc analyses revealed that in studies that included smoking as a covariate, discrimination was more strongly related to resting blood pressure. Weight status (i.e., BMI) was not found to moderate any association. It was not possible to systematically consider other health behaviors (e.g., fitness, alcohol) due to infrequent and inconsistent reporting in the literature. Prior reviews have primarily considered coping and social support as moderators (e.g., Harrell et al., 2003; Pascoe & Smart Richman, 2009). The measurement of coping and social support is extremely varied, precluding their inclusion as covariates in the meta-analysis. Marital status, which some consider as a crude proxy of social support, did not moderate the association for any analyses. Finally, contextual-level racial discrimination (e.g., neighbourhood racial segregation) has been previously associated with hypertension and blood pressure (e.g., Kershaw et al., 2011; McGrath, Matthews, & Brady, 2006). Few studies report these findings with measures of perceived discrimination, thus limiting neighbourhood segregation from being considered as a moderator.

Methodological factors may also moderate the relation between perceived discrimination and hypertension, especially the measurement of these constructs themselves. The conceptualization and measurement of perceived discrimination poses a significant challenge in the study of racial disparities (cf., Brondolo, Gallo, & Myers, 2009; Landrine et al., 2006). Perceived discrimination inherently reflects one's lifetime burden of discrimination, the occurrence and frequency of everyday and lifetime discrimination, personal attribution and coping resources, as well as the effect of skin color and acculturation. In the present meta-

analysis, perceived discrimination was coded based on the measures themselves and three underlying constructs (nature, type, time-frame). While the Everyday Discrimination Scale was used most frequently, its results were inconsistent as it was associated with hypertensive status but not resting blood pressure. For the nature of discrimination, non-specific measures were significantly associated with hypertensive status, yet, the magnitude of the effect was not meaningfully different than racism measures. For the type of discrimination, institutional-setting measures were significantly associated with both resting blood pressure measures; however, items predominantly inquired about interpersonal discrimination within institutional settings (e.g., "Have you ever been made to feel inferior because of your race or color at work?"), rather than specific policies or procedures of the institution. Finally, time-frame of discrimination appeared to only matter for Blacks. Past-year measures were significantly associated with hypertensive status while lifetime measures were significantly associated with resting diastolic blood pressure. Limited psychometric standards in the measurement of perceived discrimination have been previously criticized (cf., Brondolo et al., 2003; 2011). As well, prior reviews considered how measurement of discrimination may moderate observed findings. Others concluded there is weak evidence linking interpersonal-type discrimination with hypertensive status, and that there is a more consistent relation with ambulatory blood pressure (Brondolo et al., 2011). Institutional-type discrimination has been suggested to be more clearly associated with hypertension incidence in population-based studies using contextual-level indicators of neighborhood segregation (cf., Brondolo et al., 2011). Discrimination time-frame findings are inconsistent, with some suggesting that past year may be more sensitive than lifetime (Paradies, 2006), others reporting no differences across chronic, acute, recent, or lifetime, and others observing an association among Blacks alone for chronic, but not acute discrimination (Pascoe & Smart Richman, 2009); however, these findings are limited to general or physical health outcomes. Finally, discrimination measures with more items have been suggested to yield stronger association with health generally (Paradies, 2006).

Methodological differences in the diagnosis of hypertensive status and measurement of blood pressure also moderated current findings. Discrimination was significantly and more strongly related to hypertensive status that was defined by physician diagnosis or prescription medication, including self-report, compared to clinic or laboratory measured blood pressure values exceeding 140/90 mmHg. According to the JNC Task Force Guidelines (Chobanian et

al., 2003; Pickering et al., 2005), having only one to two blood pressure readings on a single day is insufficient to diagnosis hypertension. Physician diagnosis or use of prescribed blood pressure medications are more valid indicators of actual hypertensive status. Most studies use self-report of diagnosis or medication use; and, there is evidence that self-report is accurate against medical record (Alonso et al., 2005). Prior reviews have typically drawn conclusions from the combined results of studies with physician diagnosis, self-report of diagnosis, and measured blood pressure, as a "proxy for documented diagnosis" (e.g., Brondolo et al., 2003; 2011; Paradies, 2006; Williams & Mohammed, 2009). Resting blood pressure was not associated with perceived discrimination; however, moderators revealed significant associations within subgroups. Consistent with observations of prior reviews (e.g., Brondolo et al., 2011), ambulatory nighttime systolic and diastolic blood pressure were associated with perceived discrimination. Ambulatory dipping was also associated for diastolic blood pressure, and although not significant, a similar magnitude effect was observed for systolic blood pressure. Further, these effect sizes were the largest observed across all meta-analysis associations, accounting for 14% of the variance in the association between perceived discrimination and hypertension. There are plausible reasons why ambulatory measures may yield more accurate measures of blood pressure than clinic based assessments. First, ambulatory measures are thought to be more ecologically valid as they capture daily fluctuations of blood pressure in one's natural living environment. Second, ambulatory measures are more stable from a measurement perspective as they capture longer time intervals (e.g., 12 to 24 hours), thus reducing measurement error. Ambulatory studies have the potential to yield important insight into contextual-level determinants, including neighborhood segregation.

There are current limitations in the extant literature that constrain our comprehensive understanding of the association between perceived discrimination and hypertension. First, sample participants were young, with an average age of 38.25 years. Hypertension prevalence increases with age (e.g., <40 years, 7%; 60+ years, 65%; Ong, Cheung, Man, Lau, & Lam, 2007). The association between perceived discrimination and hypertension may be less apparent given the young age of participants in most included studies. However, blood pressure levels track over the lifecourse and the largest effects were observed for hypertensive status rather than resting blood pressure. Second, participants were most typically women (67.25%). Men are more likely to have uncontrolled hypertension as well as greater end-organ damage and mortality than

women (AHA, 2005). The strength of the association between perceived discrimination and hypertension was greater in studies with higher percentages of men, which suggests this association may be even more pronounced within men. Third, the findings are based on self-report of perceived racial discrimination. Several additional individual- and contextual-level factors may explain the observed racial disparities in hypertension, including identification with one's racial group, level of interacting with members of the racial majority group, perceived group discrimination, coping resources, and neighborhood racial segregation.

The cumulative burden of social and environmental disparities among races has direct implications for racial health disparities (Hicken et al., 2011). Hypertension is an established risk factor for other cardiovascular diseases and mortality (O'Donnell et al., 1997). Mortality attributable to hypertension accounts for the largest racial disparity in potential life-years lost (15%), over HIV, diabetes, and homicide (Wong, Shapiro, Boscardin, & Ettner, 2002). Addressing racial disparities in hypertension would contribute greatly to lowering mortality among Blacks. The challenge in developing effective public policy is targeting the intersection of social, behavioural, and environmental determinants that underlie health disparities.

In conclusion, despite methodological limitations, there is a small, significant association between perceived racial discrimination and hypertensive status. Perceived discrimination was most strongly associated with nighttime ambulatory blood pressure, especially among Blacks, which may more accurately capture blood pressure variation in response to racial discrimination. Future research should further examine nighttime blood pressure and contextual-level indicators of institutional-setting discrimination (e.g., neighbourhood racial segregation, social cohesion) as possible moderators of the association between perceived racial discrimination and hypertension. Taken together, perceived discrimination may partly explain racial health disparities.

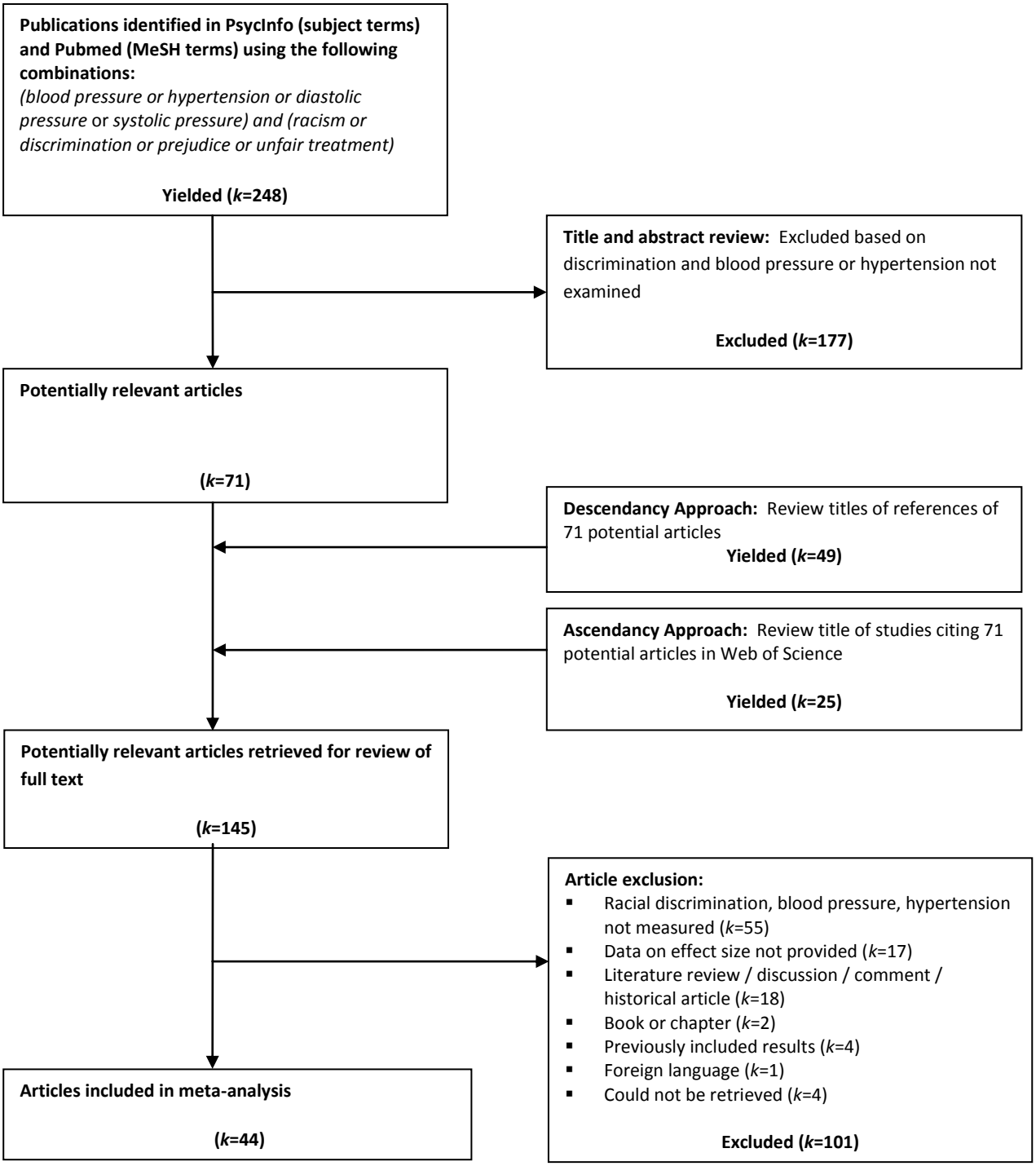


Figure 1. Flow Chart for Article Identification and Inclusion.

Table 1

Descriptive Characteristics and Frequencies of 44 Studies Included

Characteristic	*K*	*N*	Minimum	Maximum	*M (SD)*
Age (years)	41	28,761	9.5	74.1	38.25 (15.22)
Sex (% male)	43	30,144	0.0	100.0	32.75% (20.05)
Ethnicity (% Black)	44	32,651	0.0	100.0	62.61% (39.86)
Marital Status (% married)	14	12,725	12.0	100.0	46.69% (23.12)
SES					
Household income ($)	12	3,886	7,000.0	43,942.0	26,889 (13,094)
Education (% HS degree or less)	28	23,922	0.0	58.0	15.93% (18.31)
BMI kg/m^2	27	18,927	22.4	30.7	27.41 (2.23)
Smoking (% smokers)	16	8,898	0.0	48.6	14.85% (12.83)
Hypertensive status					
Hypertensive (% diagnosed)	32	22,414	0.0	71.0	24.21% (24.21)
Prescribed medication (% taking)	22	16,346	0.0	44.4	6.60% (11.82)
Blood pressure					
SBP resting mmHg	27	19,754	103.6	135.3	120.71 (9.60)
DBP resting mmHg	27	19,754	59.0	82.9	73.05 (7.38)
No. laboratory BP readings	22	18,415	2.0	9.0	2.91 (1.60)

Note. k= number of studies reporting this information; *N* = total number of participants; *M* = Mean; *SD* = standard deviation; HS = high school.

Table 2

Hypertensive Status and Discrimination (Random Effects Models)

Comparison	Effect sizes	*N*	*Fisher Z*	Bootstrap *95% CI*	Q_T	Fail-safe *n*
All studies	24	18,987	**.048**	**(.013, .087)**	40.413*	1132
Moderators						
Age	20	15,097	**.053**	**(.009, .096)**	34.991*	1032
Sex (% male)	23	16,480	**.044**	**(.008, .085)**	38.004*	978
Ethnicity						
Black only	13	6,312	**.045**	**(.002, .102)**	18.605	578
Hispanic only	2	1,404	-.033	(-.094, .000)	1.000	0
White only	-	-	-	-	-	-
% Black	24	18,987	**.048**	**(.012, .087)**	40.205*	1133
Marital status (% married)	10	8,220	.026	(-.041, .084)	11.681	249
SES						
Household income	4	1,892	-.018	(-.078, .049)	2.328	0
Education (% HS degree or less)	21	16,325	**.051**	**(.012, .093)**	33.677*	1041
BMI	9	7,643	.032	(-.018, .074)	13.107	278
Smoking (% smokers)	12	7,259	.053	(-.014, .130)	20.337*	624
Hypertensive status						
% hypertensive	22	16,398	**.051**	**(.009, .095)**	41.085	1100
% taking medication	6	5,317	.024	(-.055, .105)	7.286	138
HTN assessment						
Physician diagnosis/medication (self-report)	15	10,727	**.072**	**(.025, .118)**	23.729*	1076
Measured blood pressure (>140/90 mmHg)	9	8,260	.014	(-.036, .059)	12.984	119
Discrimination assessment						
Discrimination measure						
Perceived Racism Scale	-	-	-	-	-	-
Experiences of Discrimination	8	2,365	.047	(-.002, .090)	9.706	369
Everyday Discrimination Scale	7	8,170	**.060**	**(.020, .100)**	6.825	416
Other standardized measure	6	4,467	.045	(-.013, .125)	7.017	266
Researcher-defined questions	3	3,985	.034	(-.034, .119)	1.734	98
Nature of discrimination						
Racism	15	9,086	.053	(-.007, .120)	25.604*	786
Discrimination (non-specific)	9	9,901	**.049**	**(.007, .087)**	10.866	433
Discrimination type						
Interpersonal (non-specific)	11	10,315	.041	(-.003, .083)	13.809	439
Institutional setting	3	1,496	.055	(.000, .250)	2.904	164
Discrimination time-frame						
Lifetime	16	9,796	.041	(-.011, .093)	27.100*	639
Past year	5	6,549	.054	(-.009, .096)	5.445	266
Blacks only						
Moderators						
Education (% HS degree or less)	10	3,650	**.050**	**(.055, .117)**	11.635	491
Smoking (% smokers)	5	1,329	**.114**	**(.044, .229)**	4.410	565
Physician diagnosis/medication (self-report)	7	4,016	**.075**	**(.032, .155)**	8.839	516
Experiences of Discrimination	3	670	**.128**	**(.016, .360)**	2.370	380
Other standardized measure	3	1,043	**.096**	**(.008, .250)**	2.103	286
Past year	3	1,294	**.064**	**(.016, .087)**	0.868	190

Note. Effect sizes = Number of non-redundant effect sizes, *N* = total number of participants; Q_T = heterogeneity test statistic; Fail-safe *n* using Orwin's method; "-" = not applicable due to limited number of effect sizes; HS = high school; *$p < .05$; significant effects are bolded.

Table 3

Resting Systolic Blood Pressure and Discrimination (Random Effects Models)

Comparison	Effect sizes	*N*	*Fisher Z*	Bootstrap *95% CI*	Q_T	Fail-safe *n*
All studies	40	19,449	.011	(-.006, .031)	46.001	407
Moderators						
Age	38	17,812	.007	(-.011, .028)	43.670	244
Sex (% male)	40	19,449	.011	(-.006, .032)	45.474	416
Ethnicity						
Black only	20	5,139	.023	(-.005, .049)	19.589	444
Hispanic only	7	1,922	.029	(-.016, .090)	7.304	197
White only	4	1,991	-.012	(-.019, .052)	1.945	0
% Black	40	19,449	.012	(-.007, .031)	45.263	420
Marital status (% married)	11	8,971	.007	(-.024, .037)	14.608	64
SES						
Household income	11	3,431	.006	(-.029, .032)	4.324	51
Education (% HS degree or less)	23	16,005	.007	(-.012, .033)	30.145	144
BMI	28	14,945	.007	(-.012, .030)	33.159	130
Smoking (% smokers)	15	7,715	**.012**	**(.002, .038)**	9.938	160
Hypertensive status						
% hypertensive	30	11,953	.007	(-.005, .023)	26.433	190
% taking medication	31	15,738	-.005	(-.015, .014)	28.903	0
Clinic/laboratory/school	35	18,727	.013	(-.004, .036)	40.549	406
No. of readings	28	15,662	.012	(-.006, .038)	28.172	270
Ambulatory BP						
Daytime	6	791	.034	(-.054, .172)	5.586	200
Nighttime†	5	579	**.153**	**(.038, .193)**	2.275	761
24 hours†	4	561	.043	(-.096, .082)	2.900	167
Dipping†	3	517	-.129	(-.244, .020)	2.195	0
Discrimination assessment						
Discrimination measure						
Perceived Racism Scale	6	584	-.007	(-.067, .109)	5.107	0
Experiences of Discrimination	5	2,716	.029	(.000, .044)	1.564	139
Everyday Discrimination Scale	16	12,683	-.004	(-.018, .016)	15.261	0
Other standardized measure	8	1,493	.034	(-.018, .106)	7.656	266
Researcher-defined questions	6	2,136	-.005	(-.117, .094)	9.382	0
Nature of discrimination						
Racism	29	7,268	.017	(-.013, .047)	33.911	471
Discrimination (non-specific)	11	12,181	-.006	(-.019, .022)	10.134	0
Discrimination type						
Interpersonal (non-specific)	19	13,170	.001	(-.015, .025)	20.605	0
Institutional setting	4	2,871	**.046**	**(.040, .060)**	0.275	178
Discrimination time-frame						
Lifetime	30	14,671	.007	(-.010, .033)	32.452	190
Past year	9	4,504	.012	(-.032, .085)	9.482	140
Blacks only						
Moderators						
Smoking (% smokers)	7	1,138	**.021**	**(.001, .121)**	5.013	137
Nighttime ABP	3	161	**.171**	**(.070, .245)**	0.759	511
Experiences of Discrimination	3	2,103	**.037**	**(.000, .056)**	0.949	107
Institutional setting	3	1,749	**.050**	**(.048, .080)**	0.196	147

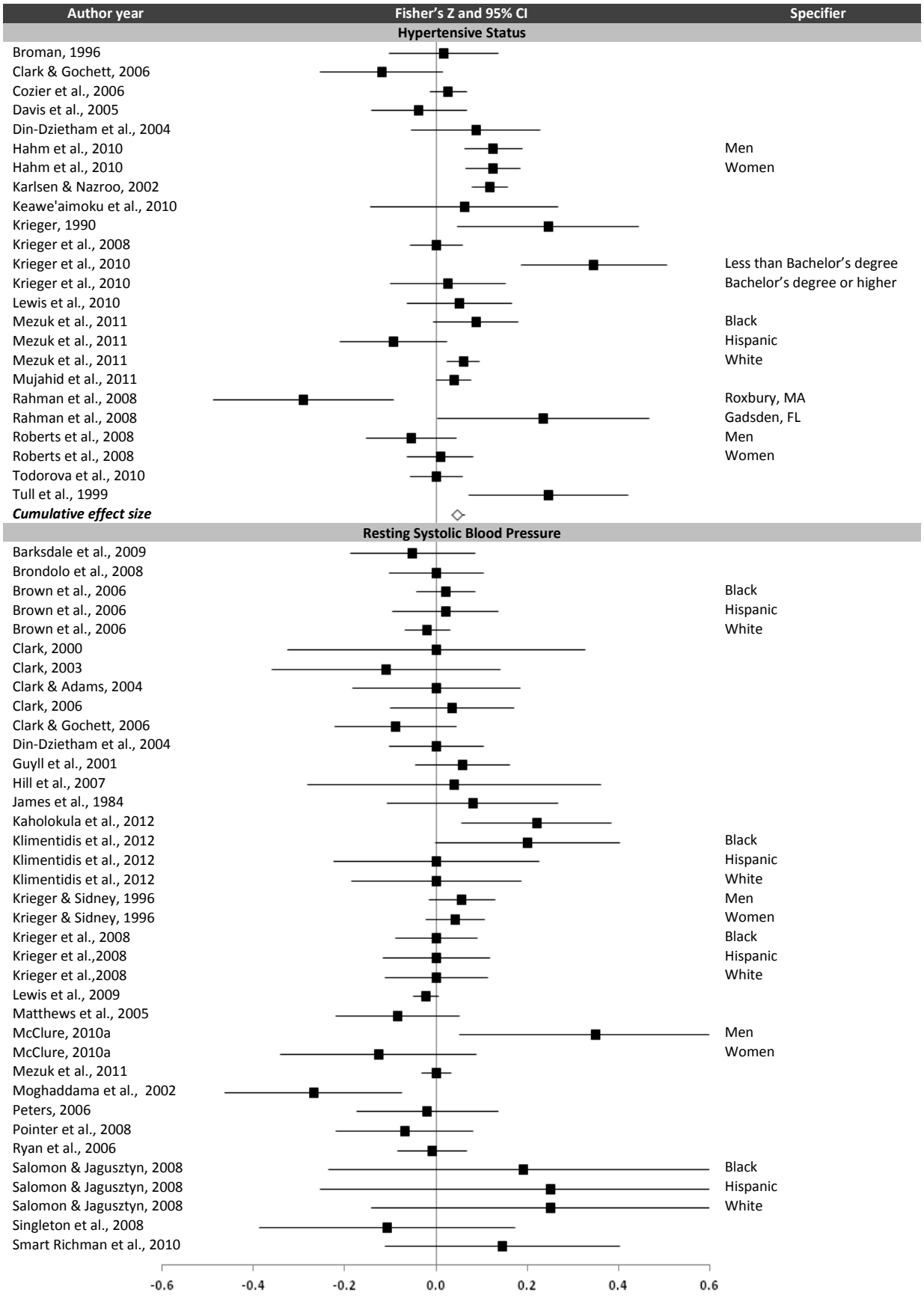
Note. Effect sizes = Number of non-redundant effect sizes, *N* = total number of participants; Q_T = heterogeneity test statistic; Fail-safe *n* using Orwin's method; HS = high school; **p* < .05; significant effects are bolded; † = not included in resting blood pressure analyses.

Table 4

Resting Diastolic Blood Pressure and Discrimination (Random Effects Models)

Comparison	Effect sizes	*N*	*Fisher Z*	Bootstrap *95% CI*	Q_T	Fail-safe *n*
All studies	40	15,602	.016	(-.006, .034)	37.923	597
Moderators						
Age	38	13,965	.013	(-.011, .034)	38.014	435
Sex (% male)	40	15,602	.016	(-.005, .034)	37.114	587
Ethnicity						
Black only	21	7,965	**.039**	**(.006, .056)**	19.118	803
Hispanic only	7	1,869	.056	(-.006, .076)	2.270	387
White only	5	3,859	**-.029**	**(-.063, -.011)**	4.481	0
% Black	40	15,602	.017	(-.005, .037)	39.912	622
Marital status (% married)	10	5,177	.011	(-.031, .049)	13.212	99
SES						
Household income	11	3,378	.019	(-.031, .057)	9.640	199
Education (% HS degree or less)	23	12,158	.018	(-.007, .043)	22.221	395
BMI	28	11,098	.015	(-.010, .047)	22.858	390
Smoking (% smokers)	14	3,921	.033	(-.004, .062)	10.714	447
Hypertensive status						
% hypertensive	29	8,159	.012	(-.014, .041)	30.570	313
% taking medication	30	11,944	.009	(-.014, .032)	28.125	237
Clinic/laboratory/school	33	3,200	.012	(-.013, .036)	31.623	377
No. of readings	27	11,744	.019	(-.010, .047)	25.379	481
Ambulatory BP						
Daytime	6	791	.057	(-.034, .199)	5.727	337
Nighttime†	5	579	**.138**	**(.091, .237)**	2.574	687
24 hours†	4	561	.031	(-.077, .060)	2.002	119
Dipping†	3	517	**-.139**	**(-.266, -.085)**	2.151	0
Discrimination assessment						
Discrimination measure						
Perceived Racism Scale	6	584	-.009	(-.088, .121)	5.292	0
Experiences of Discrimination	6	2,878	**.030**	**(.005, .046)**	1.618	176
Everyday Discrimination Scale	16	8,888	.010	(-.021, .039)	13.166	138
Other standardized measure	7	1,331	**.034**	**(.004, .082)**	3.275	230
Researcher-defined questions	6	2,083	-.007	(-.115, .057)	5.554	0
Nature of discrimination						
Racism	29	7,215	.021	(-.006, .046)	27.944	590
Discrimination (non-specific)	11	8,387	.011	(-.029, .043)	10.429	105
Discrimination type						
Interpersonal (non-specific)	19	9,323	.009	(-.021, .039)	14.875	160
Institutional setting	4	2,871	**.060**	**(.045, .077)**	0.693	236
Discrimination time-frame						
Lifetime	31	14,671	.019	(-.005, .039)	29.472	565
Past year	8	657	-.070	(-.131, .002)	4.408	0
Blacks only						
Moderators						
Sex (% male)	21	7,965	**.039**	**(.008, .056)**	18.956	788
Nighttime ABP	3	161	**.227**	**(.179, .255)**	0.172	679
Other standardized measure	5	828	**.032**	**(.003, .087)**	1.522	152
Institutional setting	3	1,749	**.048**	**(.045, .051)**	0.018	140
Lifetime	14	7,305	**.041**	**(.007, .058)**	13.000	555

Note. Effect sizes = Number of non-redundant effect sizes, N = total number of participants; Q_T = heterogeneity test statistic; Fail-safe *n* using Orwin's method; HS = high school; *$p < .05$; significant effects are bolded; † = not included in resting blood pressure analyses.


Author year
Fisher's Z and 95% CI
Specifier
Hypertensive Status
Broman, 1996
Clark & Gochett, 2006
Cozier et al., 2006
Davis et al., 2005
Din-Dzietham et al., 2004
Hahm et al., 2010
Men
Hahm et al., 2010
Women
Karlsen & Nazroo, 2002
Keawe'aimoku et al., 2010
Krieger, 1990
Krieger et al., 2008
Krieger et al., 2010
Less than Bachelor's degree
Krieger et al., 2010
Bachelor's degree or higher
Lewis et al., 2010
Mezuk et al., 2011
Black
Mezuk et al., 2011
Hispanic
Mezuk et al., 2011
White
Mujahid et al., 2011
Rahman et al., 2008
Roxbury, MA
Rahman et al., 2008
Gadsden, FL
Roberts et al., 2008
Men
Roberts et al., 2008
Women
Todorova et al., 2010
Tull et al., 1999
Cumulative effect size
Resting Systolic Blood Pressure
Barksdale et al., 2009
Brondolo et al., 2008
Brown et al., 2006
Black
Brown et al., 2006
Hispanic
Brown et al., 2006
White
Clark, 2000
Clark, 2003
Clark & Adams, 2004
Clark, 2006
Clark & Gochett, 2006
Din-Dzietham et al., 2004
Guyll et al., 2001
Hill et al., 2007
James et al., 1984
Kaholokula et al., 2012
Klimentidis et al., 2012
Black
Klimentidis et al., 2012
Hispanic
Klimentidis et al., 2012
White
Krieger & Sidney, 1996
Men
Krieger & Sidney, 1996
Women
Krieger et al., 2008
Black
Krieger et al.,2008
Hispanic
Krieger et al.,2008
White
Lewis et al., 2009
Matthews et al., 2005
McClure, 2010a
Men
McClure, 2010a
Women
Mezuk et al., 2011
Moghaddama et al., 2002
Peters, 2006
Pointer et al., 2008
Ryan et al., 2006
Salomon & Jagusztyn, 2008
Black
Salomon & Jagusztyn, 2008
Hispanic
Salomon & Jagusztyn, 2008
White
Singleton et al., 2008
Smart Richman et al., 2010
-0.6
-0.4
-0.2
0.0
0.2
0.4
0.6

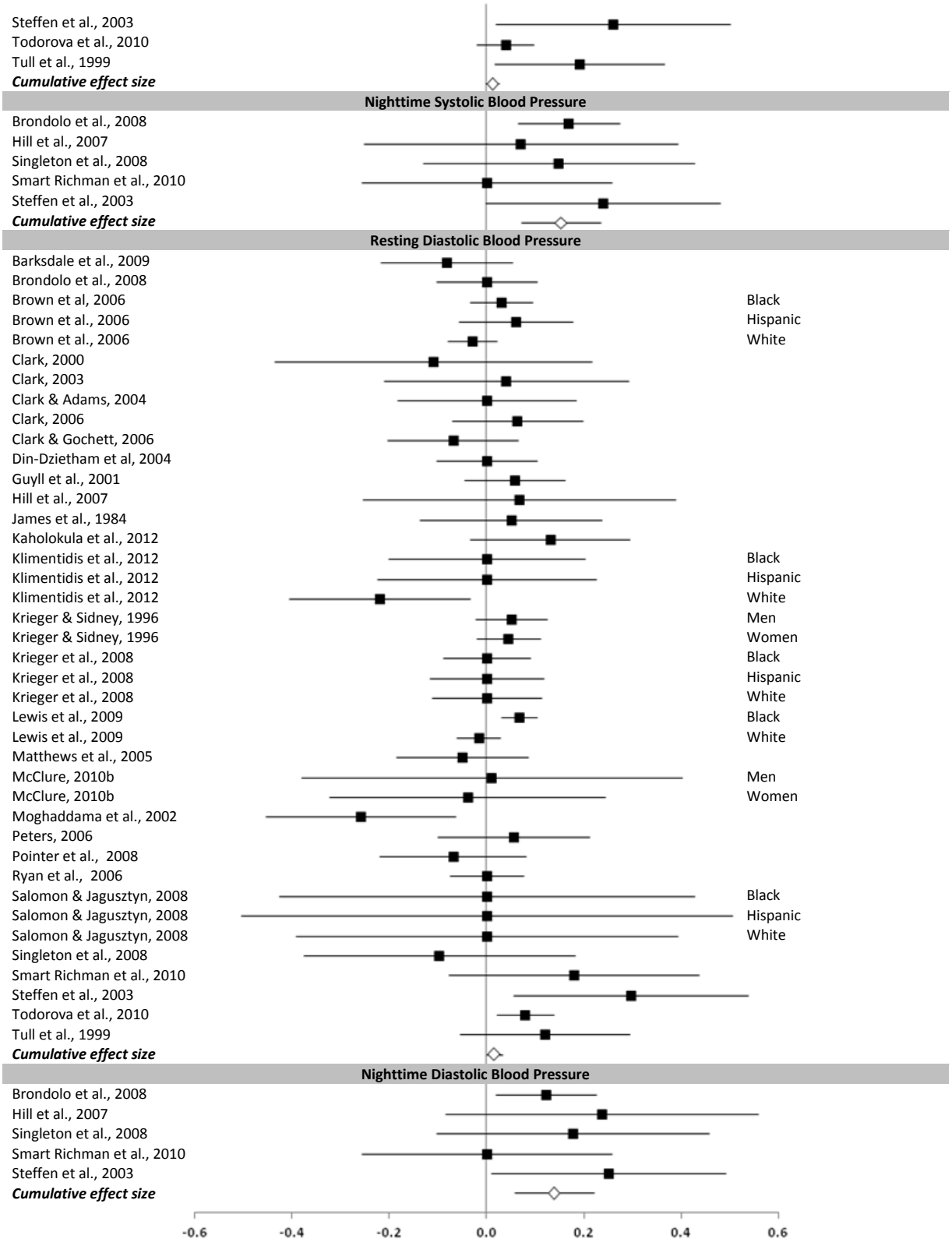


Figure 2. Forest Plot of Effect Sizes for Perceived Racial Discrimination and Hypertension (Hypertensive Status, Resting SBP, Nighttime SBP, Resting DBP, Nighttime DBP)

Study Two:

Racial- and Sex-Related Differences in Cardiovascular Reactivity during Acute Stress in Black and White Canadians

Dolezsar, C.M. & Miller, S.B. Racial- and Sex-Related Differences in Cardiovascular Reactivity during Acute Stress in Black and White Canadians. (under review).

Abstract

Objective: The increased incidence of hypertension in Blacks may in part be explained through an exaggerated vascular response to stress. To date, this model has almost exclusively been tested with American participants. The purpose of this study was therefore to examine racial differences in cardiovascular reactivity to acute stress in Black and White Canadians.

Methods: Cardiovascular responses to three laboratory stress tasks (cold pressor, anger-recall, and mental arithmetic) were examined in healthy Black (n=45) and White (n=49) Canadians (mean age, 23 years).

Results: Black men had greater peripheral vascular responses (diastolic blood pressure, total peripheral resistance, and mean arterial pressure) and diminished myocardial responses (heart rate, and stroke volume) to stress as compared to White men, as well as Black and White women. Black participants overall also had greater forearm vascular resistance that did White participants (ps<.05). Differences were primarily observed during the cold pressor task. Racial differences in reactivity were not observed in women.

Conclusion: Results are consistent with the literature. Previous findings of vascular hyper-reactivity in African-Americans may be extended to Blacks in other countries with different immigration histories such as Canadians. Future research focusing on additional psychosocial factors would be important in understanding the role of discrimination in the cardiovascular response and disease risk of Black individuals.

Racial- and Sex-Related Differences in Cardiovascular Reactivity during Acute Stress in Black and White Canadians

Hypertension is more prevalent, with an earlier age of onset, and associated with greater mortality and morbidity in Black than in White individuals (American Heart Association, 2008; Kurian & Cardarelli, 2007; Lui et al., 2010; Malan et al., 2010). An interaction of genetic factors, physiological differences, environmental stressors, and personality traits has been posited to contribute to racial differences in development of hypertension. The cardiovascular reactivity hypothesis (Krantz & Manuck, 1984) postulates that individuals who show increased cardiovascular responses to stress have a greater risk of developing cardiovascular diseases. Over time, exaggerated cardiovascular activity may to lead to structural changes in arterial walls that may then lead to a sustained increase of vasculature resistance, and thus, high blood pressure (Anderson, McNeilly, & Myers, 1992; Krieger, 1990). Increased cardiovascular responses to stress have been found to be predictive of future elevations in resting blood pressure and hypertension, even when accounting for traditional risk factors (Carroll, 2003; Chida & Steptoe, 2010; Matthews, 2003; 2004; Treiber, 2003).

Racial discrimination and its consequences result in greater exposure to stress among Blacks individuals relative to Whites (Williams et al., 2008; Sternthal, Slopen, & Williams, 2011) and has been found to be related to hypertension (Dolezsar, McGrath, Herzig, & Miller, 2014). Furthermore, physiological differences in hemodynamic profile may also put Blacks at a disadvantage. A vascular rather than a myocardial pattern of regulating blood pressure is more frequently observed in Blacks as compared to Whites (Anderson, Lane, Monou, Williams, & Houseworth, 1988; Anderson, Lane, Muranaka, Williams, & Houseworth, 1988b). Vascular patterns may be particularly harmful given the role of increased total peripheral resistance (TPR) in hypertension (Devereux, Savage, Sachs, & Laragh, 1983). Furthermore, research has linked elevations in TPR at rest and during stress with markers of disease risk (Goldberg et al., 1996; Sherwood, Johnson, Blumenthal, & Hinderliter, 1999; Treiber et al., 1993). Thus, differential exposure to stress, alongside divergent mechanisms of blood pressure regulation are likely contributors to well-documented Black–White disparities in hypertension.

Research examining exposure to stress in Black versus White men has demonstrated an exaggerated cardiovascular response in Black men to a variety of laboratory stressors (Saab et

al., 1992; Treiber et al., 1990; Anderson et al., 1988b; Dorr, Brosschot, Sollers, & Thayer, 2007; Reimann et al., 2012), mediated largely through an increase in peripheral vascular resistance (Ray & Monahan, 2002; Stein, Lang, Singh, He, & Wood, 2000). Similarly, a recent review of ethnic differences in the functioning and mechanics of resistance in arteries reported that normotensive Black as compared to White individuals exhibit increased vascular reactivity to sympathetic stimulation, attenuated responses to vasodilators, and a relatively narrow vascular lumen (Taherzadeh, Brewster, van Montfrans, & van Bavel, 2010).

The above mentioned racial differences in reactivity have been more consistently observed in men versus women (Saab et al., 1997; Light, Turner, Hinderliter, & Sherwood, 1993; Kelsey, Patterson, Barnard, & Alpert, 2000). This may be related to the fact that men in general tend to have greater blood pressure increases, while women exhibit greater increases in heart rate (Kajantie & Phillips, 2005). Since racial differences in reactivity are primarily vascular, they may be more likely to be observed in men who are also more likely to demonstrate a vascular pattern of blood pressure regulation.

Taken together, the increased incidence of hypertension in Blacks may in part be explained through an exaggerated vascular response to stress. To date, this model has almost exclusively been tested with American participants. It would be important to replicate these finding in a different cultural milieu. Canadian Blacks are also a minority group but differ from the Black population in the U.S. in a number of ways. The majority of American Blacks are descendants of immigrants who moved to the U.S. involuntarily through the slave trade, whereas Canadian Blacks are mostly self-selecting immigrants and their descendants (Attewell, Kasinitz, & Dunn, 2010). The majority (72%) of Canadian Blacks are first generation whereas most American Blacks are third or more generation (86%; Attewell et al., 2010). Thus, examining the stress response of Canadian Blacks would be valuable in further testing the proposed mechanism of an exaggerated cardiovascular response to psychosocial stress as explaining racial disparities in hypertension. To date, no research is available on the cardiovascular response patterns of Canadian Blacks.

The purpose of this study was therefore to examine racial differences in cardiovascular reactivity to acute stress in Black and White Canadians. Specifically, it was hypothesized that Black Canadian men would exhibit greater peripheral vascular responses to a variety of laboratory stressors as compared to White Canadians as indexed by diastolic blood pressure

(DBP), total peripheral resistance (TPR), forearm blood flow (FBF), forearm vascular resistance (FVR), and mean arterial pressure (MAP). It was also hypothesized that Black as compared to White Canadians would exhibit as similar or diminished myocardial responses as indexed by heart rate (HR), cardiac output (CO), and stroke volume (SV). Finally, it was hypothesized that racial differences in reactivity will be observed in men and not women.

Method

Participants

Participants were healthy normotensive young adults composed of 45 Black Canadians (28 men, 17 women) and 49 White Canadians (28 men, 21 women) with an average age of 23 (range 19 to 36). Exclusion criteria were a resting blood pressure over 130/80 mmHg, a history of disease or current use of medication affecting the cardiovascular system, chronic or current substance abuse, and a history of psychiatric disorders. Black participants had to report both their parents to be Black to participate. Participants were asked to refrain from caffeine intake the day of their session as well as smoking and alcohol the night prior. Informed consent was obtained in accordance with the Research Ethics Unit of Concordia University, Montreal.

Procedures

Participants were instrumented with physiological recording equipment while in a seated position by a trained, race-matched laboratory technician. Three resting blood pressure measurements were taken to confirm participants' normotensive status followed by a rest period of 20 minutes. During the last five minutes, blood pressure measurements were taken every minute, and impedance measurements (e.g. CO, SV and TPR) taken continuously and averaged to obtain baseline means. After this initial period, three stress tasks (cold pressor, anger-recall and mental arithmetic task) were administered in counterbalanced order. Instructions for all tasks were presented immediately prior to each task. Each task was followed by a 20-minute recovery period. Participants then completed a health history questionnaire and Black participants, the Perceived Racism Scale (PRS; McNeilly et al., 1996). The PRS is a 51-item self-report instrument in a Likert-type scale response format on which 0 indicates *not applicable* and 5 indicates *several times a day* measuring the perceived frequency of exposure to racism in

four domains of life. McNeilly et al. (1996) reported internal reliability of the frequency of exposure with Cronbach's Alpha of .96. The PRS score reflected the cumulated scores of perceived racism in the last year on all four domains of the scale.

Stress tasks

The *cold pressor task* is a well-established potent pressor stimulus causing increases in blood pressure resulting from alpha-adrenergically mediated vasoconstriction (Sherwood, Allen, Obrist, & Langer, 1986). A thin plastic bag filled with ice and water, measured to be 4 degrees Celsius, was placed against the participant's forehead for two 2 1/2-minute periods separated by one minute. The *anger-recall task,* adapted from the Social Competence Interview (Ewart & Kolodnar, 1991)*,* required participants to recall an incident from their past in which they felt discriminated against and angered by it. The nature of the discrimination was left to the participant to choose and participants typically recounted discriminatory experiences relating to race, gender, age, etc. The anger recall task has been shown to produce a large increase in blood pressure (Krantz, Kop, Santiago, & Gottdiener, 1996; Ironson et al., 1992) and may have more ecological validity than more traditional laboratory stressors in that it involves the recalling of an actual life event. The *mental arithmetic task* (Sherwood & Turner, 1993) required participants to mentally complete subtraction equations. The task was designed to so that each participant attained 50-60% correct and equations became either more difficult or easy depending on the participant's response. Participants were informed that if an unspecified number of their responses were incorrect, they were to receive an electric shock, though no shocks were actually delivered. This task has been found to be a potent elicitor of blood pressure increases mediated primarily by beta-adrenergic myocardial responses (Sherwood, Allen, Obrist, & Langer, 1986).

Cardiovascular measures

SBP and DBP (in mm Hg) were measured oscillometrically using an IBS Model 700A automated blood pressure and pulse rate monitor (IBS Corp, Waltham, MA). A blood pressure cuff was placed on the participant's left thigh and values were corrected for distance from heart.

FBF (in cc/100cc/min) was obtained by using venous occlusion plethysmography. Pneumatic cuffs were placed above the elbow and at the wrist of the left arm that was elevated by a support at a 45% angle. Cuffs were inflated using a Hokanson rapid cuff inflation system

and FBF was derived from changes in forearm girth measured by strain gauge plesmythography during venous occlusion. The slope of the curve resulting from venous occlusion was computed and related to the girth of forearm circumscribed by the strain gauge.

Other cardiovascular measures included HR (in bpm), SV (in ml) and CO (in 1/min). These were measured using impedance cardiography and computer software was used for on-line acquisition and processing of the impedance signals. Thoracic impedance using a tetrapolar electrode system (band electrodes) and a standard ECG signal was used. Ensemble averaged values were based on impedance data collected continuously for 50 seconds of each minute during stress testing. Deflation of the IBS blood pressure cuff was temporally matched to the 50 second impedance data acquisition to ensure concurrent recording of blood pressure values. The data acquisition program (C.O.P. system) automatically detects the necessary components of the impedance cardiogram and generates CO and HR values. The Kubicek equation was used to derive SV from the impedance signals recorded. TPR (in dyne-sec.cm -5) was computed as (MAP/CO) x 80 where MAP (in mmHg) was calculated using the equation DBP + (SBP-DBP)/3. FVR ($dyn \cdot s/cm^5$) was computed by MAP/FBF.

Data reduction

Cardiovascular measurements were recorded each minute of each stressor task. Cardiovascular data were averaged over all minutes of each task period to obtain stress means for each task. Reactivity was evaluated for each cardiovascular measure by subtracting the baseline means from the stress mean of the corresponding cardiovascular measure. These change scores were used in analyses for task reactivity.

Data analysis

Potential racial and sex differences in cardiovascular reactivity were evaluated using a series of analysis of covariance (ANCOVA) in a mixed factorial design. Race (2 levels) and sex (2 levels) were the between-subjects factors, and stressor (3 levels) was the within-subjects factor for the nine ANCOVAs involving SBP, DBP, TPR, FBF, FVR, MAP, HR, CO, and SV change scores. Given the potential impact of BMI (Clark, Greenberg, Harris, & Carson, 2012), smoking (Hughes & Higgens, 2010), and parental history of hypertension (Frazer, Larkin, & Goodie, 2002) on cardiovascular reactivity, these were used as covariates. An α level of .05 was adopted

for all analyses. Effect sizes (partial eta squared) are reported for all F-tests. Significant interaction effects were evaluated further using ANCOVA techniques for simple effects analysis.

Results

Preliminary Analyses

Table 1 shows the descriptive characteristics of the sample, the groups significantly differed on BMI where Black men were significantly heavier than Black and White women; and smoking status where fewer Black men and more White men smoked than expected. Table 2 presents means and standard deviations for cardiovascular baseline levels and reactivity scores for each stressor task.

To assess the efficacy of the stress protocol, nine repeated measures analyses of variance were conducted for each of the cardiovascular measures with the within-subjects factor being task (four levels including baseline, cold pressor, anger-recall, and mental arithmetic). Pairwise comparisons indicated that there were significant increases from baseline to stress for cold pressor, anger-recall and mental arithmetic for all cardiovascular measures with the exception of FBF and HR during the cold pressor task, FVR during the anger-recall task (see Table 2). Overall, each stress task effectively increased cardiovascular function.

Pairwise comparisons also showed that the tasks varied in the response they elicited (see Table 2). For DBP, TPR, FBF, FVR, CO, increases during the cold pressor task were significantly greater than during the anger-recall or mental arithmetic tasks. For MAP and HR, increases during cold pressor were greater than during mental arithmetic. For SV, increases during cold pressor were greater than during anger-recall. For DBP, TPR, FVR, MAP, HR, and SV, increases during anger-recall were greater than during mental arithmetic.

Univariate correlations indicated that PRS was not associated with any of the cardiovascular measures during the baseline period or any of the tasks (data not shown).

Race and Sex Differences in Baseline Cardiovascular Function

To assess whether there were race and sex differences in baseline cardiovascular functioning, nine ANCOVAs on baseline levels of each cardiovascular measure were run with race and sex as the between-subjects factors. Differences in baseline levels between racial

groups were generally not observed with the exception of CO and HR where White participants had greater myocardial baseline levels than Black participants. A main effect of sex was found for SBP, TPR, MAP, CO, and SV showing that men typically had greater baseline cardiovascular levels than women.

Specifically, the analyses revealed that a significant sex by race interaction was found for HR, $F(1,90) = 4.156$, partial $\eta^2 = .046$, $p<.05$. Pairwise comparisons indicated that White men had significantly higher baseline HR than Black men. There were no racial differences for women. Analyses for CO revealed a significant main effect of race $F(1,90) = 4.250$, partial $\eta^2 = .047$, $p<.05$ where Whites had significantly higher baseline CO than Blacks and a significant main effect of sex $F(1,90) = 20.706$, partial $\eta^2 = .192$, $p<.05$ where men had significantly higher baseline CO than women. The main effect of sex reached significance for SBP, $F(1,90) = 23.078$, partial $\eta^2 = .210$, $p<.05$, where men had significantly higher baseline SBP than women. It did not reach significance for DBP. A significant main effect of sex was observed for TPR, $F(1,90) = 15.685$, partial $\eta^2 = .151$, $p<.05$, where women had significantly higher baseline TPR than men. The analysis examining FBF and FVR revealed no significant main effects of sex or race or sex by race interactions. A significant main effect of sex was observed for MAP, $F(1,90) = 10.561$, partial $\eta^2 = .108$, $p<.05$, where men had significantly higher baseline MAP than women. A significant main effect of sex was found for SV, $F(1,90) = 22.149$, partial $\eta^2 = .203$, $p<.05$, where men had significantly higher baseline SV than women.

Race and Sex Differences in Cardiovascular Reactivity to Stress

To assess racial-and sex-based differences in reactivity, nine ANCOVAs on changes in each cardiovascular measure were run with race and sex as the between-subjects factor and task as the within-subjects factor. Overall, sex by race interactions were found for DBP, TPR, MAP, HR, and SV showing that Black men had greater peripheral vascular responses and diminished myocardial responses to stress as compared to White men and Black and White women. These differences were primarily observed during the cold pressor task.

SBP. A significant sex by task interaction was observed for SBP change scores ($F(2,82) = 7.177$, $\eta^2 = .078$, $p< .05$). Planned comparisons following up this interaction indicated that men had greater increases in SBP as compared to women during the cold pressor task.

DBP. The analyses examining DBP change scores revealed a significant main effect of sex ($F(1,82) = 5.252$, $\eta^2 = .058$, $p < .05$) and task ($F(2,82) = 3.224$, $\eta^2 = .037$, $p < .05$), a sex by race interaction ($F(1,82) = 8.023$, $\eta^2 = .002$, $p<.05$), and a sex by task interaction ($F(2,82) = 15.776$, $\eta^2 = .157$, $p < .05$). However, the interpretation of these main effects and 2-way interactions was modified by a significant 3-way task by sex by race interaction ($F(2,82) = 4.558$, $\eta^2 = .012$, $p<.05$). Planned comparisons indicated that Black men had greater increases in DBP as compared to White men during the cold pressor task and mental arithmetic task. Black men were also found to have demonstrated greater increases in DBP as compared to Black women and to White women during the cold pressor task. In addition, White women had greater increases in DBP as compared to Black women during the cold pressor task (Figure 1).

TPR. A significant sex by race interaction was observed for TPR change scores ($F(1,82) = 5.068$, $\eta^2 = .056$, $p<.05$). Black men had greater increases in TPR as compared to White men during the mental arithmetic task (Figure 2).

FVR. A significant race by task interaction was found for FVR change scores ($F(2,82) = 6.424$, $\eta^2 = .074$, $p<.05$). Blacks had greater increases in FVR as compared to Whites during the cold pressor task (Figure 3).

MAP. Analyses examining MAP change revealed a significant main effect of sex ($F(1,82) = 5.360$, $\eta^2 = .059$, $p < .05$). The ANCOVA also revealed a significant sex by task interaction ($F(2,82) = 17.463$, $\eta^2 = .170$, $p<.05$) and a significant sex by race interaction ($F(1,82) = 7.712$, $\eta^2 = .083$, $p < .05$). Men had greater increases in MAP as compared to women during the cold pressor task. Black men were found to have greater increases in MAP than Black women, White men, and White women. White women were found to have marginally greater increases in MAP than Black women. A marginally significant 3-way task by sex by race interaction was observed ($F(2, 82) = 2.743$, $\eta^2 = .031$, $p=.067$) where Black men had greater MAP changes as compared to White men, Black women, and White women during the cold pressor task. White women had greater MAP changes as compared to Black women (Figure 4) during the cold pressor task.

HR. Analyses on HR change scores revealed a significant 3-way task by sex by race interaction ($F(2, 82) = 4.368$, $\eta^2 = .049$, $p<.05$). Black men's HR scores were different than Black women, White men, and White women during the cold pressor task. Black men also had marginally smaller increases in HR as compared to White men and smaller increases as compared to Black women (Figure 5) during the anger-recall task.

SV. Analyses on SV change revealed a significant main effect of race ($F(1,82) = 4.080$, $\eta^2 = .046$, $p < .05$). The ANCOVA also revealed a significant sex by task interaction ($F(1,82) = 4.175$, $\eta^2 = .047$, $p<.05$), a significant race by task interaction ($F(2,82) = 6.754$, $\eta^2 = .074$, $p< .05$), and a significant 3-way task by sex by race interaction ($F(2,82) = 4.466$, $\eta^2 = .050$, $p<.05$). White men showed greater decreases in SV as compared to Black men and White women during the cold pressor task. Black men showed marginally smaller decreases in SV as Black women during the cold pressor task. Black men showed smaller decreases in SV as compared to White men (Figure 6) during the mental arithmetic task.

No effects were observed for FBF or CO change scores.

Discussion

This study was the first to evidence patterns of cardiovascular reactivity to stress in Black and White Canadians. Individuals with increased cardiovascular responses to stress have been shown to be at a greater risk of developing cardiovascular diseases Carroll, 2003; Chida & Steptoe, 2010; Matthews, 2003; 2004; Treiber, 2003). Prior research conducted almost exclusively with U.S. Blacks has evidenced vascular hyperreactivity when exposed to stress, a response theorized to be in part related to exposure to racial discrimination. The current study sought to examine the stress response of Canadian Blacks given that they are also a marginalized group but differ from American Blacks in important ways, such as immigration history and immigrant generation. With a different sample, this study further tested the proposed mechanism that exaggerated cardiovascular response to psychosocial stress partly explains racial disparities in hypertension. The present results indicated that Canadian Black men had greater peripheral vascular responses (DBP, TPR, and MAP specifically) and diminished myocardial responses (HR and SV specifically) to stress as compared to White men as well as Black and White women. These differences were primarily observed during the cold pressor task.

These findings are consistent with studies conducted in the U.S. which have similarly found Blacks to exhibit greater vascular responses to stressors as compared to Whites (Anderson et al., 1988b; Dorr et al., 2007; Kelsey et al., 2000, Light et al., 1993; Reimann et al., 2012; Saab et al., 1992; Thomas, Nelesen, Ziegler, Natarajan, & Dimsdale, 2009; Treiber et al., 1990). These results also correspond with studies that found U.S. Blacks to have similar or diminished cardiac responses to stressors as compared to Whites (Light et al., 1993; Saab et al., 1992; Saab

et al., 1997). Together, these results suggest that there is a racial difference in the pattern of cardiovascular responding to stress where Blacks exhibit greater vascular reactivity as well as similar or diminished myocardial reactivity as compared to Whites. The increased incidence of hypertension in Blacks may be specifically related to this vascular dysfunction given that Blacks have greater mortality and morbidity from hypertension-related disease than other ethnic groups (Burt et al., 1995; Flack & Wiist, 1995) and that Blacks are more frequently treated with diuretics, which reduce blood volume, as compared to Whites (Miller & Doshi, 2006).

It also appears that Canadian participants perceive a similar degree of racial discrimination as do their U.S. counterparts. Black participants in the current study scored an average of 48.5 on the PRS (men: 55.75; women: 40.56). Male participants' score on the PRS is consistent with those of U.S. studies where scores ranged from 49.9 to 58.1 on the PRS (Comb et al., 2006; Hall & Carter, 2006; Hill, Kobayash, & Hughes, 2007; Nessor, 2008; Steffen, McNeilly, Anderson, & Sherwood, 2003). The Canadian Black population, particularly men, therefore seems to perceive their experience with racism similarly to their U.S. counterparts. A direct association between perceived racism scores and reactivity was not found. This may be a function of statistical power given the relatively small number of study participants. Other studies have reported mixed support for a direct relationship between racism and cardiovascular reactivity, and typically only in interaction with other psychosocial factors. Clark (2000) found that exposure to racism was positively related to DBP changes during a speech stressor. However, Clark & Adams (2004) reported that perceived racism was only predictive of blood pressure reactivity when it interacted with John Henryism and in another study, Clark (2003) found that perceived racism was predictive only in interaction with social support.

The current study found that racial differences in reactivity were more prominent in men as compared to women. Light et al. (1993) found a similar pattern of results in a mixed sample where differences in reactivity were more consistently found in men. Furthermore, Chida and Steptoe's meta-analysis (2010) reported that the association between greater reactivity to stress and future cardiovascular risk was stronger in men compared to women (r=0.117, $p<.05$ vs. r=0.015, $p>.05$). Taken together, the predictive utility of elevated response to stress seems to be particularly relevant to Black men.

Racial differences were primarily observed during the cold pressor task rather than the anger-recall or mental arithmetic tasks. These results are similar to those of several U.S. studies

that have also failed to observe racial differences in reactivity, all of which used psychological tasks as stressors such as a speech or mental arithmetic task (al'Absi et al., 2006; Delehanty, Dimsdale, & Mills, 1991; Dimsdale, Ziegler, Mills, Delehanty, & Berry, 1990; Saab et al., 1997), Other studies, however, have observed racial differences in reactivity to psychological stressors (Dorr et al., 2007; Reinmann et al., 2012; Saab et al., 1992; Treiber et al., 1990). These conflicting findings in the literature likely relate to the efficacy of psychological tasks in eliciting a large stress response. Preliminary analyses with the current sample showed that all three tasks significantly caused a stress reaction; however, the cold pressor generally elicited significantly greater changes than did the anger-recall or mental arithmetic. Thus, it may have been difficult to observe any possible racial difference given that the psychological stressors elicited a smaller change in cardiovascular functioning than did the cold pressor task, particularly when the sample size of this study was small.

Studies comparing psychological stressors with cold pressor have yielded inconsistent findings. Some studies have shown an association between the two types of tasks (McKinney et al., 1985; Turner, Sherwood, & Light, 1990; 1994) whereas others have not (Allen & Crowell, 1989; Parati et al., 1988). It would therefore be imprudent to draw conclusions of a lack of racial differences when considering the current results during the anger-recall and mental arithmetic tasks. Individuals tend to regulate blood pressure via cardiac or vascular mechanisms, and this tendency persists across different tasks (Kasprowicz, Manuck, Malkoff, & Krantz, 1990; Sherwood, Dolan, & Light, 1990). Despite this, it may be that racial differences in reactivity can only be observed when using stressors eliciting primarily an alpha-adrenergic response such as the cold pressor (Sherwood et al., 1986) and not observed when using primarily beta-adrenergic stressors such as the mental arithmetic task (Sherwood & Turner, 1993). Furthermore, cold stress is commonly used in laboratory research on hypertension (Allen, Obrist, Sherwood, & Crowell, 1987; Kanayama, Khatun, Belayet, She, & Terao, 1999; Kelsey, Alpert, Patterson, & Barnard, 2000) and it has been shown in a number of studies to predict later blood pressure and the development of hypertension (Menkes et al., 1989; Kasagi, Akahoshi, & Shimaoka, 1995; Wood, Sheps, Elveback, & Schirger, 1984). This lends support to the current findings that Black Canadians demonstrate exaggerated cardiovascular reactivity to stress.

In conclusion, the current study observed differences in reactivity to acute stress where Canadian Black men exhibited greater vascular responses and diminished myocardial responses

to stress as compared to White individuals. Racial differences in reactivity were not observed in women. These results suggest that previous findings of vascular hyper-reactivity in African-Americans may be extended to Blacks in other countries with different immigration histories. Future research focusing on additional psychosocial factors would be important in understanding the role of discrimination in the cardiovascular response and disease risk of Black individuals.

Table 1

Descriptive Characteristics of Sample as a Function of Race and Sex (n=94).

	Black men (*n*=28)	Black women (*n*=17)	White men (*n*=28)	White women (*n*=21)
Age (yr)	24.0	23.4	22.7	22.7
BMI (kg/m^2)[a]	25.4	21.9	23.6	22.1
Smoking status (% current smoker) [a]	0%	6%	39%	28%
Parental history of hypertension (% yes)	57%	61%	54%	48%
PRS	55.75	40.56	n/a	n/a

Note. [a] Group means significantly different ($p < .05$).

Table 2

Means (SD) Baseline and Changes in Cardiovascular Reactivity during Cold Pressor, Anger-Recall and Mental Arithmetic Tasks as a Function of Race and Sex (n=94).

		Baseline	Δ Cold pressor	Δ Anger-recall	Δ Mental arithmetic
SBP (mm/Hg)					
Black	Men	120.2 (12.3)	18.7 (10.8)	17.5 (7.9)	17.2 (8.6)
	Women	109.9 (12.4)	9.5 (5.6)	16.2 (7.8)	13.3 (8.4)
	Combined	116.3 (13.2)	15.0 (10.1)	17.0 (7.8)	15.6 (8.7)
White	Men	125.7 (11.3)	16.5 (10.4)	14.8 (7.7)	15.3 (11.8)
	Women	108.3 (8.0)	11.6 (5.9)	17.7 (8.3)	15.8 (9.5)
	Combined	117.9 (13.1)	14.4 (9.0)	16.1 (8.0)	15.5 (10.8)
DBP (mm/Hg)					
Black	Men	71.4 (8.7)	24.2 (10.6)	15.4 (6.6)	12.0 (6.0)
	Women	67.5 (8.1)	10.3 (6.6)	14.7 (7.2)	10.1 (6.6)
	Combined	69.9 (8.6)	18.7 (11.4)	15.1 (6.8)	11.2 (6.3)
White	Men	67.9 (8.4)	18.8 (8.0)	13.6 (7.5)	8.0 (8.2)
	Women	66.3 (9.0)	17.4 (7.0)	16.0 (6.1)	9.8 (6.7)
	Combined	66.3 (9.0)	18.2 (7.6)	14.7 (6.9)	8.8 (7.6)
TPR (Dynes-s •cm^5)					
Black	Men	834.1 (178.4)	279.4 (170.1)	125.4 (131.5)	73.3 (105.8)
	Women	1075.4 (286.3)	219.1 (110.4)	90.8 (234.3)	93.0 (171.7)
	Combined	925.2 (251.7)	245.7 (172.4)	111.7 (177.5)	81.1 (134.1)
White	Men	790.7 (195.1)	219.1 (110.4)	85.6 (119.6)	10.4 (94.9)
	Women	966.2 (339.9)	248.5 (139.3)	139.7 (130.7)	68.1 (142.4)
	Combined	865.6 (278.0)	231.7 (123.1)	108.8 (126.1)	35.1 (119.8)
FBF (cc/100cc/min)					
Black	Men	3.4 (1.3)	-.4 (1.1)	.7 (1.3)	1.0 (1.3)
	Women	2.3 (.9)	.0 (.7)	.5 (.8)	.5 (.8)
	Combined	3.0 (1.3)	-.3 (1.0)	.6 (1.1)	.9 (1.2)
White	Men	3.0 (1.7)	-.2 (1.3)	.6 (1.6)	.5 (.7)
	Women	2.4 (1.4)	.2 (.6)	.5 (1.7)	1.4 (3.0)
	Combined	2.7 (1.6)	-.0 (1.1)	.6 (1.6)	.9 (2.0)
FVR (dyn·s/cm^5)					
Black	Men	30.4 (13.0)	19.6 (17.2)	.3 (8.9)	-1.9 (4.5)
	Women	41.8 (17.3)	11.6 (25.2)	-1.1 (17.4)	-2.8 (11.1)
	Combined	34.2 (15.4)	16.9 (20.2)	-.1 (12.2)	-2.2 (7.2)
White	Men	41.2 (27.3)	7.0 (13.8)	-.6 (9.0)	-.6 (4.4)
	Women	40.4 (18.6)	.8 (9.8)	.9 (16.8)	-7.6 (22.9)
	Combined	40.9 (23.6)	4.6 (12.7)	-.0 (12.4)	-3.2 (14.7)
MAP (mmHg)					
Black	Men	87.6 (6.6)	22.5 (8.4)	16.0 (5.5)	13.4 (5.6)
	Women	81.6 (7.0)	10.0 (5.1)	15.1 (6.8)	11.3 (5.8)
	Combined	85.3 (7.3)	17.5 (9.5)	15.6 (6.0)	12.6 (5.7)

White	Men	86.9 (8.5)	18.0 (7.7)	14.1 (6.9)	10.4 (8.5)
	Women	78.9 (7.5)	15.4 (5.4)	16.5 (5.6)	12.3 (5.1)
	Combined	83.5 (9.0)	16.9 (6.8)	15.1 (6.4)	11.2 (7.2)
HR (BPM)					
Black	Men	61.4 (8.8)	-3.2 (6.6)	8.5 (7.1)	10.2 (6.9)
	Women	65.6 (11.8)	.9 (3.1)	12.0 (9.3)	8.0 (5.6)
	Combined	63.0 (10.1)	-1.5 (5.8)	9.9 (8.1)	9.3 (6.4)
White	Men	67.1 (11.6)	.4 (6.7)	11.3 (8.4)	7.9 (8.8)
	Women	61.7 (10.2)	2.4 (4.6)	10.0 (6.7)	12.5 (17.1)
	Combined	64.8 (11.2)	1.2 (5.9)	10.7 (7.7)	9.9 (13.1)
CO (1/min)					
Black	Men	8.6 (1.6)	-.4 (1.0)	.4 (1.1)	.6 (1.0)
	Women	6.3 (1.3)	-.2 (.5)	.8 (1.4)	.5 (1.0)
	Combined	7.8 (1.8)	-.3 (5.8)	.5 (1.2)	.6 (1.0)
White	Men	9.3 (2.4)	-.4 (.7)	.4 (1.3)	.9 (1.4)
	Women	7.1 (1.9)	-.3 (.4)	.3 (.8)	.8 (1.7)
	Combined	8.3 (2.5)	-.4 (.6)	.3 (1.1)	.9 (1.5)
SV (ml)					
Black	Men	142.4 (24.9)	1.7 (15.5)	-10.8 (13.7)	-10.3 (11.2)
	Women	99.5 (23.8)	-4.8 (5.8)	-5.1 (12.1)	-4.7 (11.6)
	Combined	126.2 (32.0)	-.8 (12.9)	-8.5 (13.3)	-8.0 (11.6)
White	Men	142.0 (38.1)	-8.5 (9.6)	-15.1 (13.3)	-2.3 (10.7)
	Women	115.1 (23.6)	-9.0 (8.2)	-11.0 (9.6)	-7.2 (9.9)
	Combined	130.5 (33.5)	-8.7 (9.0)	-13.3 (11.9)	-4.4 (10.5)

Note. Δ Cold Pressor, Δ Anger-Recall and Δ Mental Arithmetic calculated as change from baseline to stress.

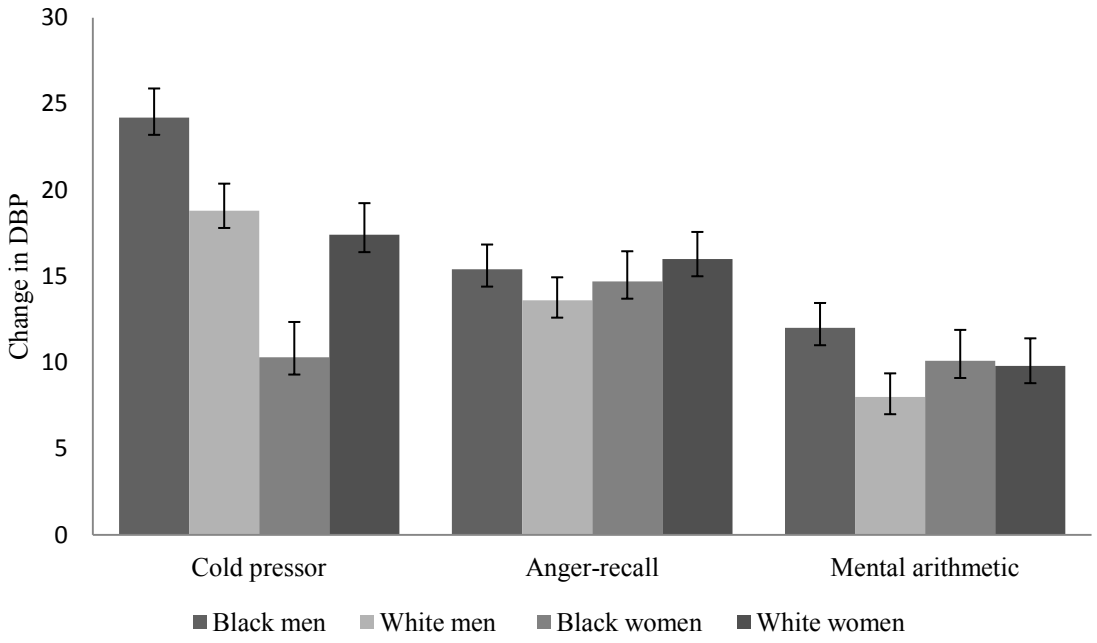


Figure 1. Changes in DBP (± SE) during cold pressor, anger-recall and mental arithmetic tasks for Black men, White men, Black women and White women.

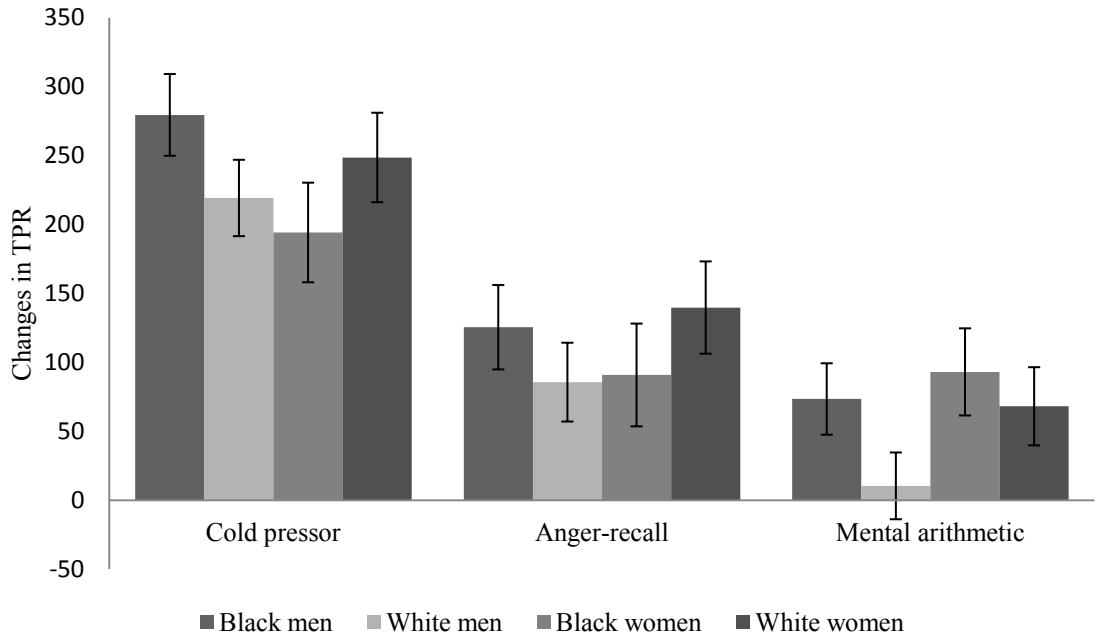


Figure 2. Changes in TPR (± SE) during cold pressor, anger-recall and mental arithmetic tasks for Black men, White men, Black women and White women.

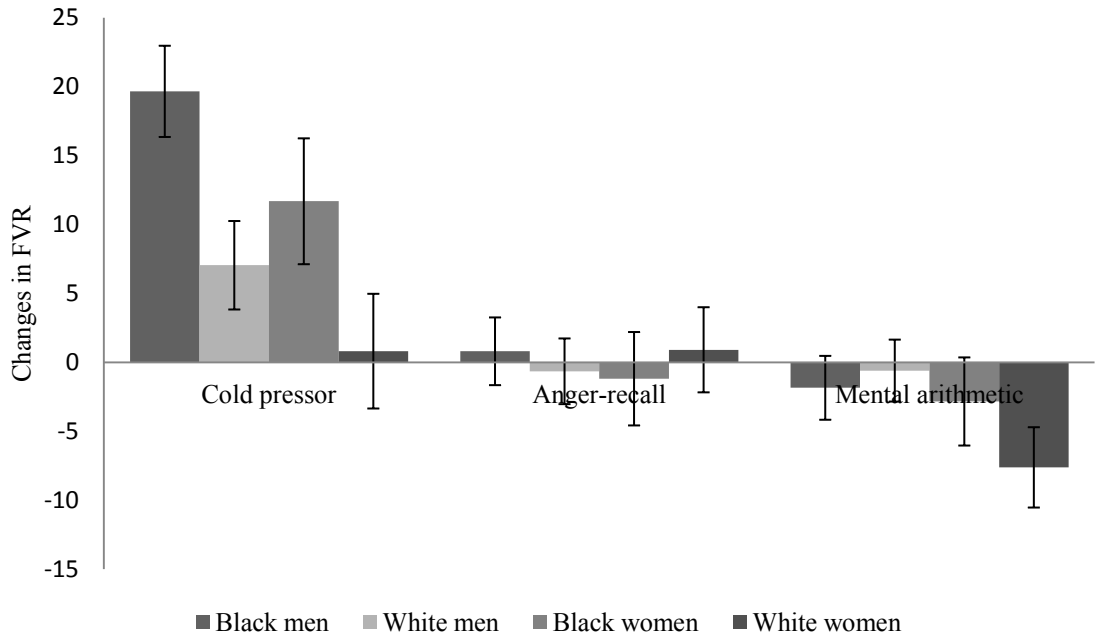


Figure 3. Changes in FVR (± SE) during cold pressor, anger-recall and mental arithmetic tasks for Black men, White men, Black women and White women.

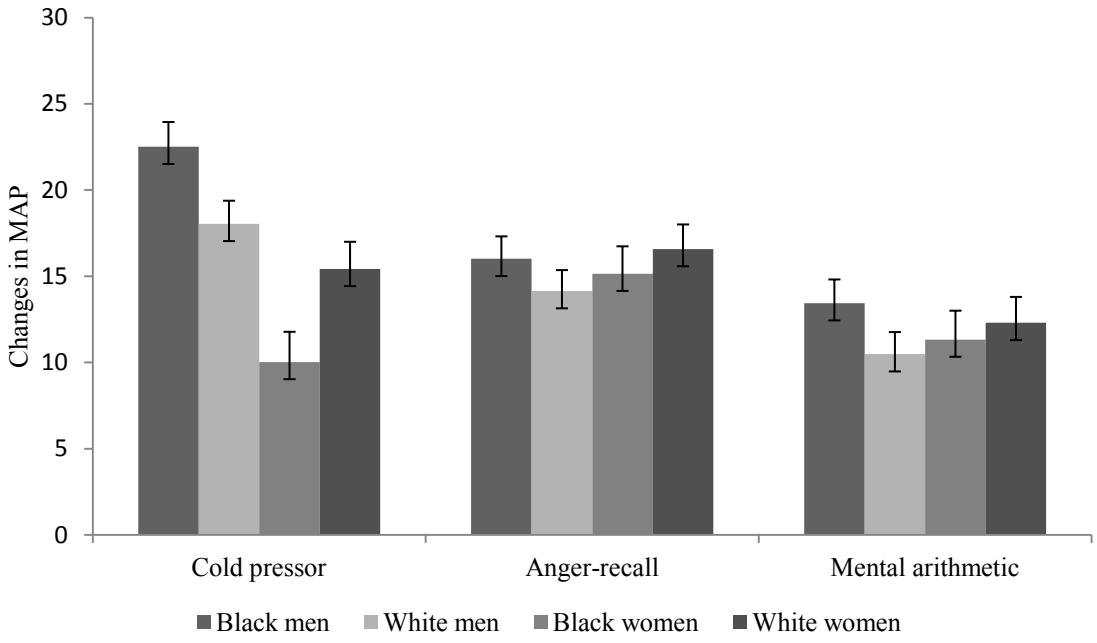


Figure 4. Changes in MAP (± SE) during cold pressor, anger-recall and mental arithmetic tasks for Black men, White men, Black women and White women.

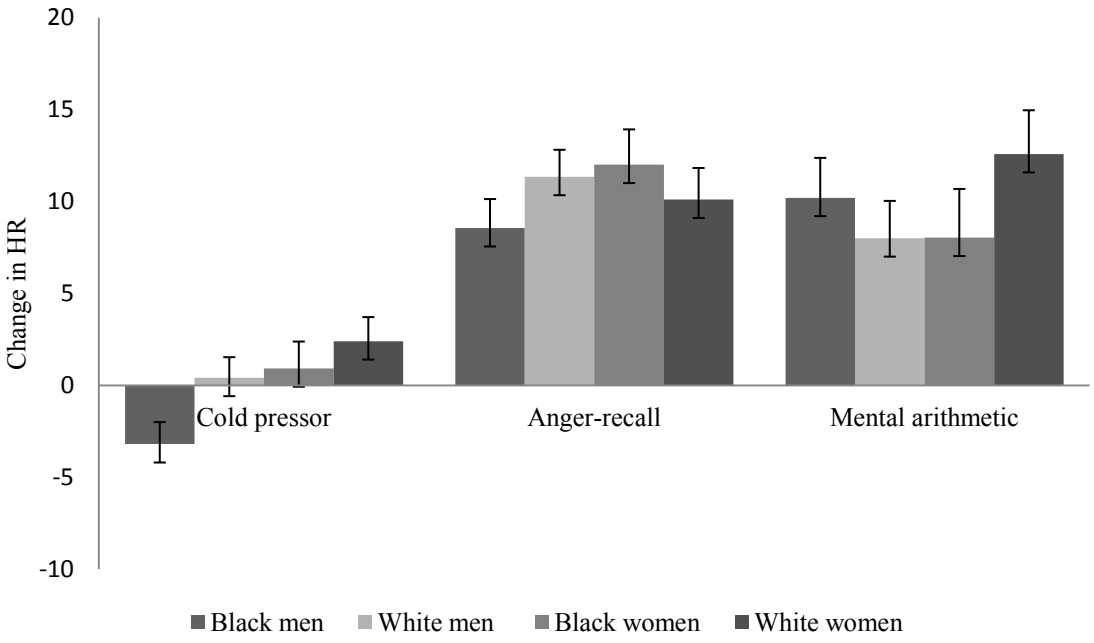


Figure 5. Changes in HR (± SE) during cold pressor, anger-recall and mental arithmetic tasks for Black men, White men, Black women and White women.

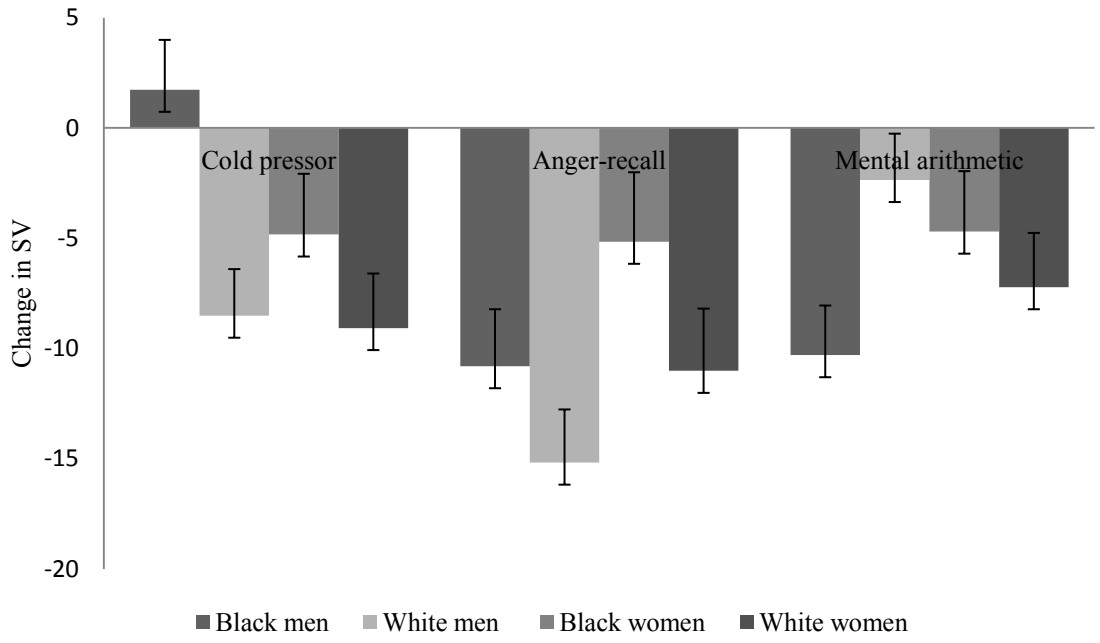


Figure 6. Changes in SV (± SE) during cold pressor, anger-recall and mental arithmetic tasks for Black men, White men, Black women and White women.

Study Three:

Perceived Racism and Anger Expression Predict Cardiovascular Reactivity to Stress in Black Canadians

Dolezsar, C.M. & Miller, S.B. Perceived Racism and Anger Expression Predict Cardiovascular Reactivity to Stress in Black Canadians. (under review).

Abstract

Objective: Evidence suggests that racial discrimination contributes to the increased risk of hypertension in Blacks. Several reviews suggest that the effects of racism may be mitigated by individual differences. Few studies have examined the relationship between racism, anger expression, and blood pressure. The goal of the present study was to examine the direct and interactive effects of perceived racism and anger expression in predicting cardiovascular reactivity to stress.

Methods: Cardiovascular responses to three laboratory stress tasks (cold pressor, anger-recall, and mental arithmetic) were examined in Black participants (n=38; mean age, 24 years). Participants reported on the Perceived Racism Scale and the anger-out and -in subscales of the Speilberger Anxiety Expression Scale.

Results: Hierarchical linear regressions indicated that perceived racism did not independently predict reactivity. Anger-out and anger-in independently predicted cardiac reactivity. Findings also revealed that anger expression interacted with perceived racism. Having a greater tendency to either express anger outwardly or suppress anger resulted in greater vascular and decreased cardiac reactivity in participants who perceived a lesser degree of racism as compared to those perceiving a greater degree of racism.

Conclusion: High levels of both outward expression and suppression of anger may serve as protective factors in individuals who are exposed to a high degree of stress of racial discrimination. It is unclear why both high anger-out and -in have similar patterns in predicting reactivity. These interactions may be dependent on other contextual variables in which anger expression takes place. More research is needed to fully understand these relationships.

Perceived Racism and Anger Expression Predict Cardiovascular Reactivity to Stress in Black Canadians

Black, as compared to White, individuals have a greater incidence of hypertension and are more likely to die of hypertension-related consequences (Singh, Kochanek & MacDorman, 1994; Hall et al., 1997; Klag et al., 1997). There is little evidence that genetic factors account for this disparity (Cooper, 2005; Hertz, Unger, Cornell, & Saunders, 2005) and differences in traditional risk factors do not adequately explain the increased incidence (Cornoni-Huntley, LaCroix, & Havlik, 1989). Researchers have thus examined environmental factors that may be implicated.

Racial discrimination is a potent and chronic psychosocial stressor that has been posited to contribute to racial disparities in hypertension (Clark, Anderson, Clark & Williams, 1999; Brondolo, Gallo & Myers, 2009). Recent studies have reported that Blacks experience racism when seeking education or employment, getting loans for housing, or in everyday interactions with others (Clark, Anderson, Clark & Williams, 1999; Krieger, Sidney & Coakley, 1998). One of the predominant theories explaining the relationship between psychosocial stressors and hypertension is the cardiovascular reactivity hypothesis where heightened reactivity to stress is hypothesized to play a causal role in the development of hypertension (Krantz & Manuck, 1984). Over time, exaggerated cardiovascular activity from exposure to stress is hypothesized to lead to vasculature changes that culminate in the development of CVD (Anderson, McNeilly & Myers, 1992; Krieger, 1990).

Several studies have examined the link between perceived racism and risk for hypertension. Racist stimuli have been found to elicited greater blood pressure responses than neutral or anger-provoking stimuli (Fang & Myers, 2001; Guyll, Matthews & Bromberger, 2001; McNeilly et al., 1995; Sutherland & Harrell, 1986). Blacks who report more frequent experiences with racism also exhibit exaggerated blood pressure reactivity (Clark, 2000; Clark, 2003; Clark, 2006b; Thomas, Nelesen, Malcarne, Ziegler, & Dimsdale, 2006; Fang & Myers, 2001; Armstead, Lawler, Gordon, Cross, & Gibbon, 1989; Jones, Harrell, Morris-Prather, Thomas, & Omowale, 1996; McNeilly et al., 1995; Morris-Prather et al., 1996). A recent meta-analysis documented a small association between racial discrimination and hypertension which was stronger among certain subgroups (Dolezsar et al., 2014). In other reviews, Brondolo et al. (2011) reported a small and complex relationship between racism and hypertension where individual differences

likely moderate this relationship. In this regard, Harrell et al. (2003) and Pascoe and Smart Richman (2009) considered coping and social support as moderators. Taken together, perceptions of racism have been demonstrated to be associated with an increased risk for cardiovascular disease, which is thought to be mitigated by individual difference factors.

Anger may be a particularly relevant factor given the well-documented association between anger and the risk of cardiovascular disease (Fang & Myers, 2001; Hogan & Linden, 2004; Schum, Jorgensen, Verhaeghen, Sauro, & Thibodeau, 2003) and because anger is one of the most frequently reported psychological effects of racism (Bullock & Housten, 1987; Landrine & Klonoff, 1996). Blacks report higher levels of anger than Whites (Barefoot, Dahlstrom & Williams, 1983; Scherwitz, Perkins, Chesney & Hughes, 1991) and it is the primary reason why Blacks seek out psychotherapy (National Institute of Mental Health, 1983).

How one expresses anger is likely also relevant. The expression of anger refers to how one copes with angry feelings (Spielberger et al., 1985). Anger can be expressed outwardly towards others by verbal or physical aggressive behaviour (anger-out) or it can be suppressed (anger-in). Some evidence suggests that these patterns consist of two dimensions rather than representing a continuum (Spielberger & Sydeman, 1994). Anger expression has been linked to cardiovascular disease progression in several studies, although the mode of anger expression remains unclear. Some studies have found that expressing anger outwardly is associated with greater reactivity or cardiovascular disease (Mendes de Leon, 1992; Siegman, Dembroski, & Ringel, 1987), while others have found that the inhibition of anger is more predictive (Haynes, Feinleib, & Kennel, 1980; Suarez & William, 1990).

The influence of anger expression styles on cardiovascular outcomes, and specifically anger-in, may be particularly important for Black individuals. Harburg et al. (1973; 1979) reported that anger inhibition was more predictive of cardiovascular risk factors in Blacks than in Whites. Johnson, Schork & Spielberger (1987) demonstrated that blood pressure increased at lower levels of anger-in among Black than White women. Brownley et al. (1996) found that low hostility was related to higher blood pressure in Black men who suppressed their anger. Poole et al. (2006) found that anger-in, but not anger-out, played a modulating role in predicting reactivity in Black but not in White participants. Dorr et al. (2007) found that Black, as compared to White participants, who expressed their anger recovered more slowly.

If the magnitude of the stress response to discrimination varies as a function of individual differences and anger is a common response to perceived racism; it is likely that anger expression style interacts with perceived racism to influence cardiovascular functioning. Most research, however, has examined the influence of perceived racism, anger, and anger expression independently. Of the few studies that looked at these connections, Steffen et al. (2003), found that perceived racism was positively related to anger inhibition but not to outwardly expressed anger in Black and White participants. They also found that greater perceived racism predicted higher daytime ambulatory blood pressure and those who reported holding their anger in had increased nighttime ambulatory blood pressure. Anger inhibition, however, did not account for the relationship between discrimination and blood pressure. Clark (2006a) reported that trait anger and perceived racism did not independently predict blood pressure in Black adolescents but that trait anger mediated the relationship between racism and blood pressure. Contrary to what was expected, racism was inversely related to blood pressure, only among those who were low in trait anger. Conversely, Clark (2000) reported that perceived racism predicted diastolic blood pressure changes to a speech stressor, but that this relationship was not mitigated by anger in Black women.

The effects of anger expression and perceived racism, directly and in interaction clearly require further elucidation. Though the literature in this area is somewhat contradictory, the preponderance of evidence suggests that anger inhibition is a more powerful predictor of cardiovascular hypereactivity than anger-out. Nevertheless, the present study sought to examine the direct effects of anger-in, anger-out, and perceived racism as well as their interactive effects. It was hypothesized that direct effects of perceived racism and anger expression would not be observed. It was also hypothesized that perceived racism would be positively related to blood pressure, only among those who tend to suppress their anger. Finally, it was hypothesized that interactive effects of anger-out with perceived racism would not emerge to predict blood pressure.

Method

Participants

Participants were healthy normotensive Black Canadians (22 men, 16 women) with a mean age of 24 (ranging from 19 to 36 years). Exclusion criteria were a resting blood pressure over 130/80 mmHg, a history of cardiovascular disease or current use of medication affecting the cardiovascular system, chronic or current substance abuse, or a history of psychiatric disorders. Participants had to report both their parents as Black to participate. Participants were asked to refrain from caffeine intake the day of their session and alcohol from the night prior.

Cardiovascular measures

SBP and DBP (in mm Hg) were measured oscillometrically using an IBS Model 700A automated blood pressure and pulse rate monitor (IBS Corp, Waltham, MA) with the cuff placed on the participant's left thigh (values corrected for distance from heart).

FBF (in cc/100cc/min) was obtained by using venous occlusion plethysmography. Pneumatic cuffs were placed above the elbow and at the wrist of the left arm that was elevated at a 45% angle. Cuffs were inflated using a Hokanson rapid cuff inflation system and FBF was derived from changes in forearm girth measured by strain gauge plesmythography during venous occlusion. The slope of the curve resulting from venous occlusion was computed and related to the girth of forearm circumscribed by the strain gauge.

Other cardiovascular measures included HR (in bpm), SV (in ml) and CO (in 1/min). These were measured using impedance cardiography and C.O.P. software (Chapel Hill, NC) computer software was used for on-line acquisition and processing of the impedance signals. This system uses ensemble averaging to effectively filter out respiratory and movement artifact from the cardiac component of the thoracic impedance signal. Ensemble averaged values were based on impedance data collected continuously for 50 seconds of each minute during stress testing. Deflation of the IBS blood pressure cuff was temporally matched to the 50 second impedance data acquisition to ensure concurrent recording of blood pressure values. The data acquisition program automatically detects the necessary components of the impedance cardiogram and generates CO and HR values. The Kubicek equation was used to derive SV from the impedance signals recorded. TPR (in dyne-sec.cm -5) was computed as (MAP/CO) x 80 where MAP (in mmHg) was calculated using the equation DBP + (SBP-DBP)/3. FVR ($dyn \cdot s/cm^5$) was computed by MAP/FBF.

Questionnaires

Perceived racism. The Perceived Racism Scale (PRS; McNeilly et al., 1996) is a 51-item self-report instrument that assesses experiences with racism across four domains of life (racism on the job, in academic setting, in public setting, and exposure to racist statements) in the past year and over the course of their lives. McNeilly et al. (1996) reported internal reliability of the frequency of exposure with Cronbach's Alpha of .96. Perceived racism was measured by the sum of participants' frequency of exposure to racism across their lifetime in all four domains of life.

Anger. The Speilberger Anger Expression Scale (AX; Spielberger et al., 1985) assesses how often angry feelings are experienced and expressed or suppressed. AX consists of Anger-out (tendency to express anger), Anger-in (tendency to suppress anger), and total Anger Expression subscales. The AX scale demonstrated Cronbach's alpha coefficients of .73 to .84. In the current study, anger-out and anger-in were used to predict cardiovascular reactivity.

Procedures

Participants were instrumented with physiological recording apparatus while in a seated position by a trained Black laboratory technician. Three resting blood pressure measurements were taken to confirm participants' normotensive status followed by a rest period of 20 minutes. During the last five minutes, blood pressure measurements were taken every minute, and impedance measurements taken continuously and averaged to obtain baseline means. Three stress tasks were then administered in counterbalanced order and instructions for tasks were presented immediately prior to each task. Tasks were followed by a 20-minute recovery period. Participants then completed a health history questionnaire, PRS, and AX.

Stress tasks

The *cold pressor task* is a well-established stimulus causing increases in blood pressure resulting from alpha-adrenergically mediated vasoconstriction (Sherwood, Allen, Obrist, & Langer, 1986). A thin plastic bag filled with ice and water at 4^{o} C was placed against the forehead for two 2 1/2-minute periods separated by one minute. The *anger-recall task,* adapted from the Social Competence Interview (Ewart & Kolodnar, 1991) required participants to recall

an incident from their past in which they felt discriminated against and angered by it. The anger-recall task has been shown to produce a large increase in blood pressure (Krantz, Kop, Santiago, & Gottdiener, 1996; Ironson et al., 1992) and may have more ecological validity than more traditional laboratory stressors in that it involves the recalling of an actual life event. The *mental arithmetic task* (Sherwood & Turner, 1993) required participants to mentally complete subtraction equations and causes increases in blood pressure resulting from beta-adrenergic myocardial responses (Sherwood, Allen, Obrist, & Langer, 1986). The task was designed to so that each participant attained 50-60% correct and participants were informed that if an unspecified number of their responses were incorrect, they were to receive an electric shock, though no shocks were actually delivered.

Data reduction and analysis

Criterion variables. Cardiovascular data were averaged over all minutes of each task period to obtain stress means for each task. Reactivity was evaluated for each cardiovascular measure by subtracting the stress mean from the baseline mean of the corresponding cardiovascular measure.

Predictor and control variables. The centered values for perceived racism and anger-out were the primary predictor variables in the hierarchical linear regression analyses. Multiplying those centered values created the perceived racism x anger expression interaction terms.

The potential for confounding by sex (Kajantie & Phillips 2005), BMI (Clark, Greenberg, Harris, & Carson, 2012), smoking (Hughes & Higgens, 2010), and parental history of hypertension (Frazer, Larkin, & Goodie, 2002) was assessed by Pearson correlation and T-test analyses. All of these were found to be related to several cardiovascular change scores. Therefore, sex, BMI, smoking, and parental history of hypertension were used as control variables.

Analyses. The individual and interactive effects of perceived racism and anger-out on reactivity were measured with nine hierarchical linear regression models involving SBP, DBP, TPR, FBF, FVR, MAP, HR, CO, and SV changes. Anger-out and -in were not significantly correlated ($r=.29$, $p>.05$) and were therefore run in separate models due to power limitations. The individual and interactive effects of perceived racism and anger-in on reactivity were

measured in another series of nine hierarchical linear regression models involving all cardiovascular change scores. The control variables were entered in Step 1, the primary predictor variables (perceived racism and anger-out or anger-in) were entered in Step 2, and the interaction term was entered in Step 3. Two participants were missing data on two variables which were replaced with the variable mean. All statistical analyses were computed using SPSS software package (Version 16.0). An α level of .05 was adopted for all analyses.

Results

Preliminary Analyses

Table 1 shows descriptive characteristics of the sample. Bivariate relationships are shown in Table 2. Repeated measures analyses of variance showed that there were significant increases from baseline period to task period for all three tasks and nine cardiovascular outcomes.

Perceived Racism, Anger-out, and Cardiovascular Changes during Cold Pressor Task

Table 3 illustrates the final step of the hierarchical linear regression analyses examining the relationship of perceived racism and anger-out to changes on each cardiovascular measure during each stress task.

DBP. In Step 1, none of the control variables were predictors (ps>.05). In Step 2, the perceived racism and anger-out variables were not predictors (ps>.05). In the final step ($F(7,30) = 4.86$, $p < .01$), perceived racism interacted with anger-out to predict DBP change ($B = -.028$, $SE = .013$, $p<.05$). Anger-out interacted with perceived racism to affect DBP such that those who were higher on anger-out exhibited less reactivity, particularly among participants who perceived more discrimination (figure 1).

FVR. In Step 1, none of the control variables were predictors (ps>.05). In Step 2, anger-out but not perceived racism was a predictor ($B = 2.18$, $SE = 0.87$, $p <.05$). In the final step ($F(7,30) = 3.54$, $p < .05$), perceived racism interacted with anger-out to predict FVR change ($B = -.053$, $SE = .023$, $p<.05$). Anger-out interacted with perceived racism to affect FVR such that

those who were higher on anger-out exhibited less reactivity among participants who perceived more discrimination (figure 2).

MAP. In Step 1, only sex predicted change in MAP ($p < .05$). In Step 2, the perceived racism and anger-out variables were not predictors ($ps>.05$). In the final step, the interaction term did not reach significance in predicting MAP change.

CO. In Step 1, none of the control variables were predictors ($ps>.05$). In Step 2, anger-out but not perceived racism was a predictor ($B = -.097$, $SE = .041$, $p < .05$) where participants with greater scores on anger-out tended to react less. In the final step, the interaction term did not predict CO change.

Other outcomes. In models examining the predictors of SBP, TPR, HR, SV changes, the omnibus F statistics for Steps 1 through 3 were not significant ($ps>.05$). Further, an examination of the independent and interactive effects associated with these steps did not reveal any significant relationships between predictor variables and cardiovascular changes. In models examining FBF, the omnibus F statistics for Steps 1 through 3 were significant ($ps<.05$), however predictor variables and the interaction term were not significantly associated with FBF changes.

Perceived Racism, Anger-out, and Cardiovascular Changes during Anger-Recall Task

HR. In Step 1, only smoking predicted HR change ($p < .05$). In Step 2, the perceived racism and anger-out variables were not predictors ($ps>.05$). In the final step ($F(7,30) = 3.14$, $p < .05$), perceived racism interacted with anger-out to predict HR change ($B = .021$, $SE = .010$, $p<.05$) . Anger-out interacted with perceived racism to affect HR such that those who were higher on anger-out exhibited more cardiac reactivity among participants who perceived more discrimination (figure 3).

SBP. In Step 1, none of the control variables were predictors ($ps>.05$). In Step 2, anger-out but not perceived racism was a predictor ($B = .743$, $SE = .352$, $p < .05$) where participants with greater scores on anger-out tended to react more. In the final step, the interaction term did not predict SBP change.

In models examining the predictors of DBP, TPR, FBF, FVR, MAP, and SV changes, the omnibus F statistics for Steps 1 through 3 were not significant (ps>.05). Further, an examination of the independent and interactive effects associated with these steps did not reveal any significant relationships between predictor variables and cardiovascular changes. In models examining CO, the omnibus F statistics for Steps 1 through 3 were significant (ps<.05), however predictor variables and the interaction term were not significantly associated with CO changes.

Perceived Racism, Anger-out, and Cardiovascular Changes during Mental Arithmetic Task

In models examining the predictors of SBP, DBP, TPR, FBF, FVR, MAP, HR, CO, and SV changes, the omnibus F statistics for Steps 1 through 3 were not significant (ps>.05). Further, an examination of the independent and interactive effects associated with these steps did not reveal any significant relationships between predictor variables and cardiovascular changes.

Summary of Findings for Anger-out

In sum, a greater tendency to express anger outwardly resulted in lower vascular reactivity in participants who perceived a high degree of racism (as index by DBP and FVR during the cold pressor). The reverse was found for HR during anger-recall where those with a greater tendency to express their anger outwardly had greater cardiac reactivity in participants who perceived a high degree of racism. Direct effects of perceived racism were not observed.

Perceived Racism, Anger-in, and Cardiovascular Changes during Cold Pressor Task

DBP. In Step 1, only sex was a predictor ($p < .05$). In Step 2, the perceived racism and anger-in variables were not predictors (ps>.05). In the final step, the interaction term did not reach significance in predicting DBP change.

TPR. In Step 1, none of the control variables were significant predictors of TPR change (ps>.05). In Step 2, the perceived racism and anger-in variables were not predictors (ps>.05). In the final step, only anger-in was a predictor ($B = 36.131$, $SE = 14.843$, $p < .05$).

FVR. In Step 1, none of the control variables were predictors (ps>.05). In Step 2, the perceived racism and anger-in variables were not predictors (ps>.05). In the final step ($F(7,30) =$

2.47, $p < .05$), perceived racism interacted with anger-in to predict FVR change (B = -.058, SE = .023, $p<.05$). Anger-in interacted with perceived racism to affect FVR such that those who were higher on anger-in exhibited less reactivity among participants who perceived more discrimination (figure 4).

CO. In Step 1, none of the control variables were significant predictors (ps>.05). In Step 2, the perceived racism and anger-in variables did not reach significance to predict change in CO (ps>.05). In the final step, only anger-in was a predictor (B = -0.150, SE = .077, $p < .05$).

SV. In Step 1, none of the control variable were predictors (ps>.05). In Step 2, anger-in but not perceived racism was a predictor (B = -1.210, SE = .419, $p < .05$). In the final step, anger-in continued to be the only significant predictor (B = -2.645, SE = .912, $p < .05$).

Other outcomes. In models examining the predictors of SBP and HR, the omnibus F statistics for Steps 1 through 3 were not significant (ps>.05). Further, an examination of the independent and interactive effects associated with these steps did not reveal any significant relationships between predictor variables and cardiovascular changes. In models examining FBF and MAP, the omnibus F statistics for Steps 1 through 3 were significant (ps<.05), however predictor variables and the interaction term were not significantly associated with FBF or MAP changes. Table 4 illustrates the final step of the hierarchical linear regression analyses during all tasks.

Perceived Racism, Anger-in, and Cardiovascular Changes during Anger-Recall Task

TPR. In Step 1, only smoking predicted TPR change ($p < .05$). In Step 2, the perceived racism and anger-in variables were not predictors (ps>.05). In the final step ($F(7,30) = 3.25$, $p < .05$), perceived racism interacted with anger-in to predict TPR change (B = -.530, SE = .206, $p<.05$). Anger-in interacted with perceived racism to affect TPR such that those who were higher on anger-in exhibited less reactivity among participants who perceived more discrimination (figure 5).

FBF. In Step 1, none of the control variables were predictors (ps>.05). In Step 2, neither anger-in nor perceived racism predicted FBF change (ps>.05). In the final step, only anger-in was a predictor (B = 0.201, SE = .100, $p < .05$).

HR. In Step 1, only smoke predicted HR change ($p < .05$). In Step 2, the perceived racism and anger-in variables were not predictors ($ps > .05$). In the final step ($F(7,30) = 3.56$, $p < .05$), perceived racism interacted with anger-in to predict HR change ($B = .024$, $SE = .009$, $p < .05$). Anger-in interacted with perceived racism to affect HR such that those who were higher on anger-in exhibited more reactivity among participants who perceived more discrimination (figure 6).

CO. In Step 1, smoke and parental history were predictors ($p < .05$). In Step 2, the perceived racism and anger-in variables were not predictors ($ps > .05$). In the final step ($F(7,30) = 4.73$, $p < .05$), perceived racism interacted with anger-in to predict CO change ($B = .003$, $SE = .001$, $p < .05$). Anger-in interacted with perceived racism to affect CO such that those who were higher on anger-in exhibited more reactivity among participants who perceived more discrimination (figure 7).

SV. In Step 1, only smoke predicted SV change ($p < .05$). In Step 2, anger-in but not perceived racism was a predictor ($B = -1.014$, $SE = .421$, $p < .05$) where participants with greater scores on anger-in tended to react less. In the final step, the interaction term did not predict SV change.

In models examining the predictors of SBP, DBP, MAP, and FVR changes, the omnibus F statistics for Steps 1 through 3 were not significant ($ps > .05$). Further, an examination of the independent and interactive effects associated with these steps did not reveal any significant relationships between predictor variables and cardiovascular changes.

Perceived Racism, Anger-in, and Cardiovascular Changes during Mental Arithmetic Task

TPR. In Step 1, only smoke predicted TPR change ($p < .05$). In Step 2, anger-in but not perceived racism was a predictor ($B = 9.039$, $SE = 4.392$, $p < .05$) where participants with greater scores on anger-in tended to react more. In the final step, the interaction term did not predict DBP change.

SV. In Step 1, only smoke predicted SV change ($p < .05$). In Step 2, anger-in but not perceived racism was a predictor ($B = -1.081$, $SE = .375$, $p < .05$) where participants with greater

scores on anger-in tended to react less. In the final step, the interaction term did not predict SV change.

In models examining the predictors of SBP, DBP, FBF, FVR, MAP, HR, and CO changes, the omnibus F statistics for Steps 1 through 3 were not significant (ps. > .05). Further, an examination of the independent and interactive effects associated with these steps did not reveal any significant relationships between predictor variables and cardiovascular changes.

Summary of Findings for Anger-in

In sum, a greater tendency to suppress anger resulted in lower vascular reactivity in participants who perceived a high degree of racism (as indexed by FVR during the cold pressor and TPR during anger-recall). The reverse was found for HR and CO during anger-recall where those with a greater tendency to suppress anger had greater cardiac reactivity in participants who perceived a high degree of racism. Direct effects of perceived racism were not observed. Direct effects of anger-in were observed where a tendency to suppress anger was generally related to increased vascular reactivity and decreased cardiac reactivity.

Discussion

The goal of the present study was to examine the direct and interactive effects of perceived racism and anger expression in predicting cardiovascular reactivity to stress. Direct effects of perceived racism were not observed. Direct effects of anger-out and anger-in did emerge. Both anger expression and anger suppression were positively associated with vascular measure of reactivity and inversely associated with cardiac measures of reactivity.

If several factors are implicated in the increased risk of hypertension in Blacks (Clark, 2006, Brondolo et al., 2011, Harrell et al., 2003, Pascoe and Smart Richman, 2009), it is likely that the association of perceived racism to blood pressure depends on the interactive effects more than the direct effects of these variables. The present findings mostly confirmed the hypotheses, showing that the relation of perceived racism with cardiovascular reactivity varied as a function of anger-out. Having a greater tendency to express anger resulted in less vascular reactivity (as indexed by DBP and FVR during the cold pressor) when participants perceived a high degree of racism suggesting that outward expressions of anger may therefore serve as a protective factor in

individuals who are exposed to a high degree of stress of racial discrimination. The fact that a tendency to express anger was associated with a greater reactivity in lesser racism experiences is more difficult to explain however. Interestingly, a similar pattern of results was observed with anger-in where the relationship of perceived racism to reactivity was moderated by anger-in. Having a greater tendency to suppress anger resulted in less vascular reactivity (as indexed by FVR during the cold pressor and TPR during the anger-recall) when participants perceived a higher degree of racism as compared to when they perceive a lesser degree of racism. Again, the fact that that a tendency to hold anger in led to greater reactivity in lesser racism experiences is more difficult to explain. Nonetheless it does seem that high levels of both outward expressions of anger and suppression of anger may serve as protective factors in individuals who are exposed to a high degree of stress of racial discrimination.

The opposite pattern of results was observed in cardiac measures of reactivity. The relationship of perceived racism to reactivity was moderated in both models with anger-out and anger-in. However here, having a greater tendency to express anger outwardly resulted in less cardiac reactivity (as indexed by HR during the cold pressor) when participants perceived a lesser degree of racism as compared to when they perceive a greater degree of racism. Similarly, having a greater tendency to suppress anger resulted in less cardiac reactivity (as indexed by HR and CO during anger-recall) when participants perceived a lesser degree of racism.

It is unclear why the two different modes of anger expression lead to the same result. The Anger Expression Scales (Spielberger et al., 1986) were developed to evaluate the expression of anger and exhibit good internal consistency and are relatively stable over time (Fuqua et al., 1991, Musante et al., 1999, Spielberger et al., 1995, Spielberger, 1988 and Spielberger & Sydeman, 1994). During the initial development of the anger-out and anger-in scales, it was theorized that these subscales would represent opposite ends of the anger expression spectrum. However, structural analysis of these scales found instead that they were largely independent of each other (Knight et al., 1988 and Spielberger, 1988). Analyses showed that the correlation between anger-out and anger-in approached significance ($r = .29$, $p < .10$), but that only 8.4% of the variance in one of the subscales is shared by the other subscale ($r^2 = 0.084$). Individuals who score high on the anger-out subscale frequently express anger in physically aggressive acts or verbally in the form of criticism, sarcasm, insults, and profanity.

Those who score high on the anger-in subscale frequently experience intense angry feelings and tend to suppress them rather than expressing these feelings in aggressive manners. Very little is discussed in the literature on individuals who score high on both these subscales. Of the few authors who do address this, Spielberger et al. (2009) suggests that those with high anger-in and anger-out scores may frequently express their anger in some situations but suppress it in others. Spielberger (1999) also reported that individuals with high scores on both subscales may be at high risk for heart attacks and coronary artery disease.

In line with Spielberger et al.'s (2009) assertion, other research has shown that anger expression styles depend on the situation. The measurement of anger expression in the current study and above-mentioned research was from a trait perspective, assuming that it is consistent across different situation. It has been shown, however, that contextual factors (i.e. at home vs. work) strongly influence anger expression style (al'Absi & Bongard, 2006; Bishop, 2008). Social learning of ways of responding to anger instigation in a socially appropriate manner is postulated to vary depending on the context, such as social status, gender, culture (Hokanson and Burgess, 1962). This may help explain why current results revealed a similar pattern in respondents who tended to score high on anger-in and anger-out subscales. A clearer picture of the relation between racism anger expression styles might emerge if respondents were asked about their expressive styles in different contexts.

Few published empirical reports examine the association between racism, anger expression style and blood pressure. Door et al. (2007) also found that both anger inhibition and expression were related to blood pressure. In their study, however, anger-out and anger-in was related to poorer cardiovascular functioning (delayed recovery from debate stressors) in Black participants. This was observed in racially-themed debates and neutral debate stressors. Armstead et al.'s (1989) results were in a similar direction that those currently reported. They found that anger-out was negatively associated with blood pressure reactivity to a racist film. In contrast, Steffen et al. (2003) found anger inhibition and perceived racism to be independent predictors of ambulatory blood pressure, but anger inhibition did not mediate this relationship. Together these suggest that anger expression styles have a different relation with perceived racism depending on the cardiovascular measure used (i.e. reactivity to stress, recovery from

stress, or ambulatory blood pressure), however this research area is very undeveloped and much more investigation is needed to thoroughly understand this relationship.

It is unclear why the current results suggest that being at the extreme of either anger expression styles is protective at high levels of racism but harmful at lower levels of racism in relation to vascular reactivity. At high levels of perceived racism, either expressing or suppressing anger may be socially acceptable depending on the situation and be adaptable ways to react to stress. At lower levels of perceived racism, it is difficult to understand why a greater tendency to suppress or express anger would lead to greater reactivity to stress. Clearly more research is necessarily to further understand these associations.

Interestingly, perceived racism was not found to predict cardiovascular reactivity to stress directly, but only in interaction with anger expression. Other studies have also found that perceived racism predicted cardiovascular reactivity only in interaction with another factor. Clark (2003) found that perceived racism interacted with the quality and quantity of social support to predict blood pressure changes in Black college students during a mental arithmetic challenge. In conditions of high perceived racism, high social support was associated with greater reactivity. Clark and Gochett (2006) found that perceived racism interacted with "accepting it" and "talking to someone" as coping responses to racism in predicting resting blood pressure in Black youth. Several authors having written qualitative and quantitative reviews on the impact of racism on health outcomes have suggested that this relationship is likely moderated by individual differences (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Harrell, Hall, & Taliaferro, 2003, Pascoe and Smart Richman, 2009). Results from this study support this notion that the effects of perceived racism on health are regulated by other factors, namely anger expression styles.

In conclusion, perceived racism and anger expression may function in interaction in their contribution to the etiology of cardiovascular disease in Blacks. Direct effects of perceiving racism were not found, but rather its effect on cardiovascular reactivity depends on one's habitual style of expressing anger. At high levels of perceived racism, those who had a strong tendency to either suppress anger or express their anger outwardly showed less vascular reactivity to stress. High levels of either suppression of anger and outward expressions of anger may therefore serve as protective factors in individuals who perceive a high degree of stress of

racial discrimination. However, the direction of this interaction remains unclear, and may be dependent on other social, contextual variables. More research is needed to fully elucidate these relationships.

Table 1

Characteristics of Sample (n=38).

	M	*SD*	Range	*N*	%
Age (yr)	23.8	4.1	19.0-36.0		
BMI (kg/m^2)	23.9	4.5	17.0-40.3		
Perceived racism	61.8	30.1	5.0-144.0		
Anger-out	15.4	3.4	9.0-24.0		
Anger-in	16.4	4.7	8.0-25.0		
Sex (% male)				20	52.6
Smoking status (% never smoked)				35	92.1
Parental history of hypertension (% yes)				22	57.9
Baseline SBP (mmHg)	116.0	13.4	73.5-139.3		
Baseline DBP (mmHg)	69.4	8.9	51.0-90.3		
Baseline TPR (Dynes-s •cm^5)	963.3	273.3	528.3-1669.8		
Baseline FBF (cc/100cc/min)	3.1	1.4	0.7-7.0		
Baseline FVR ($dyn \cdot s/cm^5$)	33.8	15.1	13.4-91.2		
Baseline MAP (mmHg)	84.9	7.8	65.2-99.7		
Baseline HR (BPM)	64.3	10.5	42.0-86.2		
Baseline CO (1/min)	7.6	2.0	4.2-12.1		
Baseline SV (ml)	119.5	33.0	60.7-191.4		

Table 2

Descriptive and Bivariate Relationships among Study Variables during Cold Pressor Task (n=38).

	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*	*10*	*11*	*12*	*13*	*14*	*15*	*16*	*M(SD)*
1. Sex[a]	-																52.6%
2. BMI	-.42*	-															23.9 (4.5)
3. Smoking [b]	.00	-.15	-														92.1%
4. Parental history [c]	-.06	-.05	-.23	-													57.9%
5. Perceived racism	-.30**	.28**	-.08	.09	-												61.8 (30.1)
6. Anger-out	.25	-.15	-.14	-.01	-.15	-											15.4 (3.4)
7. Anger-in	.28**	.01	-.23	.01	-.03	.29**	-										16.4 (4.7)
8. ΔSBP	-.41*	.11	-.06	.12	-.08	-.04	-.17	-									13.0 (7.7)
9. ΔDBP	-.67*	.23	-.01	-.01	.18	-.25	-.32**	.45*	-								18.6 (11.7)
10. ΔTPR	-.29**	.19	.22	-.06	.08	-.05	.16	.40*	.63*	-							261.6 (186.6)
11. ΔFBF	.34	-.39*	-.28	-.21	.02	.11	.27	-.32**	-.24	-.26	-						-0.4 (1.0)
12. ΔFVR	-.22	.28	.16	.22	-.08	.30**	-.06	.17	.26	.39*	-.57*	-					17.0 (20.8)
13. ΔMAP	-.69*	.23	-.02	.02	.14	-.23	-.32	.66*	.97*	.62*	-.29	.26	-				16.8 (9.2)
14. ΔHR	.42*	-.26	-.07	.09	-.11	-.10	.22	-.02	-.37*	-.34*	.40*	-.44*	-.31**	-			-1.7 (6.14)
15. ΔCO	.19	-.14	-.25	.20	-.18	-.24	-.18	-.06	-.21	-.68*	.24	-.36*	-.19	.64*	-		-0.4 (0.9)
16. ΔSV	-.32**	.17	-.22	.17	-.01	-.22	-.43*	-.05	.17	-.41*	-.15	.06	.14	-.38*	.41*	-	-0.9 (12.5)

Note. [a]For sex, men = 1 and women = 2. [b] For smoking, yes = 1 and 2 = no. [c]For parental history, yes = 1 and 2 = no.

Δ indicates change scores.

*p < .05. **p < .10.

Table 3

Final Step of Hierarchical Regression Analyses of Perceived Racism and Anger-out Predicting Cardiovascular Changes during Cold Pressor Task, Anger-Recall, and Mental Arithmetic (n=38).

Variable	SBP		DBP		TPR		FBF		FVR		MAP		HR		CO		SV	
	B	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*
Cold Pressor																		
Perceived racism	-.06	.04	-.03	.05	-.23	1.11	.01	.01	-.16*	.10	-.04	.04	.00	.03	-.01	.01	-.06	.07
Anger-out	.00	.67	1.09	.79	19.25	16.62	-.03	.08	4.87	1.45	.71	.62	-.25	.51	-.08	.08	-1.08	1.09
Interaction	.00	.01	-.03*	.01	-.31	.27	.00	.00	-.05*	.02	-.02**	.01	.00	.01	.00	.00	.01	.02
Anger-Recall																		
Perceived racism	-.04	.04	-.03	.04	-.96	1.03	-.01	.01	-.02	.07	-.02	.04	.01	.04	.00	.01	.04	.07
Anger-out	.89	.63	.22	.67	18.91	15.50	-.10	.11	.89	.99	.45	.57	-1.41	.64	-.12	.10	-.43	1.07
Interaction	.00	.01	.00	.01	-.17	.25	.00	.00	.00	.02	.00	.01	.02*	.01	.00	.00	-.00	.02
Mental Arithmetic																		
Perceived racism	-.01	.05	.01	.04	.21	.74	-.01	.01	.02	.04	.01	.03	-.02	.04	.00	.01	.03	.06
Anger-out	1.36	.73	-.06	.53	-2.08	11.07	-.06	.11	.85	.65	.42	.47	-.50	.53	.09	.08	1.45	.96
Interaction	-.01	.01	.00	.01	.03	.18	.00	.00	.00	.01	-.01	.01	.01	.01	.00	.00	-.02	.02

*p < .05. **p < .10.

Table 4

Final Step of Hierarchical Regression Analyses of Perceived Racism and Anger-in Predicting Cardiovascular Changes during Cold Pressor Task, Anger-Recall, and Mental Arithmetic (n=38).

Variable	SBP		DBP		TPR		FBF		FVR		MAP		HR		CO		SV	
	B	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*	*B*	*SE B*
Cold Pressor																		
Perceived racism	-.06	.04	.01	.05	.19	1.00	.01	.01	-.10	.10	-.01	.04	.00	.04	-.01	.01	-.07	.06
Anger-in	-.50	.64	.95	.77	36.13*	14.84	.06	.07	3.51*	1.51	.46	.60	-.16	.51	-.15*	.07	-2.64*	.91
Interaction	.01	.01	-.02**	.01	-.40**	.23	.00	.00	-.06*	.02	-.01	.01	.01	.01	.00	.00	.03**	.01
Anger-Recall																		
Perceived racism	-.04	.04	-.02	.04	-.62	.92	-.01	.01	-.01	.06	-.02	.04	-.02	.04	-.01	.01	.04	.07
Anger-in	.30	.64	.80	.62	40.82*	13.48	.20*	.10	.68	.93	.61	.55	-1.47	.60	-.23*	.09	-1.6	.96
Interaction	-.01	.01	-.02	.01	-.53*	.21	.00	.00	-.02	.01	-.01	.01	.02	.01	.00*	.00	.01	.01
Mental Arithmetic																		
Perceived racism	-.01	.05	.02	.03	.17	.68	-.01	.01	.02	.04	.02	.03	-.02	.04	.00	.01	.06	.06
Anger-in	-.08	.74	-.06	.48	7.88	10.05	-.05	.11	-.10	.64	.02	.44	-.11	.52	-.02	.08	-.64	.85
Interaction	-.01	.01	-.01	.01	.02	.15	.00	.00	-.01	.01	-.01	.01	.00	.01	.00	.00	-.01	.01

*p < .05. **p < .10.

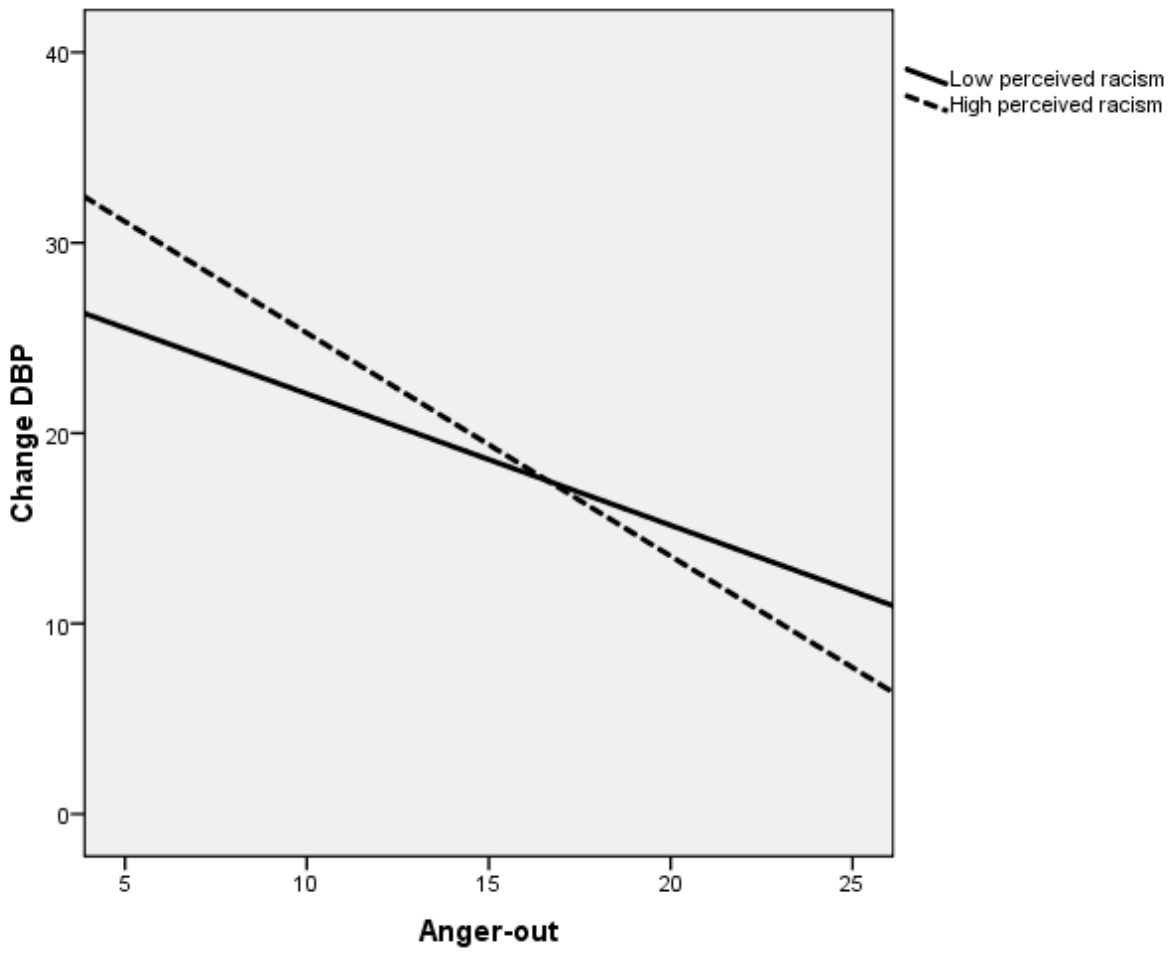


Figure 1. Change in DBP during cold pressor task as a function of anger-out for individuals high/low on perceived racism.

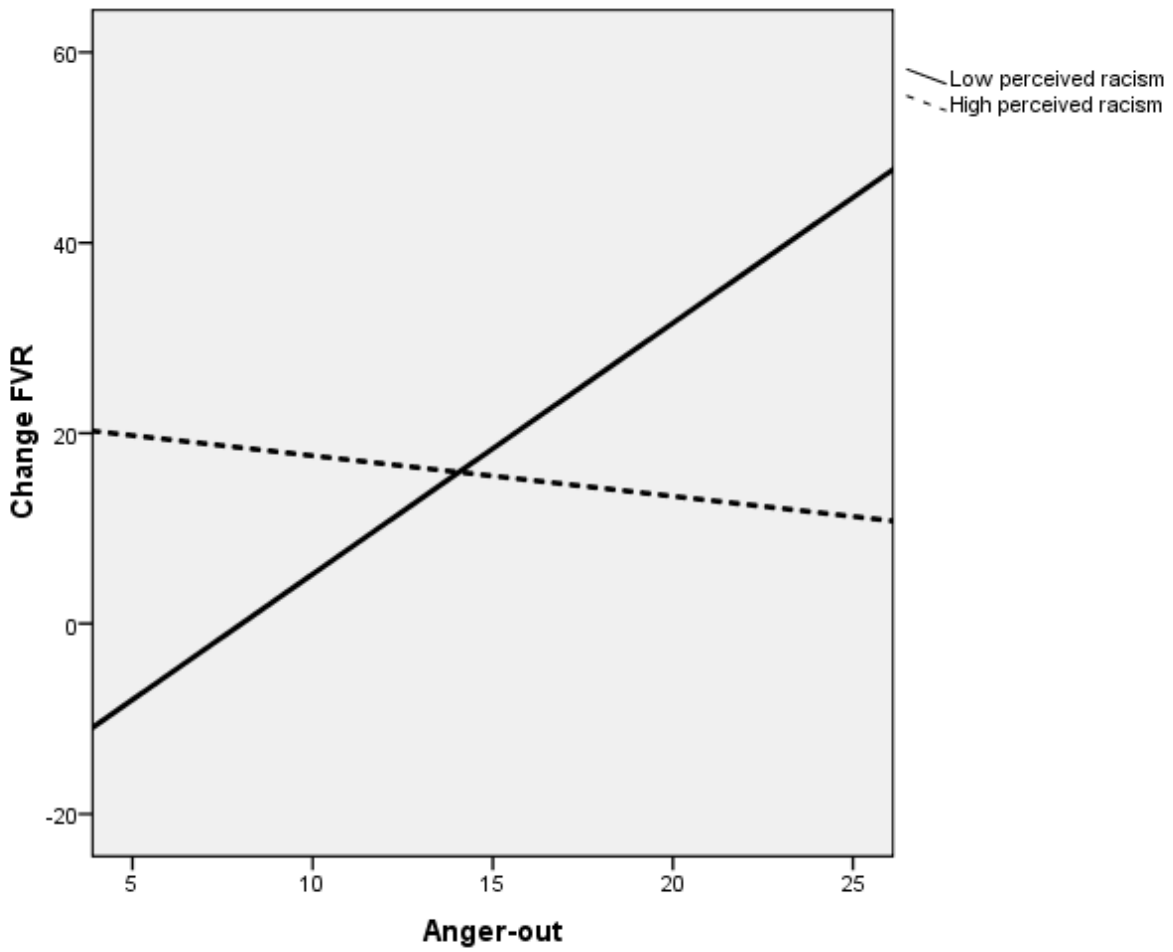


Figure 2. Change in FVR during cold pressor task as a function of anger-out for individuals high/low on perceived racism.

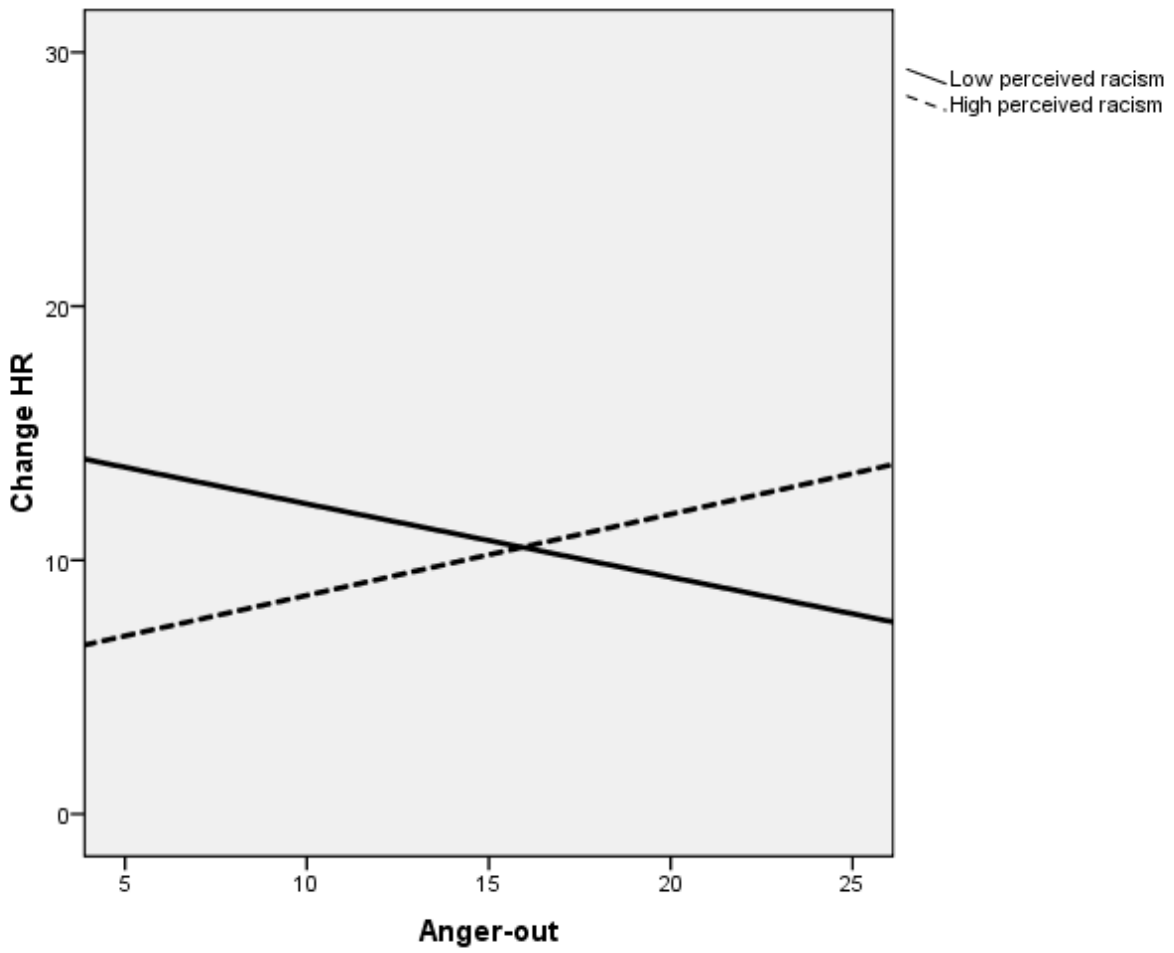


Figure 3. Change in HR during anger-recall task as a function of anger-out for individuals high/low on perceived racism.

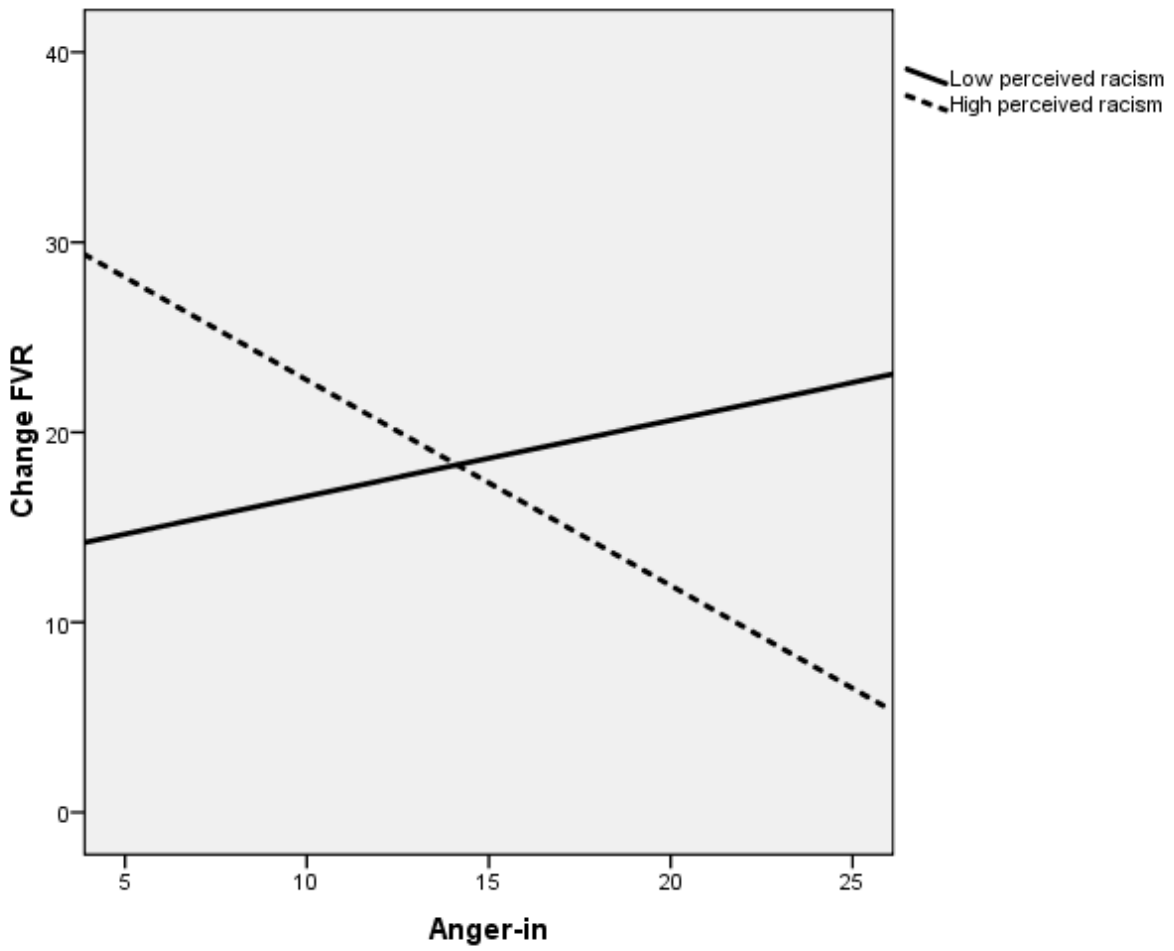


Figure 4. Change in FVR during cold pressor task as a function of anger-in for individuals high/low on perceived racism.

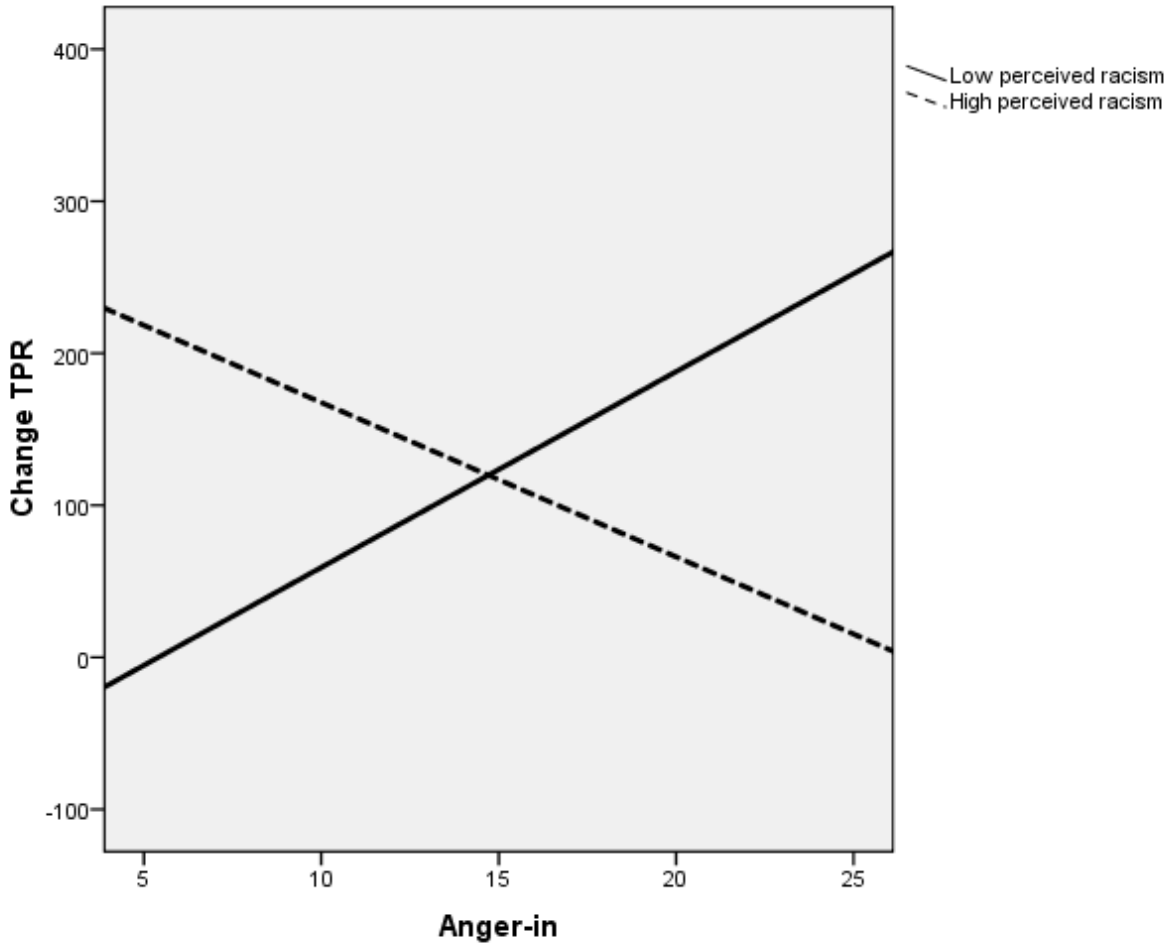


Figure 5. Change in TPR during anger-recall task as a function of anger-in for individuals high/low on perceived racism.

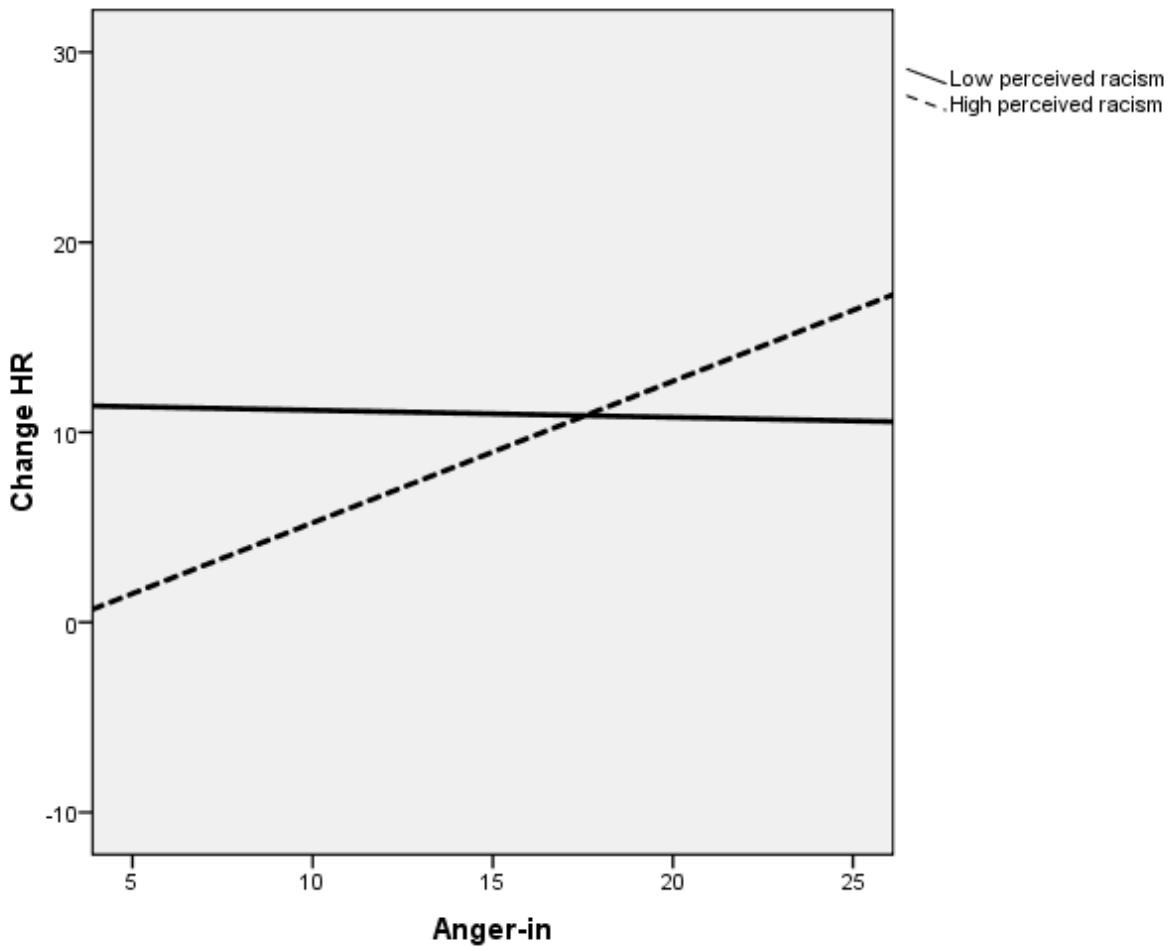


Figure 6. Change in HR during anger-recall task as a function of anger-in for individuals high/low on perceived racism.

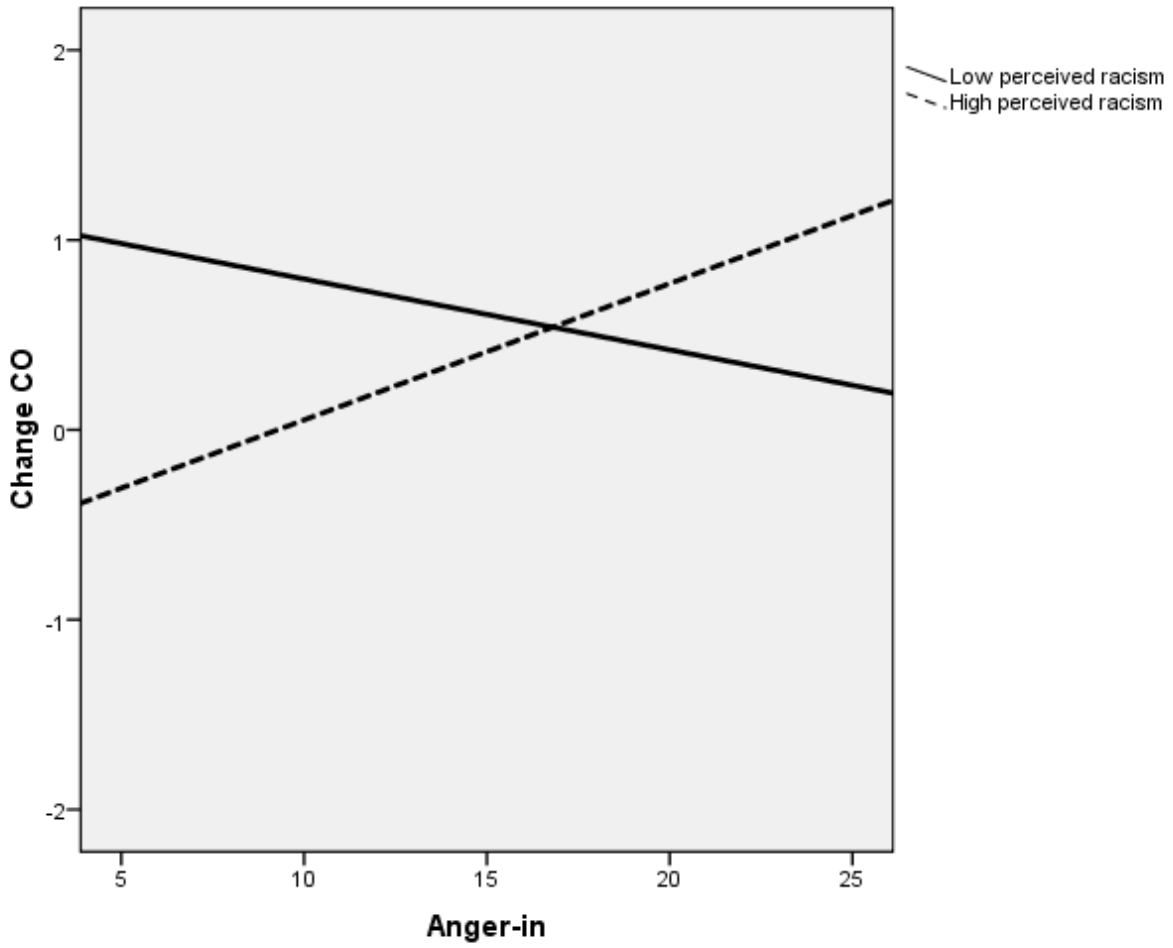


Figure 7. Change in CO during anger-recall task as a function of anger-in for individuals high/low on perceived racism.

General Discussion

Researchers have posited that the increased risk of hypertension seen in Blacks is related to being exposed to the stress of racism and its consequence (Lui et al., 2010; Malan et al., 2010). This thesis aimed to further understand this relationship between racism and cardiovascular functioning by addressing several gaps in the literature. Extensive research has been conducted to estimate the association between perceived discrimination and blood pressure but findings have been mixed. Several literature reviews have been conducted, but have been qualitative and thus did not ascertain the magnitude of this relationship (Williams & Mohammed, 2009; Brondolo, Rieppi, Kelly, and Gerin, 2003; Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Harrell, Hall, & Taliaferro, 2003; Paradies, 2006). They have also been unable to quantify the effect of moderator variables. Therefore, a meta-analysis of previous research examining the link between perceived racism and blood pressure was conducted. Results revealed a small relation between perceived racism and hypertension, but not with resting blood pressure. The effect was found to be stronger in men, Blacks, older participants, lower education attainment, and hypertensive status. The next study looked at reactivity to stress in a laboratory setting. Blacks experience a greater degree of stress than Whites due to racial discrimination and over time, sustained activation from stress has been posited to lead to structural changes in the vascular system resulting in disease (Brondolo, Gallo, & Myers, 2009; Krantz & Manuck, 1984). Exaggerated cardiovascular reactivity to stress has been found to be predictive of future cardiovascular disease (Chida & Steptoe, 2010). Several studies have shown Blacks to have greater vascular reactivity to stress than Whites, however, they have exclusively been tested on American samples (Saab et al., 1992; Treiber et al., 1990; Anderson et al., 1988b; Dorr, Brosschot, Sollers, & Thayer, 2007; Reimann et al., 2012). The second study therefore sought to examine reactivity in a Canadian population, where the immigration histories are different than for American Blacks, as well as economic and realities. Black men were shown to exhibit greater vascular response to laboratory stress.

Several researchers have proposed that other factors likely moderate the relation between discrimination and cardiovascular functioning. Anger has extensively been studied in relation to cardiovascular disease but few studies have looked at its relation to perceiving racism. The final study revealed that there were no direct effects of perceived racism but rather that its influence

on blood pressure reactivity depends on how anger is typically expressed. High levels of both outward expression and suppression of anger may serve as protective factors in individuals who are exposed to a high degree of stress of racial discrimination.

As can be seen from these studies and those reviewed in the general introduction, the relation between racial discrimination and cardiovascular disease has been examined from several approaches. Taken together, racial differences in cardiovascular disease are likely the byproduct of a complex interaction of variables over time. However, the multiple pathways through which racism relates to cardiovascular outcomes have mostly been examined in isolation. Myers (2009) proposed a comprehensive model (Figure 1) that integrates the separate literatures on the biological, behavioural, psychosocial, and cultural rationales for racial differences in health outcomes. His model is based on the work of several stress, discrimination, and life-course theorists (McEwen 1998, 2004; Elder and Crosnoe 2002; Wickrama et al. 1999; Singer and Riff 1999; Geronimus 1992; Clark et al., 1999; Gallo & Matthews, 2003; Smedley et al., 2003; Brondolo et al., 2003; Paradies, 2006; Mays et al., 2007; Hertzman, 2004; Lu & Halfon, 2003). The model proposes that racial disparities are the result of the complex interactions between race and SES factors. These affect health through six pathways: (1) long-term chronic exposure to stress, (2) psychosocial reserve capacity, (3) cognitive-emotional processing, (4) health promoting and injurious behaviors, (5) biological, and (6) health care. It is proposed that these pathways intersect in recurrent and reciprocal ways over time to culminate in biopsychosocial vulnerabilities that shape the health trajectory of disadvantaged racial minorities.

Previous research and findings from these three studies have tested the individual components of Myers' (2009) model. The model proposes that race and SES interact (path A) over the lifespan to influence health by shaping exposure to psychosocial adversities (path B). Results from Study One contribute to the evidence supporting this path. Research findings indicate that individuals from low SES, particularly racial minorities, report a greater number of negative life events and more frequent exposure to life stressors (i.e. financial, occupational, relationships, parental, etc.; Gallo and Matthews 2003). They also perceive general life adversities to be more stressful and report greater psychological distress from these experiences (Collins et al. 1998; Chen and Matthews 2001) than do White individuals. The effects of race and SES are further worsened by the psychosocial adversity of racial discrimination and Study

One demonstrates that perceived racism is related to hypertension. As will be discussed further, in addition to adding to overall stress burden, racism may also exacerbate the impact of other life stresses by limiting economic opportunities which further worsen health outcomes. Further research on institutional forms of racism is needed to fully understand path B.

Research suggests that exposure to psychosocial adversities influences health through the dysregulation of a variety of biological mechanisms. Findings on racial group differences in biological vulnerabilities for adverse health outcomes have been reviewed. Study Two adds to the biological stress process pathway in Myers' (2009) model. It also showed that exaggerated responses to stress in Blacks, predictive of future risk, exist in a macro-environment where Blacks are less economically disadvantaged in Canada than in the U.S. Further research is needed to better articulate the relation between SES, race, and race differences in reactivity to stress.

As suggested in Myers' (2009) model, health disparities have been shown to be partly explained by low SES and negative cognitive-emotional factors such as anger, anxiety, depression, hopelessness, etc. Economically disadvantaged individuals are exposed to more stressors and are more likely to feel those negative emotions which have, in turn, have been associated with negative health outcomes. Study Three contributes to this body of evidence, showing how the stress of racial discrimination is related to anger expression to affect blood pressure.

The next step in understanding the multifaceted nature of the relation between racism and cardiovascular health would be to look at these pathways all together. Future research should create data sets that include multiple measures of racism, SES, and potential psychosocial and biological factors. Structural equation modeling would facilitate testing these multiple direct and indirect pathways that drive racial disparities.

Methodological issues

Looking at the results of the numerous studies conducted in the area suggests that racial discrimination is indeed related to cardiovascular functioning, however the effect sizes reported are relatively small. Findings from the meta-analysis revealed that perceived discrimination accounted for 5% of the variance in hypertensive status in a total sample size of 18, 987 individuals and was not significantly related to resting blood pressure. Brondolo, Love,

Pencille, Schoenthaler, & Ogedegbe (2011) also described the relation as small. Given the amount of attention racism has received in this literature, it would be expected to have found a larger effect size. However, several methodological factors make it difficult to estimate the true effect of racism. These include the measurement of racism, the overlap between SES and racism, and additional moderating factors.

Measuring racism

Quantifying exposure to racist experiences is a major difficulty in this area of research. First, Blacks tend to underreport racism either possibly because they tend to err on the side of minimizing, or not seeing, discrimination when it is directed at themselves. Studies have also shown that people report that they personally experience less discrimination than does the average member of their social group (Taylor, Wright, & Porter 1994). As well, respondents often avoid classifying their negative experiences as racist, even when these experiences qualify as such (Vorauer and Kumhyr 2001).

Estimating the impact of racism is also difficult because it is oftentimes invisible. Progress has been made on reducing overt racism, which involves publically endorsing the view of White superiority. In spite of this, evidence suggests that racial attitudes persist in contemporary society. Americans show high levels of negative feelings and beliefs about Blacks, Latinos, obese people, and homosexuals (Nosek et al., 2007). A study having looked at word pairs showed that the word black was associated with poor, violent, religious, lazy, cheerful, dangerous, charming, merry, ignorant and musical (Verhaeghen, Aikman, & Van Gulick, 2011). Even some individuals who support antiracist attitudes may also hold unconscious racial biases which cause them to behave in a racially biased manner (Dovidio, 2001; Quillian, 2008). Nosek et al. (2007) showed that nearly 70% of Americans have implicit biases that favor Whites over Blacks. These implicit biases make discrimination common, but through behaviors that the perpetrator does not experience as intentional (Dovidio & Gaertner, 2004). Racial microaggression, or sometimes referred to as everyday discrimination, is more subtle and ambiguous than other forms of racism (Sue et al., 2007). Microaggressions are chronic, everyday, routine, minor expressions of racism on an interpersonal level. They include "mild" assaults such as name calling, racial slurs which are communications that are perceived as rude, offensive, or insulting. These are conscious or unconscious and stem from the prejudicial

beliefs of the perpetrator. The intent of the perpetrator may, however, be difficult to discern. Individuals are more likely to allow prejudicial attitudes to affect their behaviors in ambiguous situations where multiple explanations are possible (Dovidio, 2001). This way, the perpetrator can provide other plausible explanations without having to reveal their racial attitudes (i.e., I pulled her over because she was driving irresponsibly, not because she was Black). Therefore, contemporary forms of racism are subtle manifestations of racial prejudice which are difficult to uncover. Despite the fact that racist acts are now more subtle and difficult to identify as racism, they continue to act as a stressor. Noh, Kaspar, and Wickrama (2007) found that individuals who experienced subtle, but not overt racial discrimination, were more likely to experience emotional arousal such as sadness, anger, aggression, and cognitive appraisal reactions such as experiencing exclusion, powerlessness, and shame. Blascovitch, Spencer, Quinn, & Steele (2001) also found that the activation of stigma of inferiority caused increases in blood pressure in Black but not White students.

Internalized racism, due to its very nature, is extremely difficult to measure. Internalized racism is the incorporation of racist attitudes, beliefs, or ideologies into an individual's worldview. Blacks who internalize racism accept the hierarchal stratification of race that places them at the bottom and the negative stereotypes about their abilities and intrinsic worth, such as being "physically gifted" and "mentally inferior" (Jones, 2000). Importantly, it can go unnoticed by those suffering from it. It is self-degrading and self-alienating (Watts-Jones, 2002). It is difficult to capture one's experience of discrimination given that internalized racism is so ingrained into one's world view. Internalized racism has not been well researched with respect to hypertension. Of the few studies that have looked at it, internalized racism is related to cardiovascular risk factors in women, but not men (Chambers, et al., 2004; Tull, Cort, Gwebu, & Gwebu, 2007). Chae, Lincoln, Adler, and Syme (2010) found that internalized racism interacted with perceived discrimination to affect cardiovascular disease risk. It has also been associated with perceived stress among Black women (Tull, Sheu, Butler, and Cornelious, 2005).

Blacks therefore may have difficulty identifying racism due to its subtle nature and because they have internalized the negative stereotypes. Self-report questionnaires such as those used in studies included in the meta-analysis and in Studies Two and Three likely underestimate the magnitude of discrimination. Furthermore, reporting discrimination on questionnaires has been shown to vary by sociodemographic variables, such as SES and age, though the direction of

this effect is not agreed upon in the literature (Paradies, 2006). Men generally report racism more than women do (Paradies, 2006). Taken together, the effect size of the relation between racism and hypertension found in Study One may be an under estimate due to these methodological factors and the fact that the study sample was mostly women (67%).

Overlap between SES and racism

Teasing out the effects of racism from SES is also a major challenge in this research area. Isaacs and Schroeder (2004) argued that looking at racial disparities without controlling for SES can attribute too much influence to race rather than low SES that is more associated with poor health outcomes. Adler & Rehkopf (2008) did find that socioeconomic measures accounted for much of the racial differences in health outcomes, however independent effects of race are also observed. Though most of the racial differences observed in cardiovascular outcomes are accounted for statistically by SES, SES itself is largely affected by institutional racism. Institutional racism limits resources and economic opportunities and can "cause" SES. Institutional racism continues to be widespread and often difficult to identify as racism. Blacks earn less than Whites at comparable years of schooling and Blacks have less purchasing power at any given level of income (Pamuk et al., 1998). Race-based residential segregation is often used as an index of institutional racism. Segregation was enforced in the late 19th and early 20th century in the U.S. to create racially distinctive neighborhoods and limit the closeness of Blacks to White homes (Cell, 1982). Despite the Fair Housing Act of 1968 in the U.S. having made residential segregation illegal, the racial distribution of housing has remained surprisingly stable with only small declines in recent years (Glaeser & Vigdor, 2001; Lieberson, 1980; Massey & Denton, 1993). The overrepresentation of Blacks and other minorities in the U.S. prison system is another example of institutional racism. High rates of incarceration in Blacks are driven by racial differences in the criminalization and investigation of certain behaviors and discrimination in prosecution and sentencing (Alexander, 2010).

Findings from Study One lend support this idea. Interpersonal forms of racism within institutional settings yielded a stronger relationship with hypertension as well as resting blood pressure that does general interpersonal racism. In a qualitative review, Brondolo, Love, Pencille, Schoenthaler, and Ogedegbe (2011) wrote that research suggests that racism is related

to hypertension and that these findings were more consistently observed in institutional racism than in interpersonal racism.

Altogether, the finding that economic disadvantage fully explains the observed inequalities in cardiovascular outcomes can be erroneous. SES may be a proxy for institutional racism, or at the least, is greatly affected by it. Myers (2009) argues that health disparities are best conceptualized by considering race and SES as bidirectional factors rather than affecting health alone. In research on racism, controlling for the effects of SES may mask some of the effects caused by racism itself. Most of the research on discrimination measures the perception of interpersonal racism and therefore only captures a subset of the oppressed experience. Thus, the 5% estimated effect found in Study One may be an underestimate of the true impact of racism on cardiovascular health.

Moderating factors

Studies conducted in this area oftentimes fail to take into account other psychosocial factors when estimating the influence of perceived racism on cardiovascular outcomes. The relation between perceived discrimination and blood pressure may not be readily apparent when simply looking at their direct relationship. Myers' (2009) model emphasizes that race and SES impact health through several pathways, namely long-term chronic exposure to stress, such as the stress of racial discrimination, and cognitive-emotional processing. Consistent with this, in Study 3, perceived racism was not found to predict cardiovascular reactivity to stress directly, but only in interaction with anger expression. Thus, results from Study Three demonstrate that direct effects of perceiving racism were not found, but rather its effects on cardiovascular reactivity depends on one's habitual style of expressing anger. Had anger expression not be considered, a relation between perceived racism and hypertension would not have been apparent. Most studies in this area control for traditional risk factors, but rarely look at interacting psychosocial factors. The research findings summarized in Study One therefore may not have captured the full effects of perceived racism. The aggregate findings that perceived racism accounts for 5% of the variance in hypertensive status may therefore be an underestimate of the true effect.

Several authors having written qualitative and quantitative reviews on the impact of racism on health outcomes have suggested that this relationship is likely moderated by individual differences (Brondolo, Love, Pencille, Schoenthaler, & Ogedegbe, 2011; Harrell, Hall, & Taliaferro, 2003, Pascoe and Smart Richman, 2009). Clark (2003) found that those who perceived a greater degree of racism and who had higher levels of social support showed greater reactivity to stress. Clark and Gochett (2006) reported that perceived racism interacted with coping responses, "accepting it" and "talking to someone", in predicting resting blood pressure in Black youth. In both these studies, social support only acted as a buffer in those who were exposed to low levels of perceived racism. Accordingly, perceived racism considered in isolation may not be critical, but is important to cardiovascular health when considered in interaction with other psychosocial variables. Several other factors have been shown to relate to blood pressure among Blacks. It would be useful to examine how these relate to racism to impact blood pressure functioning. Daniels, Harrell, Floyd, and Bell (2001) found that blood pressure was higher among Black men with stronger mainstream American cultural values of materialism, individualism, and competition. Dressler (1990) and Dressler and Bindon (2000) found that individuals who report materialistic aspirations beyond their means or espouse cultural values that differ from community consensus tend to have elevated blood pressure levels. The effects of such values may be compounded in individuals who perceive a large degree of racism and blocked economic opportunities due to discrimination. Further research on the interrelationship of these variables is necessary. Building on Clark (2003) and Clark and Gochett's (2006) findings in the area of coping would also be helpful. McNeilly et al. (1995) reported that support from a confederate did not reduce cardiovascular reactivity to racist provocation in a laboratory setting but did reduce self-reported anger. This finding further supports the need to look at the impact of racism on health from a structural equation modeling perspective to better understand these complex relationships. Spirituality, racial identity, emotional responses to racism are other avenues that should be examined in relation to perceived racism. Thus, a more comprehensive assessment of the psychosocial variables involved in the experience of racism, specifically how the perception of racism interacts with other factors, is necessary to better understand the impact of racism on cardiovascular health.

Future directions and conclusions

In addition to examining the effects of racism in a comprehensive framework, as proposed by Myers (2009), future research should also aim to address these methodological issues. Research on the role of internalized racism on blood pressure may shed more light on this area. There is also a need to develop measures of exposure to racism independent of self-report. As discussed above, more research is needed on institutional racism and its relation to cardiovascular health.

Another consideration for future studies is the use of older samples. The majority of research in this area uses younger samples. The average age of participants in the studies included in the meta-analysis was 38. Moderator analyses revealed that the relation between discrimination and hypertension was stronger among older participants. Given that the effects of discrimination are posited to be cumulative over the course of one's life time, a larger effect will likely be observed in older samples.

Much more research on factors that mitigate or exacerbate the effects of racism is critical. Study Three demonstrated that anger expressive styles interact with perceived racism to influence blood pressure but much more research is needed to fully articulate this relationship. Research on other factors, such as coping, social support, and other psychosocial variables is needed.

In conclusion, results from these studies add to the body of evidence that support the hypothesis that racism may help explain the increased risk of hypertension and cardiovascular disease among North American Blacks relative to other racial groups. Research findings reveal a small effect size between racism and cardiovascular functioning. Methodological issues that make the experience of racism difficult to quantify accurately and the multiplicity of the variables involved also contribute to the difficulty of accurately estimating its effect on health.

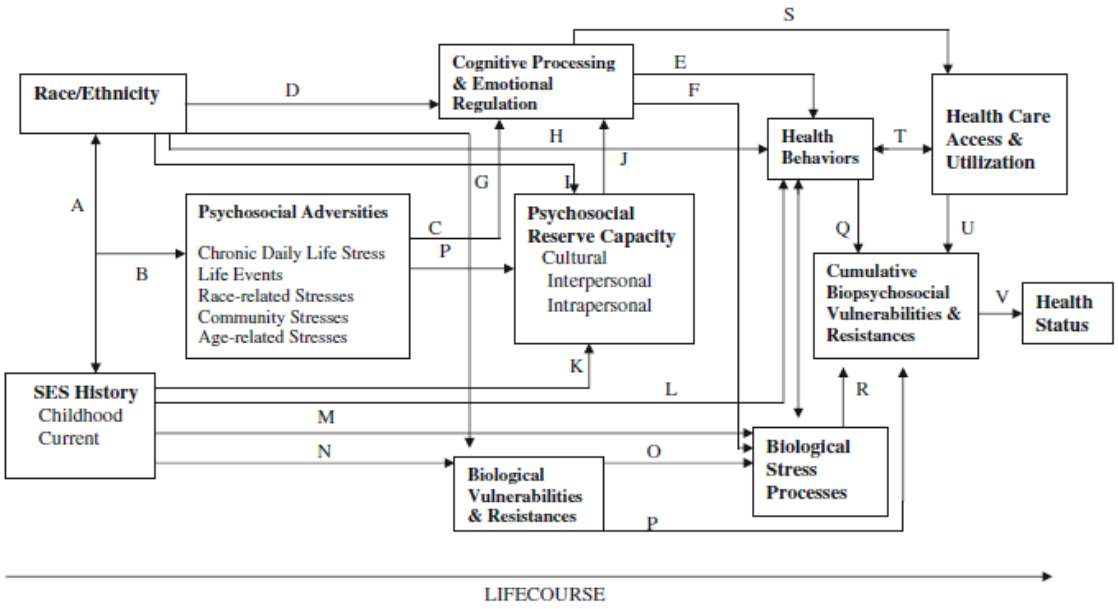


Figure 1. Myers' (2009) comprehensive biopsychosocial model.

References

Abel J. L., Larkin, K. T., & Edens, J. L. (1995). Women, anger, and cardiovascular responses to stress. *Journal of Psychosomatic Research, 39*, 251–259.

Adler, N. E., & Snibbe, A. C. (2003). The role of psychosocial processes in explaining the gradient between socioeconomic status and health. *Current Directions in Psychological Science, 12,* 119-123.

Adler, N. E., & Rehkopf, D. H. (2008). U.S. disparities in health: Descriptions, causes, and mechanisms. *Annual Review of Public Health, 29*, 235-252.

al'Absi, M., Devereux, R. B., Rao, D. C., Kitzman, D., Oberman, A., Hopkins, P., & Arnett, D. K. (2006). Blood pressure stress reactivity and left ventricular mass in a random community sample of African-American and Caucasian men and women. *American Journal of Cardiology*, *97*(2), 240-244.

Al'Absi, M., & Bongard, S. (2006). Neuroendocrine and behavioral mechanisms mediating the relationship between anger expression and cardiovascular risk: Assessment considerations and improvements. *Journal of Behavioral Medicine*, *29*(6), 573-591.

Alexander, M. (2010*) The New Jim Crow: Mass Incarceration in the Age of Colorblindness.* New York, NY: The New Press.

Allen, M. T., Obrist, P. A., Sherwood, A., & Crowell, M. D. (1987). Evaluation of myocardial and peripheral vascular responses during reaction time, mental arithmetic, and cold pressor tasks. *Psychophysiology, 24*, 648–656.

Allen, M. T., & Crowell, M. D. (1989). Patterns of autonomic response during laboratory stressors. *Psychophysiology, 26*, 603-614.

Allen, M. T., Boquet, A. J. Jr., & Shelley, K. S. (1991). Cluster analyses of cardiovascular responsivity to three laboratory stressors. *Psychosomatic Medicine*, 53(3), 272-88.

Alonso, A., Beunza, J. J., Delgado-Rodríguez, M., & Martínez-González, M. A. (2005). Validation of self reported diagnosis of hypertension in a cohort of university graduates in Spain. BMC Public Health, 5, 94-101.

American Heart Association. (2005). Heart Disease and Stroke Statistics - 2005 Update. Dallas, Texas: American Heart Association.

American Heart Association. (2008). *Heart Disease and Stroke Statistics - 2008 Update*. Dallas, Texas: American Heart Association.

Anderson, N. B., Lane, J. D., Muranaka, M., Williams, R. B., & Houseworth, S. J. (1988a). Racial differences in blood pressure and forearm vascular responses to the cold face stimulus. *Psychosomatic Medicine, 50,* 57-63.

Anderson, N. B., Lane, J. D., Monou, H., Williams, R. B. J., & Houseworth, S. A. (1988b). Racial differences in cardiovascular reactivity to mental arithmetic. *International Journal of Psychophysiology, 6*, 161-164.

Anderson, N. B., McNeilly, M., & Myers, H. (1991). Autonomic reactivity and hypertension in blacks: A review and proposed model. *Ethnicity & Disease, 1*(2), 154–170.

Anderson, N., McNeilly, M., & Myers, H. F. (1992). Toward understanding race differences in autonomic reactivity. In J. R. Turner, A. Sherwood, & K. C. Light (Eds.), *Individual differences in cardiovascular response to stress: Perspectives on individual differences*, (pp. 125-145). New York, NY: Plenum Press.

Armstead, C. A., Lawler, K. A., Gordon, G., Cross, J., & Gibbons, J. (1989). Relationship of racial stressors to blood pressure responses and anger expression in black college students. *Health Psychology, 8,* 541–556.

Attewell, P., Kasinitz, P., & Dunn, K. (2010). Black Canadians and Black Americans: Racial income inequality in comparative perspective. *Ethnic and Racial Studies, 33*, 473-495.

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.

Barefoot, J. C., Peterson, B. L., Dahlstrom, W. G., Siegler, I. C., Anderson, N.B., & Williams, R. B. (1991). Hostility patterns and health implications: Correlates of the Cook–Medley Hostility Scale scores in a national survey. *Health Psychology, 10,* 18–24.

Baum, A., Cohen, L., & Hall, M. (1993). Control and intrusive memories as possible determinants of chronic stress. *Psychosomatic Medicine, 55,* 274–286.

Baumeister, R. F., DeWall, C. N., Ciarocco, N. J., & Twenge, J. M. (2005). Social exclusion impairs self-regulation. *Journal of Personality and Social Psychology, 88*, 589–604. doi:10.1037/ 0022-3514.88.4.589.

Becker, L.C., Pepine, C. J., Bonsall, R., Cohen, J.D., Goldberg, A.D., Coghlan, C., … Kaufmann, P. G. (1996). Left ventricular, peripheral vascular, and neurohumoral responses to mental stress in normal middle-aged men and women. Reference group for the psychophysiological

investigations of myocardial ischemia (PIMI) study. *Circulation, 94*, 2768–2777.

Berman, G., & Paradies, Y. (2010). Racism, disadvantage and multiculturalism: towards effective anti-racist praxis. *Ethnic & Racial Studies, 33,* 214–232.

Bishop, G. C., Ngau, F., & Pek, J. (2008). Domain-specific assessment of anger expression and ambulatory blood pressure. *Personality and Individual Differences, 44*, 1726-1737.

Blank, R. M., Dabady, M., & Citro, C. F. (2004). *Measuring racial discrimination.* Washington, DC: National Academy Press.

Bloemraad, I. (2006). *Becoming a citizen: incorporating immigrants and refugees in the United States and Canada*. California: University of California Press.

Bond, M. A., Kalaja, A., Markkanen, P., Cazeca, D., Daniel, S., Tsurikova, L. … Baron, S. (2007). Expanding our understanding of the psychosocial work environment: A Compendium of Measures of Discrimination, Harassment, and Work-Family Issues (2008-104). Cincinnati, OH: Department of Health and Human Services, Centers for Disease Control and Prevention, National Institute for Occupational Safety and Health, University of Massachusetts at Lowell, & Center for Women.

Bonilla-Silva, E. (1996). Rethinking racism: Toward a structural interpretation. *American Sociological Review, 62,* 465–480. doi:10.2307/2657316.

Borrell, L. N., Jacobs, D. R. Jr., Williams, D. R., Pletcher, M. J., Houston, T. K., & Kiefe, C. I. (2007). Self-reported racial discrimination and substance use in the coronary artery risk development in adults study. *American Journal of Epidemiology, 166*, 1068–1079. doi:10.1093/aje/kwm180.

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,* 18-36.

Braveman, P., Egerter, S., & Williams, D. R. (2011). The social determinants of health: Coming of age. *Annual Review of Public Health, 32,* 381–398.

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

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

Brondolo, E., Kelly, K. P., Coakley, V., Gordon, T., Thompson, S., Levy, E., … Contrada, R. J.

(2005). The perceived ethnic discrimination questionnaire: Development and preliminary validation of a community version. *Journal of Applied Social Psychology, 35*(2), 335-365.

Brondolo, E., Libby, D. J., Denton, E. G., Thompson, S., Beatty, D. L., Schwartz, J., … Guerin, W. (2008). Racism and ambulatory blood pressure in a community sample. *Psychosomatic Medicine, 70,* 49-56.

Brondolo, E., Brady ver Halen, N., Pencille, M., Beatty, D., & Contrada, R. (2009). Coping with racism: A selective review of the literature and a theoretical and methodological critique. *Journal of Behavioral Medicine, 32,* 64-88.

Brondolo, E., Gallo, L., & Myers, H. (2009). Race, racism and health: Disparities, mechanisms, and interventions. *Journal of Behavioral Medicine, 32,* 1-8.

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

Brondolo, E., Hausmann, L. R. M., Jhalani, J., Pencille, M., Atencio-Bacayon, J., Kumar, A., … Schwartz, J. (2011). Dimensions of perceived racism and self-reported health: Examination of racial/ethnic differences and potential mediators. *Annals of Behavioural Medicine, 42,* 14–28.

Brondolo, E., Brady, N., Libby, D., & Pencille, M. (2011). Racism as a psychosocial stressor. A. Baum & R. J. Contrada (Eds.), *Handbook of stress science* (pp. 167–184). New York: Springer.

Brownley, K. A., Light, K. C., & Anderson, N. B. (1996). Social support and hostility interact to influence clinic, work, and home blood pressure in black and white men and women. *Psychophysiology, 33,* 434-445.

Brown, C., Matthews, K. A., Bromberger, J. T., & 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,* 257-262.

Bullock, S. C., & Houston, E. (1987). Perceptions of racism by black medical students attending white medical schools. *Journal of the National Medical Association, 79,* 601–608.

Burt, V. L., Whelton, P., Roccella, E. J., Brown, C., Cutler, J. A., Higgins, M., … Labarthe, D. (1995). Prevalence of Hypertension in the US Adult Population: Results From the Third National Health and Nutrition Examination Survey, 1988-1991. *Hypertension, 25,* 305-313.

Bush, D. E., Ziegelstein, R. C., Tayback, M., Richter, D., Stevens, S., Zahalsky, H., & Fauerbach, J. A. (2001). Even minimal symptoms of depression increase mortality risk after acute myocardial infarction. *American Journal of Cardiology*, *88*(4), 337-341. doi: 10.1016/S0002-9149(01)01675-7.

Cacioppo, J. T., Hawkley, L. C., & Berntson, G. G. (2003). The anatomy of loneliness. *Current Directions in Psychological Science, 12,* 71–74.

Calhoun, D. A. (1992). Hypertension in blacks: socioeconomic stress and sympathetic nervous system activity. *The American Journal of the Medical Sciences, 304*(5), 306–311.

Carroll, D. (2003). Blood pressure reactions to stress and the prediction of future blood pressure: Effects of sex, age, and socioeconomic position. *Psychosomatic Medicine, 65,* 1058-1064.

Carter, R. T. (2007). Racism and psychological and emotional injury: Recognizing and assessing race-based traumatic stress. *The Counseling Psychiatrist, 35*(1), 13–105.

Casagrande, S. S., Gary, T. L., LaVeist, T. A., Gaskin, D. J., & Cooper, L. A. (2007). Perceived discrimination and adherence to medical care in a racially integrated community. *Journal of General Internal Medicine, 22*, 389–395. doi:10.1007/s11606-006-0057-4.

Cell, J. W. (1982) *The Highest Stage of White Supremacy: The Origin of Segregation in South Africa and the American South*. New York: Cambridge University Press.

Centers for Disease Control and Prevention. (2011). CDC Health Disparities and Inequalities Report - United States, 2011. *Morbidity and Mortality Weekly Report, 60(Supp),* 1-113.

Chambers, E. C., Tull, E. S., Fraser, H. S., Mutunhu, N. R., Sobers, N., & Niles, E. (2004). The relationship of internalized racism to body fat distribution and insulin resistance among African adolescent youth. *Journal of the National Medical Association, 96,* 1594–1598.

Chida Y, & Steptoe, A. (2009) The association of anger and hostility with future coronary heart disease: a meta-analytic review of prospective evidence. *Journal of the American College of Cardiology, 53*, 936-46.

Chida, Y., & Steptoe, A. (2010). Greater Cardiovascular Responses to Laboratory Mental Stress Are Associated With Poor Subsequent Cardiovascular Risk Status. *Hypertension, 55,* 1026-1032.

Chen, E., & Matthews, K. A. (2001). Cognitive appraisal biases: an approach to understanding the relation between socioeconomic status and cardiovascular reactivity in children *Annals of Behavioral Medicine, 23,* 101–111.

Chinn, S. A. (2000). A simple method for converting an odds ratio to effect size for use in meta-analysis. *Statistics in Medicine, 19,* 3127-3131.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., … National High Blood Pressure Education Program Coordinating Committee. (2003). Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: The JNC 7 Complete Report. *Hypertension, 42,* 1206–1252.

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,* 805-816. doi:10.1037/ 0003-066X.54.10.805.

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), 214-22.

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

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.

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

Clark, R. (2006b). Perceived racism and vascular reactivity in black college women: Moderating Effects of seeking social support. *Health Psychology, 25,* 1, 20–25.

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,* 1-9.

Clark, V. R., Greenberg, B., Harris, T., & Carson, B. L. (2012). Body mass index and waist circumference predictors of cardiovascular risk in African Americans. *Ethnicity and Disease, 22,* 162-167.

Collins, J. W., David, R. J., Symons, R., Handler, A., Wall, S., & Andes, S. (1998). African-American mothers' perceptions of their residential environment, stressful life events, and very low birthweight. *Epidemiology (Cambridge, MA), 9(3),* 286–289. doi:10.1097/00001648-199805000-00012.

Combs, D. R., Penn, D. L., Cassisi, J., Michael, C., Wood, T., Wanner, J., & Adams, D. (2006). Perceived Racism as a Predictor of Paranoia Among African Americans. *Journal of Black Psychology, 32,* 87-104.

Commission on Social Determinants of Health (2008) *Closing the gap in a generation: Health equity through action on the social determinants of health*. Final report. Geneva: World Health Organization.

Compare, A., Mommersteeg, P. M., Faletra, F., Grossi, E., Pasotti, E., Moccetti, T., & Auricchio, A. (2014). Personality traits, cardiac risk factors, and their association with presence and severity of coronary artery plaquein people with no history of cardiovascular disease. *Journal of Cardiovascular Medicine* (Hagerstown), *15*(5), 423-30. doi: 10.2459/JCM.0b013e328365cd8c.

Cooper, H. (1998). *Synthesizing Research*. Thousand Oaks, California: Sage Publications.

Cooper, H., & Hedges, L. V. (1994). *The Handbook of Research Synthesis*. New York: Russell Sage Foundation.

Cooper, R., Wolf-Maier, K., Luke, A., Adeyemo, A., Banegas, J., Forrester, T., ,,, Thamm, M. (2005). An international comparison of blood pressure in populations of European vs. African descent. *BMC Medicine, 3,* 2-10.

Cornoni-Huntley, J., LaCroix, A., & Havlik, R. (1989). Race and sex differentials in the impact of hypertension in the United States. The National Health and Nutrition Examination Survey I Epidemiologic Follow-up Study. *Archives of Internal Medicine, 149,* 780-788.

Cooper, R., Rotimi, C., Ataman, S., McGee, D., Osotimehin, B., Kadiri, S., … Wilks, R. (1997). The prevalence of hypertension in seven populations of West African origin. *American Journal of Public Health, 87*, 160-168.

Cooper, R., Wolf-Maier, K., Luke, A., Adeyemo, A., Banegas, J., Forrester, T. … Thamm, M. (2005). An international comparison of blood pressure in populations of European vs. African descent. *BMC Medicine, 3,* 2-10.

Cornish, P. J., Blanchard, E. B., & Jaccard, J. (1994). The relationship between 24-hour ambulatory blood pressures and laboratory measures of cardiovascular reactivity. *Biofeedback and Self Regulation, 19,* 193–209.

Cornoni-Huntley, J., LaCroix, A., & Havlik, R. (1989). Race and sex differentials in the impact of hypertension in the United States. The National Health and Nutrition Examination Survey I

Epidemiologic Follow-up Study. *Archives of Internal Medicine, 149,* 780-788.

Cose, E. (1993). *The Rage of a Privileged Class*. New York: Harper Collins.

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.

Crimmins, E. M., Kim, J. K., Alley, D. E., Karlamangla, A., & Seeman, T. (2007). Hispanic paradox in biological risk profiles. *American Journal of Public Health, 97,* 1305-1310.

Crocker J., Voelkl, K., Testa, M., & Major, B. (1991). Social stigma: The affective consequences of attributional ambiguity. *Journal of Personality and Social Psychology, 60,* 218–228.

Daniels, I.N., Harrell, J.P., Floyd, L.J., & Bell, S.R. (2001). Hostility, cultural orientation and casual blood pressure readings in African Americans. *Ethnicity and Disease, 11*, 779–787.

Davidson, K. W., Burg, M. M., Kronish, I. M., Shimbo, D., Dettenborn, L., Mehran, R., & Rieckmann, N. (2010). Association of anhedonia with recurrent major adverse cardiac events and mortality 1 year after acute coronary syndrome. *Archives of General Psychiatry*, *67*(5), 480-488. doi:10.1001/archgenpsychiatry.2010.36.

Davis, S. K., Liu, Y., Quarells, R. C., Din-Dzietham, R., & Metro Atlantic Heart Disease Study Group. (2005). Stress-related racial discrimination and hypertension likelihood in a population-based sample of African Americans: The Metro Atlanta Heart Disease Study. *Ethnicity and Disease, 15(4),* 585-593.

Delehanty, S., Dimsdale, J., & Mills, P. (1991). Psychosocial correlates of reactivity in black and white men. *Journal of Psychosomatic Research, 35,* 451-460.

Denollet, J., Gidron, Y., Vrints, C. J., & Conraads, V. M. (2010). Anger, suppressed anger, and risk of adverse events in patients with coronary artery disease. *American Journal of Cardiology, 105*(11), 1555-60. doi: 10.1016/j.amjcard.2010.01.015.

Devereux, R. B., Savage, D. D., Sachs, I., & Laragh, J. H. (1983). Relation of hemodynamic load to left ventricular hypertrophy and performance in hypertension. *American Journal of Cardiology, 51*, 171-176.

Dickens, C., McGowan, L., Percival, C., Tomenson, B., Cotter, L., Heagerty, A., & Creed, F. (2008). New onset depression following myocardial infarction predicts cardiac mortality. *Psychosomatic Medicine*, *70*(4), 450-455. Doi:10.1097/PSY.0b013e31816a74de.

Dimsdale, J. E., Ziegler, M., Mills, P., Delehanty, S. G., & Berry, C. (1990). Effects of salt, race,

and hypertension on reactivity to stressors. *Hypertension, 16,* 573-580.

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.

Dolezsar, C. M., McGrath, J., Herzig, A., & Miller, S. (2014). Perceived Racial Discrimination and Hypertension: A Comprehensive Systematic Review. *Health Psychology, 33*, 1, 20-34. doi: 10.1037/a0033718.

Dorr, N., Brosschot, J. F., Sollers, J. J. III, & Thayer, J. F. (2007). Damned if you do, damned if you don't: The differential effect of expression and inhibition of anger on cardiovascular recovery in Black and White males. *International Journal of Psychophysiology, 66,* 125-134.

Dovidio, J. F. (2001). On the nature of contemporary prejudice: The third wave. *Journal of Social Issues, 57,* 829–849. doi:10.1111/0022-4537.00244.

Dressler W.W. (1990). Lifestyle, stress, and blood pressure in a southern black community. *Psychosomatic Medicine*, 52, 182–198.

Dressler W.W. & Bindon, J.R. (2000). The health consequences of cultural consonance: cultural dimensions of lifestyle, social support, and arterial blood pressure in an African American community. *American Anthropologist, 102,* 244–260.

Dressler, W. W., Oths, K. S., & Gravlee, C. C. (2005). Race and ethnicity in public health research: models to explain health disparities. *Annual Review of Anthropology, 34*, 231–252.

Elder, G. H., Jr., & Crosnoe, R. (2002). The influence of early behavior patterns on later life. In L. Pulkkinen & A. Caspi (Eds.), *Paths to successful development: Personality in the life course* (pp. 157–176). New York: Cambridge University Press.

Elliott, H. L., Sumner, D. J., McLean, K., & Reid, J. L. (1982) Effect of age on the responsiveness of vascular alpha-adrenoceptors in man. *Journal of Cardiovascular Pharmacology, 4*, 388 – 392.

Etowa, J., Keddy, B., Egbeyemi, J., & Eghan, F. (2007) Depression: "The Invisible Grey Fog" Influencing the midlife health of African Canadian women. *International Journal of Mental Health Nursing*, *16*(3), 203-213. doi:10.1111/j.1447-0349.2007.00469.x

Ewart, C.K. & Kolodnar, K.B. (1991). Social competence interview for assessing

physiological reactivity in adolescents. Psychosomatic Medicine, 53, 289-304.

Falkner, B. (1996). The role of cardiovascular reactivity as a mediator of hypertension in African Americans. *Seminar in Nephrology, 16*(2), 117–125.

Fang, C. W., & Myers, H. F. (2001). The effects of racial stressors and hostility on cardiovascular reactivity in African American and Caucasian men. *Health Psychology, 20,* 64-70.

Feagin, J. R., & Sikes, M. P. (1994). *Living with Racism: The Black Middle-Class Experience*. Boston, MA: Beacon.

Feagin, J. R., & McKinney, K. D. (2003). *The many costs of racism*. Lanham, MD: Rowman & Littlefield.

Ferdinand, K. C., & Armani, A. M. (2007). The management of hypertension in African Americans. *Critical Pathways in Cardiology, 6*, 67–71.

Fiedorowicz, J. G. (2014). Depression and cardiovascular disease: an update on how course of illness may influence risk. *Current Psychiatry Reports, 16*(10), 492-504. doi: 10.1007/s11920-014-0492-6.

Fields, B., Reesman, K., Robinson, C., Sims, A., Edwards, K., McCall, B., … Thomas, S. P. (1998). Anger of African American women in the south. *Issues in Mental Health Nursing 19,* 353-373.

Fields, L. E., Burt, V. L., Cutler, J. A., Hughes, J., Roccella, E. J., & Sorlie, P. (2004). The burden of adult hypertension in the United States 1999 to 2000: A rising tide. *Hypertension*, *44*, 398-404.

Finch, B. K., Kolody, B., & Vega, W. A. (2000). Perceived discrimination and depression among Mexican-origin adults in California. *Hispanic Journal of Health and Social Behavior, 41,* 295-313.

Finney, M. L., Stoney, C. M., & Engebretson, T. O (2002) Hostility and anger expression in African American and European American men is associated with cardiovascular and lipid reactivity. *Psychophysiology, 38*, 340-349.

Flack, J. M., & Wiist, W. H. (1995). Epidemiology of hypertension and hypertensive target-organ damage in the United States. *Journal of the Association for Academic Minority Physicians, 2,* 143-150.

Frankenhaeuser, M., von Wright, M. R., Collins, A., von Wright, J., Sedvall, G., & Swahn, C.G. (1978). Sex difference in psychoneuroendocrine reactions to examination stress.

Psychosomatic Medicine, 40, 334–343.

Frazer, N., Larkin, K., & Goodie, J. (2002). Do behavioral responses mediate or moderate the relation between cardiovascular reactivity to stress and parental history of hypertension? *Health Psychology, 21,* 244-253.

Frasure-Smith, N., & Lesperance, F. (2008). Depression and anxiety as predictors of 2-year cardiac events in patients with stable coronary artery disease. *Archives of General Psychiatry*, *65*(1), 62-71. doi: 10.1001/archgenpsychiatry.2007.4.

Fredrikson, M., Robson, A., & Ljungdell, T. (1991). Ambulatory and laboratory blood pressure in individuals with negative and positive family history of hypertension. *Health Psychology, 10,* 371–377.

Fuqua, D.R., Leonard, E., Masters, M.A., Smith, R.J., Campbell, J.L., & Fischer, P.C. (1991). A structural analysis of the State–Trait Anger Expression Inventory. *Educational and Psychological Measurement, 51,* 439–446.

Fuster, V., & Pearson, T. A. (1996). Matching the intensity of risk factor management with the hazard for coronary disease events. *Journal of the American College of Cardiology, 27*, 957–1047.

Gallo, L. C., & Matthews, K. A. (2003). Understanding the association between socioeconomic status and physical health: Do negative emotions play a role? *Psychological Bulletin, 129,* 10-51.

Gee, G. C., Spencer, M. S., Chen, J., & Takeuchi, D. T. (2007). A nationwide study of discrimination and chronic health conditions among Asian Americans. *American Journal of Public Health*, *97*(7), 1275-1287.

Gentry, W. D. (1985) Relationship of anger-coping styles and blood pressure among Black Americans. In M. A. Chesney, & R. H. Rosenman (Eds.), *Anger and hostility in cardiovascular and behavioral disorders* (pp. 139–47). Washington (DC): Hemisphere.

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

Glaeser, E. L., & Vigdor, J. L. (2001) Racial segregation in the 2000 Census: Promising news. *The Brookings Institution, Survey Series Report*, 1–16.

Goldberg, A. D., Becker, L. C., Bonsall, R., Cohen, J. D., Ketterer, M. W., Kaufman, P. G. … Sheps, D. S. (1996). Ischemic, hemodynamic, and neurohormonal responses to mental and

exercise stress: Experience from the Psycho-physiological Investigations of Myocardial Ischemic Study (PIMI). *Circulation, 94*, 2402-2409.

Goodman, A. H. (2000). Why genes don't count (for racial differences in health). *American Journal of Public Health, 90,* 1699-1702.

Green, A. R., Carney, D. R., Pallin, D. J., Ngo, L. H., Raymond, K. L., Iezzoni, L. I., & Banaji, M. R. (2007). Implicit bias among physicians and its prediction of thrombolysis decisions for black and white patients. *Journal of General Internal Medicine, 22*(9), 1231–1238. doi:10.1007/s11606-007-0258-5.

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

Hahm, H. C., Ozonoff, A., Gaumond, J., & Sue, S. (2010). Perceived discrimination and health outcomes: A gender comparison among Asian-Americans nationwide. *Women's Health Issues, 20,* 350-358.

Hajjar, I., & Kotchen, T. A. (2003). Trends in prevalence, awareness, treatment, and control of hypertension in the United States, 1988-2000. *The Journal of the American Medical Association, 290,* 199- 206.

Hall, J. L., Duprez, D. A., Barac, A., Rich, S. S. (2012). A review of genetics, arterial stiffness, and blood pressure in African Americans. *Journal of Cardiovascular Translational Research, 5*(3), 302-8. doi: 10.1007/s12265-012-9362-y.

Hall, S. P., & Carter, R. T. (2006). The Relationship Between Racial Identity, Ethnic Identity, and Perceptions of Racial Discrimination in an Afro-Caribbean Descent Sample. *Journal of Black Psychology, 32,* 155-175.

Hall, W. D., Ferrario, C. M., Moore, M. A., Hall, J. E., Flack, J. M., Cooper, W., … Roccella, E. J. (1997). Hypertension-related morbidity and mortality in the southeastern United States. *American Journal of the Medical Sciences, 313*, 195–206.

Harburg, E., Erfurt, J. C., Hauenstein, L. S., Chape, C., Schull, W. J., & Schork, M. A. (1973). Socio-ecological stress, suppressed hostility, skin color, and black-white male blood pressure: Detroit. *Psychosomatic Medicine, 35*, 276–296.

Harburg, E., Blakelock, E. H., & Roeper, P. J. (1979). Resentful and reflective coping with arbitrary authority and blood pressure: Detroit. *Psychosomatic Medicine, 41,* 189–202.

Harrell, J. P., Hall, S., & Taliaferro, J. (2003). Physiological responses to racism and discrimination: An assessment of the evidence. *American Journal of Public Health, 93,* 243-248.

Harrell, J. 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*,143–157.

Harrell, S. P. (1997). *The Racism and Life Experiences Scales (RaLES)* (Unpublished manuscript). California School of Professional Psychology, Los Angeles, California.

Harrell, S. P., Merchant, M. A., & Young, S. A. (1997). *Psychometric properties of the Racism and Life Experience Scales (RaLES).* Poster presented at the American Psychological Association convention, Chicago: IL.

Haukkala, A., Konttinen, H. N., Laatkainen, T., & Kawachi, I. (2010). Hostility, anger control, and anger expression as predictors of cardiovascular disease. *Psychosomatic Medicine, 72,* 556-562.

Hausmann, L. R., Jeong, K., Bost, J. E., & Ibrahim, S. A. (2008). Perceived discrimination in health care and use of preventive health services. *Journal of General Internal Medicine, 23*, 1679–1684.

Haynes, S. G., Feinleib, M., & Kennel, W. B. (1980). The relationship of psychosocial factors to coronary heart disease in the Framingham study. III. Eight-year incidence of coronary heart disease. *American Journal of Epidemiology, 111,* 37–58.

Hayward, M. D., Crimmins, E. M., Miles, T. P., & Yang, Y. (2000). The significance of socioeconomic status in explaining the racial gap in chronic health conditions. *American Sociological Review, 65*, 910-930.

Heponiemi, T., Keltikangas-Ja¨rvinen, L., Kettunen, J., Puttonen, S., & Ravaja, N. (2004). BIS-BAS sensitivity and cardiac autonomic stress profiles. *Psychophysiology, 41*, 37–45.

Hermann, V., Buscher, R., Go, M. M., Ring, K. M., Hofer, J. K., Kailasam, M. T., … Insel, P. A. (2000). Beta2-adrenergic receptor polymorphisms at codon 16, cardiovascular phenotypes and essential hypertension in whites and African Americans. *American Journal of Hypertension, 13,* 1021–6.

Hertz, R. P., Unger, A. N., Cornell, J. A., & Saunders, E. (2005). Racial disparities in hypertension prevalence, awareness, and management. *Archives of Internal Medicine, 165,* 2098-2104.

Hertzman, C. (2004). The life-course contribution to ethnic disparities in health. In N. B. Anderson, R. A. Bulatao, & B. Cohen (Eds.), *Critical perspectives on racial and ethnic differences in health in late life* (pp. 145–170). Washington, DC: The National Academic Press.

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.

Hicken, M., Gragg, R., & Hu, H. (2011). How cumulative risks warrant a shift in our approach to racial health disparities: The case of lead, stress, and hypertension. *Health Affairs, 30,* 1895-1901.

Hogan, B. E., & Linden, W. (2004). Anger response styles and blood pressure: At least don't ruminate about it! *Annals of Behavioral Medicine, 27,* 38–49.

Hogan. B. E., & Linden, W. (2005). Curvelinear relationship of expressed anger and blood pressure in women but not in men: evidence from two samples. *Journal of Psychosomatic Research, 59*, 97–102.

Hokanson, J. E., & Burgess, M. (1962). The effects of three types of aggression on vascular processes. *Journal of Abnormal and Social Psychology, 64*, 446-449.

Hosseini, S. H., Mokhberi, V., Mohammadpour, R. A., Mehrabianfard, M, & Lashak, N. B. (2011). Anger expression and suppression among patients with essential hypertension. *International Journal of Psychiatry in Clinical Practice, 15*(3), 214-8. doi: 10.3109/13651501.2011.572168.

Hou, F., & Picot, G. (2003). *Visible Minority Neighborhood Enclaves and Labour Market Outcomes Of Immigrants* (2003204e). Ottawa: Statistics Canada.

Hughes, B. M., & Higgins, N. M. (2010). Habituation-sensitization of cardiovascular reactivity to repeated stress in smokers and non-smokers: An anthropometrically matched trial. *International Journal of Psychophysiology, 76,* 34-39.

Huisman, H. W., Schutte, A. E., Schutte, R., van Rooyen, J. M., Fourie, C. M., Mels, C. M., … Malan, L. (2013). Exploring the link between cardiovascular reactivity and end-organ damage in African and Caucasian men: the SABPA study. *American Journal of Hypertension, 26*(1), 68-75. doi: 10.1093/ajh/hps007.

Inzlicht, M., McKay, L., & Aronson, J. (2006). Stigma as ego depletion: How being the target of prejudice affects self-control. *Psychological Science, 17,* 262–269. doi:10.1111/j.1467-

9280.2006.01695.x.

Ironson, G., Taylor, C. B., Boltwood, M., Bartzokis, T., Dennis, C., Chesney, M., … Segall, G. M. (1992). Effects of anger on left ventricular ejection fraction in coronary artery disease. *American Journal of Cardiology, 70,* 281-285.

Isaacs, S. L., & Schroeder, S. A. (2004). Class—the ignored determinant of the nation's health. *The New England Journal of Medicine, 351*(11), 1137–42.

James, S. A., LaCroix, A. Z., Kleinbaum, D. G., & Strogatz, D. S. (1984). John Henryism and blood pressure differences among black men II. *Journal of Behavioral Medicine, 7,* 259-275.

Jamieson, J. P., Koslov, K., Nock, M. K., & Berry Mendes, W. (2012). Experiencing discrimination increases risk taking. *Psychological Science, 24*(2) 131-139.

Johnson, E. H., Schork, N. J., & Spielberger, C. D. (1987). Emotional and familial determinants of elevated blood pressure in black and white adolescent females. *Journal of Psychosomatic Research, 31,* 731–741.

Jones, C. P. (2000). Levels of Racism: A Theoretic Framework and a Gardener's Tale. *American Journal of Public Health, 90,* 1212-1215. doi:10.2105/AJPH.90.8.1212.2001-06657-00110.2105/AJPH.90.8.121210936998.

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*, 109–22.

Jones, J. M. (1997). *Prejudice and racism.* New York, NY: McGraw-Hill Inc.

Kaholokula, J., Grandinetti, A., Keller, S., Nacapoy, A., Kingi, T., & Mau, M. (2012). Association between perceived racism and physiological stress indices in Hawaiians. *Journal of Behavioral Medicine, 35,* 27-37.

Kaiser, C. R., & Major, B. (2006). A social psychological perspective on perceiving and reporting discrimination. *Law and Social Inquiry, 31*, 801–830. doi: 10.1111/j.1747-4469.2006.00036.x.

Kajantie, E., & Phillips, D. I. W. (2005). The effects of sex and hormonal status on the physiological response to acute psychosocial stress. *Psychoneuroendocrinology, 31*, 151–178. doi: 10.1016/j.psyneuen.2005.07.002

Kamala, S., Thomas, M. S., Nelesen, R. A., Malcarne, V. L., Ziegler, M. G., & Dimsdale, J. E. (2006). Ethnicity, perceived discrimination, and vascular reactivity to phenylephrine.

Psychosomatic Medicine, 68, 692–697.

Kanayama, N., Khatun, S., Belayet, H., She, L., & Terao, T. (1999). Chronic local cold stress to the soles induces hypertension in rats. *American Journal of Hypertension, 12*, 1124–1129.

Kannel, W. B. (1995). Framingham study insights into hypertensive risk of cardiovascular disease. *Hypertension Research*, *18*(3), 181-196.

Kaplan, N. (1994). Ethnic aspects of hypertension. *Lancet, 344,* 450-452.

Kaplan, N. M. (1998). *Clinical hypertension* (7th ed.). Baltimore: Williams & Wilkins.

Karlsen, S., & Nazroo, J. (2002). Relation between racial discrimination, social class, and health among ethnic minority groups. *American Journal of Public Health, 92,* 624-631.

Kasagi, F., Akahoshi, M., & Shimaoka, K. (1995). Relation between cold pressor test and development of hypertension based on 28-year follow-up. *Hypertension, 25*, 71–76.

Kasprowicz, A. L., Manuck S. B., Malkoff , S. B., & Krantz, D. S. (1990). Individual differences in behaviorally evoked cardiovascular response: temporal stability and hemodynamic patterning. *Psychophysiology*, *27*(6)*,* 605-19.

Keawe'aimoku, J., Iwane, M., & Nacapoy, A. (2010). Effects of perceived racism and acculturation on hypertension in native Hawaiians. *Hawai'i Medical Journal, 69,* 11-15.

Kelsey, R. M., Alpert, B. S., Patterson, S. M., & Barnard, M. (2000a). Racial differences in hemodynamic responses to environmental thermal stress among adolescents. *Circulation, 101*, 2284–2289.

Kelsey, R. M., Patterson, S. M., Barnard, M., & Alpert, B. (2000b) Consistency of hemodynamic responses to cold stress in adolescents. *Hypertension, 36*(6), 1013-1017.

Kershaw, K. N., Diez, Roux A. V., Burgard, S. A., Lisabeth, L. D., Mujahid, M. S., & Schulz, A. J. (2011). Metropolitan-level racial residential segregation and Black-White disparities in hypertension. *American Journal of Epidemiology, 174,* 537-545.

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,* 208-230.

Khan, F., Kulaksizoglu, B., & Cilingiroglu, M. (2010). Depression and coronary heart disease. *Current Atherosclerosis Reports*, *12*(2), 105-109. doi: 10.1007/s11883-010-0096-5.

Klag, M. J., Whelton, P. K., Randall, B. L., Neaton, J. D., Brancati, F. L., & Stamler, J. (1997). End stage renal disease in African American and white men: 16-year MRFIT findings.

Journal of the American Medical Association (JAMA), *277*, 1293–8.

Klimentidis, Y. C., Dulin-Keita, A., Casazza, K., Willig, A. L., Allison, D. B., & Fernandez, J. R.. (2012). Genetic admixture, social-behavioural factors and body composition are associated with blood pressure differently by racial-ethnic group among children. *Journal of Human Hypertension, 26,* 98-107.

Knight, R.G., Chisholm, B.J., Paulin, J.M., & Waal-Manning, H.J. (1988). The Spielberger Anger Expression Scale: some psychometric data. *Journal of Clinical Psychology, 27,* 279–281.

Krantz, D. S., & Manuck, S. B. (1984). Acute psychophysiologic reactivity and risk of cardiovascular disease: A review and methodologic critique. *Psychological Bulletin, 96,* 435-464.

Krantz, D. S., Kop, W. J., Santiago, H. T., & Gottdiener, J. S. (1996). Mental stress as a trigger of myocardial ischemia and infarction. *Cardiology Clinics, 14*(2), 271-87.

Krieger, N. (1987). Shades of difference: Theoretical underpinnings of the medical controversy on black/white differences in the United States, 1830–1870. *The International Journal of Health Services, 17,* 259–78.

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

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,* 1370-1378.

Krieger, N., Sidney, S., & Coakley, E. (1998). Racial discrimination and skin color in the CARDIA Study: implications for public health research. *American Journal of Public Health, 88*, 1308–13.

Krieger, N. (1999). Embodying inequality: A review of concepts, measures, and methods for studying health consequences of discrimination. *International Journal of Health Services, 29*(2), 295–352. doi:10.2190/M11W-VWXE-KQM9-G97Q.

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, and occupational exposures in US low-income black, white and Latino workers. *Social Science & Medicine, 67*(12)*,* 1970-1981.

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.

Kubzansky, L. D., & Kawachi, I. (2000). Going to the heart of the matter: Negative emotions and coronary heart disease. *Psychosomatic Research, 48,* 323-337.

Kudielka, B. M., Buske-Kirschbaum, A., Hellhammer, D. H., & Kirschbaum, C. (2004). Differential heart rate reactivity and recovery after psychosocial stress (TSST) in health children, younger adults, and elderly adults: the impact of age and gender. *International Journal of Behavioral Medicine, 11,* 116–121.

Kurian, A. K., & Cardarelli, K. M. (2007). Racial and ethnic differences in cardiovascular disease risk factors: A systematic review. *Ethnicity & Disease*, *17*, 143–152.

Landrine, H., & Klonoff, E. A. (1996). The schedule of racist events: A measure of racial discrimination and a study of its negative physical and mental consequences. *Journal of Black Psychology, 22,* 144-168.

Landrine, H., & Klonoff, E. A. (1996). The Schedule of Racist Events: A measure of racial discrimination and a study of its negative physical and mental consequences. J*ournal of Black Psychology*, *22*, 144-168.

Landrine, H., & Klonoff, E. A. (2000). Racial discrimination and cigarette smoking among Blacks: Findings from two studies. *Ethnicity & Disease, 10*, 195–202.

Landrine, H., Klonoff, E. A., Corral, I., Fernandez, S., & Roesch, S. (2006). Conceptualizing and measuring ethnic discrimination in health research. *Journal of Behavioral Medicine, 29,* 79-94.

Lepore, S. J., Mata Allen, K. A., & Evans, G. W. (1993). Social support lowers cardiovascular reactivity to an acute stressor. *Psychosomatic Medicine, 55*, 518–524.

Lett, H. S., Blumenthal, J. A., Babyak, M. A., Sherwood, A., Strauman, T., Robins, C., & Newman, M. F. (2004). Depression as a risk factor for coronary artery disease: Evidence, mechanisms, and treatment. *Psychosomatic Medicine*, *66* (3), 305-315. doi:10.1097/01.psy.0000126207.43307.c0

Levitt, C. (1997). The morality of race in Canada. *Society, 34,* 40-47.

Lewis, 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, 64A,* 1002-1008.

Lewis, T. T., Aiello, A. E., Leurgans, S., Kelly, J., & Barnes, L. L. (2010). Self-reported experiences of everyday discrimination are associated with elevated C-reactive protein levels in older African-American adults. *Brain, Behavior, and Immunity, 24,* 438-443.

Lieberson, S. (1980). *A piece of the pie: black and white immigrants since 1880*. Berkeley, CA: University of California Press.

Light, K. C., Turner, J. R., Hinderliter, A. L., & Sherwood, A. (1993). Race and gender comparisons: I. Hemodynamic responses to a series of stressors. *Health Psychology, 12,* 354-365.

Lin, S., & Kelsey, J. (2000). Use of race and ethnicity in epidemiologic research: Concepts, methodologic issues, and suggestions for research. *Epidemiologic Reviews, 22,* 187–202.

Lindley, D. V., & Scott, W. F. (1984). *New Cambridge Elementary Statistics Tables*. Cambridge University Press.

Lovallo, W. R., & Gerin, W. (2003). Psychophysiological reactivity: Mechanisms and pathways to cardiovascular disease. *Psychosomatic Medicine, 65*(1), 36-45.

Lu, M. C., & Halfon, N. (2003). Racial and ethnic disparities in birth outcomes: A life-course perspective. *Maternal and Child Health Journal, 7*(1), 13–30. doi:10.1023/A:1022537516969

Lui, R., So, L., Mohan, S., Khan, N., King, K., & Quan, H. (2010). Cardiovascular risk factors in ethnic populations within Canada: results from national cross-sectional surveys. *Open Medicine, 4*(3), e143-53.

Magai, C., Kerns, M. D., Gillespie, M., & Huang, B. (2003). Anger experience and anger inhibition in sub-populations of African American and European American older adults and relation to circulatory disease. *Journal of Health Psychology, 8*(4), 413-32.

Majahalme, S., Turjanmaa, V., Weder, A. B., Lu, H., Tuomisto, M., & Uusitalo, A. (1998). Office and laboratory blood pressures as predictors of daily blood pressure level in normotensive subjects and borderline and mild hypertensive subjects. *Clinical Physiology, 18,* 215–223.

Malan, L., Malan, N. T., Du Plessis, A., Wissing, M. P., Potgieter, J. C., & Seedat, Y. K. (2010). The cost of coping: A cardio-neuro-metabolic risk for Black South Africans? *Cardiovascular Journal of Africa*, *21*, 183–185.

Manuck, S. B., Kamarck, T. W., Kasprowicz, A. S., & Waldstein, S. R. (1993). Stability and patterning of behaviorally evoked cardiovascular reactivity. In J. J. Blascovich & E. S.

Katkin (Eds.), *Cardiovascular reactivity to psychological stress and disease*. APA science volumes (pp. 111-134). Washington, DC, US: American Psychological Association.

Martin, R., Watson, D., & Wan, C. K. (2000). A three-factor model of trait anger: dimensions of affect, behavior and cognition. *Journal of Personality, 68,* 869-897.

Massey, D. S., & Denton, N. A. (1993). *American Apartheid: Segregation and the Making of the Underclass*. Cambridge, Massachusetts: Harvard University Press.

Matthews, K. A., & Stoney, C. M., (1988). Influences of sex and age oncardiovascular responses during stress. *Psychosomatic Medicine, 50*, 46–56.

Matthews, K. A. (2003). Cardiovascular reactivity to stress predicts future blood pressure in adolescence. *Psychosomatic Medicine, 65,* 410-415.

Matthews, K. A. (2004). Blood pressure reactivity to psychological stress predicts hypertension in the CARDIA study. *Circulation, 110,* 74-78.

Matthews, K. A., Salomon, K., Kenyon, K., & Zhou, F. (2005). Unfair treatment, discrimination, and ambulatory blood pressure in Black and White adolescents. *Health Psychology, 24,* 258-265.

Mays, V. M., Cochran, S. D., & Barnes, N. W. (2007). Race, race- based discrimination, and health outcomes among African Americans. *Annual Review of Psychology, 58*, 201–225. doi:10.1146/annurev.psych.57.102904.190212.

McClure, H., Snodgrass, J., Martinez, A., Eddy, J., Jimenez, R., & Isiordia, L. (2010a). Discrimination, psychosocial stress, and health among Latin American immigrants in Oregon. *American Journal of Human Biology, 22,* 421-423.

McClure, H., Snodgrass, J., Martinez, A., Eddy, J., Jimenez, R., & Isiordia, L. (2010b). Discrimination-related stress, blood pressure and Epstein-Barr virus antibodies among Latin American immigrants in Oregon, US. *Journal of Biosocial Science, 42,* 433-461.

McEwen, B. S. (1998). Stress, adaptation, and disease: Allostasis and allostatic load. In S. M. McCann, J. M. Lipton, et al. (Eds.), *Annals of the New York Academy of Sciences, Vol. 840: Neuroimmunomodulation: Molecular aspects, integrative systems, and clinical advances* (pp. 33–44). New York: New York Academy of Sciences.

McEwen, B. S. (2004). Protection and damage from acute and chronic stress, allostasis and allostatic load overload and relevance to the pathophysiology of psychiatric disorders. *Annals of the New York Academy of Sciences, 1032*, 1–7. doi:10.1196/annals. 1314.001.

McGrath, J., Matthews, K., & Brady, S. (2006). Individual vs. neighborhood socioeconomic status and race as predictors of adolescent ambulatory blood pressure and heart rate. *Social Science & Medicine, 63,* 1442-53.

McKinney, M. E., Miner, M. H., Rüddel, H., McIlvain, H. E., Witte, H., … Grant, L. B. (1985). The standardized mental stress test protocol: test-retest reliability and comparison with ambulatory blood pressure monitoring. *Psychophysiology, 22*(4), 453-463.

McNeilly, M. D., Robinson, E. L., Anderson, N. B., Pieper, C. F., Shah, A., Toth, P. S., … Gerin, W. (1995). Effects of racist provocation and social support on cardiovascular reactivity in African American women. *The International Journal of Behavioral Medicine, 2*(4), 321-38.

McNeilly, M., Anderson, N., Armstead, C., Clark, R., Corbett, M., Robinson, E., … Lepisto, E. M. (1996). The perceived racism scale: a multidimensional assessment of the experience of white racism among African Americans. *Ethnicity & Disease, 6*(1-2), 154-166.

van Melle, J. P., de Jonge, P., Spijkerman, T. A., Tijssen, J. G. P., Ormel, J., van Veldhuisen, D. J., & van den Berg, M. P. (2004). Prognostic association of depression following myocardial infarction with mortality and cardiovascular events: A meta-analysis. *Psychosomatic Medicine*, *66*(6), 814-822. doi: 10.1097/01.psy.0000146294.82810.9c.

Mendes de Leon, C. F. (1992). Anger and impatience/irritability in patients of low socioeconomic status with acute coronary heart disease. *Journal of Behavioral Medicine, 15,* 273–284.

Mendes, W. B., Major, B., McCoy, S., & Blascovich J. (2008). How attributional ambiguity shapes physiological and emotional responses to social rejection and acceptance. *Journal of Personality and Social Psychology, 94,* 278–291.

Menkes, M. S., Matthews, K. A., Krantz, D. S., Lundberg, V., Mead, L. A., Qaqish, B., … Pearson, T. A. (1989). Cardiovascular reactivity to the cold pressor test as a predictor of hypertension. *Hypertension, 14*, 524–530.

Mensah, J. (2002). *Black Canadians: History, experiences, social conditions.* Halifax, Nova Scotia, Canada: Fernwood Press.

Mezuk, B., Kershaw, K. N., Hudson, D., Lim, K. A., & Ratliff, S. (2011). Job strain, workplace discrimination, and hypertension among older workers: The Health and Retirement Study. *Race and Social Problems, 3,* 38-50.

Miller, G. E., & Doshi, A. K. (2006). Diuretic Use in the Treatment of Hypertension, by Selected Population Characteristics. *Agency for Healthcare Research and Quality*, *Statistical, Brief*

#119. Retrieved from http://meps.ahrq.gov/mepsweb/data_files/publications/st119/stat119.shtml

Mittleman, M. A., Maclure, M., Sherwood, J. B., Mulry, R. P., Tofler, G. H., Jacobs, S. C., … Muller, J. E. (1995). Triggering of myocardial infarction by episodes of anger. *Circulation, 92,* 1720-1725.

Moghaddama, F., Taylor, D., Ditto, B., Jacobs, K., & Bianchi, E. (2002). Psychological distress and perceived discrimination: A study of women in India. *International Journal of Intercultural Relations, 26,* 381-390.

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,* 123–31.

Mujahid, M. S., Diez Roux, A. V., Cooper, R. C., Shea, S., & Williams, D. R. (2011). Neighborhood stressors and race/ethnic differences in hypertension prevalence. *American Journal of Hypertension, 24,* 187-193.

Murphy, J. K., Alpert, B. S., & Walker, S. S. (1994). Consistency of ethnic differences in children's pressor reactivity. 1987 to 1992. *Hypertension, 1*(suppl), l152–I155.

Musante, L., Treiber, F.A., Davis, H.C., & Waller, J.L. (1999). Assessment of self-reported anger expression in youth. *Assessment, 6*, 225–233.

Myers, H.F. (2009). Ethnicity and socio-economic status-related stresses in context: an integrative review and conceptual model. *Journal of Behavioral Medicine, 32,* 9–19, doi: 10.1007/s10865-008-9181-4.

National Institute of Mental Health. (1983). *Research Highlights: Extramural Research*. Washington, DC: U.S. Government Printing Office.

Nesson, K. L. (2008). *The relation of perceived racism and involvement with the criminal justice system among African American males.* US: ProQuest Information & Learning.

Noh, S., Kaspar, V., & Wickrama, K.A.S. (2007). Overt and subtle racial discrimination and mental health: Preliminary findings for Korean immigrants. *American Journal of Public Health, 97,* 1269–1274. doi:10.215/AJPH.2005.085316.

Nosek, B. A., Smyth, F. L., Hansen, J. J., Devos, T., Lindner, N. M., Ranganath, K. A, & Banaji, M. R. (2007). Pervasiveness and Correlates of Implicit Attitudes and Stereotypes. *European Review of Social Psychology, 18*, 36–88.

O'Donnell, C. J., Ridker, P. M., Glynn, R. J., Berger, K., Ajani, U., Manson, J. E., & Hennekens, C. H. (1997). Hypertension and borderline isolated systolic hypertension increase risks of cardiovascular disease and mortality in male physicians. *Circulation, 95,* 1132-1139.

Ong, K. L., Cheung, B. M., Man, Y. B., Lau, C. P., & Lam, K. S. L. (2007). Prevalence, awareness, treatment, and control of hypertension among United States adults 1999-2004. *Hypertension, 49,* 69-75.

Orwin, R. G. (1983). A fail-safe N for effect size in meta-analysis. *Journal of Educational Statistics, 8,* 157-159.

Pamuk, E. (ed.). (1998). *Socioeconomic status and health chartbook. Health, United States, 1998*. Hyattsville (MD): National Center for Health Statistics.

Paradies, Y. (2006a). Defining, conceptualizing and characterizing racism in health research. *Critical Public Health, 16,* 143–157.

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

Parati, G., Pomidossi, G., Casadei, R., Ravogli, A., Gropelli, A. Cesana, B., & Mancia, G. (1988). Comparison of cardiovascular effects of different laboratory stressors and their relationship with blood pressure variability. *Journal of Hypertension, 6*, 481-488.

Pascoe, E. A., & Richman, L. S. (2009). Perceived discrimination and health: a meta-analytic review. *Psychological Bulletin, 135,* 531–554.

Pearlin, L. I., Schieman, S., Fazio, E. M., & Meersman, S. C. (2005). Stress, health, and the life course: Some conceptual perspectives. *Journal of Health and Social Behavior, 46*(2) 205–219.

Pennebaker, J. W. (ed.). (1995). *Emotion, disclosure and health.* Washington, DC: APA Press.

Peters, R. M., & Flack, J. M. (2000). Salt-sensitivity and hypertension in African Americans: Implications for cardiovascular nurses. *Progress in Cardiovascular Nursing, 15*(4), 138-144.

Peters, R. M. (2006). The relationship of racism, chronic stress emotions, and blood pressure. *Journal of Nursing Scholarship, 38,* 234-240.

Peterson, R., & Brown, S. (2005). On the use of beta coefficients in meta-analysis. *Journal of Applied Psychology, 90*, 175-181.

Pickering, T. G., & Gerin, W. (1988). Ambulatory blood pressure monitoring and cardiovascular reactivity testing for the evaluation of the role of psychosocial factors and prognosis in

hypertensive patients. *American Heart Journal, 116,* 665–672.

Pickering, T. G., Hall, J. E., Appel, L. J., Falkner, B. E., Graves, J., Hill, M. N., … Roccella, E. J. (2005). Recommendations for blood pressure measurement in humans and experimental animals: Part 1. *Hypertension, 45*, 142–161.

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 and Disease, 18,* 289-293.

Poole, J. C., Sneider, H., Davis, H. C., & Treiber, F. A. (2006). Anger suppression and adiposity modulate association between ADRB2 haplotype and cardiovascular stress reactivity. *Psychosomatic medicine, 68,* 207-12.

Province, M. A., Boerwinkle, E., Chakravarti, A., Cooper, R., Fornage, M., Leppert, M., … Ranade, K. (2000). Lack of association of the angiotensinogen-6 polymorphism with blood pressure levels in the comprehensive NHLBI Family Blood Pressure Program. National Heart, Lung and Blood Institute. *Journal of Hypertension, 18,* 867–76.

Quillian, L. (2008). Does unconscious racism exist? *Social Psychology Quarterly, 71,* 6–11. doi:10.1177/019027250807100103.

Rabineau, K. M., Treiber, F. A., Poole, J., & Ludwig, D. (2005). Interactive effects of anger expression and ET-1 Lys198Asn polymorphism on vasoconstriction reactivity to behavioral stress. *Annals of Behavioral Medicine, 30, 85-89.*

Rahman, S., Hu, H., McNeely, E., Rahman, S. M., Krieger, N., Waterman, P., … Gragg, R. D. (2008). Social and environmental risk factors for hypertension in African Americans. *Florida Public Heath Review, 5,* 64-72.

Ray, C. A., & Monahan, K. D. (2002). Sympathetic vascular transduction is augmented in young normotensive Blacks. *Journal of Applied Physiology*, *92*, 651–656. doi: 10.1152/japplphysiol.00788.2001.

Redmond, N., H. J., & Hicks, L. S. (2011). Health behaviors and racial disparity in blood pressure control in the national health and nutrition examination survey. Hypertension, 57(3), 383–389.

Reimann, M., Hamer, M., Schlaich, M., Malan, N., Rüdiger, H., Ziemssen, T., & Malan, L. (2012). Autonomic responses to stress in Black versus Caucasian Africans: The SABPA Study. *Psychophysiology, 49*, 454-461. doi: 10.1111/j.1469-8986.2011.01328.x

Richman, L. S., 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 Psychology, 29,* 403-411.

Richter, S., Deter, H. C., Rudat, M., Schächinger, H., Zimmermann-Viehoff, F., & Weber, C. (2010). Anger and cardiovascular startle reactivity in normotensive young males. *International Journal of Psychophysiology, 79*(3), 364-70. doi: 10.1016/j.ijpsycho.2010.12.004.

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

Rosenberg, M. S., Adams, D., & Gurevitch, J. (2000). *MetaWin. Statistical Software for Meta-Analysis, Version 2.0*. Sunderland, Massachusetts: Sinauer Associates.

Rosenthal, R. (1991). *Meta-analytic Procedures for Social Research* (Revised edition). Newbury Park, CA: Sage.

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,* 116-132.

van Ryn, M. (2002). Research on the provider contribution to race/ethnicity disparities in medical care. *Medical Care, 40*(1), I140–I151. doi:10.1097/00005650-200201001-00015.

Saab, P. G., Llabre, M. M., Hurwitz, B. E., Frame, C. A., Reineke, L. J., Fins, A. I., … Schneiderman, N. (1992). Myocardial and Peripheral Vascular Responses to Behavioral Challenges and Their Stability in Black and White Americans. *Psychophysiology, 29,* 384-397.

Saab, P. G., Llabre, M. M., Schneiderman, N., Hurwitz, B. E., McDonald, P. G., Evans, J., … Klein, B. (1997). Influence of ethnicity and gender on cardiovascular responses to active coping and inhibitory-passive coping challenges. *Psychosomatic Medicine, 59,* 434-446.

Salomon, K., & Jagusztyn, E. (2008). Resting cardiovascular levels and reactivity to interpersonal incivility among Black, Latina/o, and white individuals: The moderating role of ethnic discrimination. *Health Psychology, 27,* 473-481.

Sánchez-Meca, J., Marín-Martínez, F., & Chacón-Moscoso, S. (2003). Effect-size indices for

dichotomized outcomes in meta-analysis. *Psychological Methods, 8,* 448-467.

Sher, Y., Lolak, S., & Maldonado, J. (2010). The impact of depression in heart disease. *Current Psychiatry Reports*, *12*(3), 255-264. doi: 10.1007/s11920-010-0116-8.

Scherwitz, L., Perkins, L., Chesney, M., & Hughes, G. (1991). Cook–Medley Hostility Scale and subsets: Relationship to demographic and psychosocial characteristics in young adults in the CARDIA study. *Psychosomatic Medicine, 53,* 36–49.

Sherwood, A., Allen, M. T., Obrist, P. A., & Langer, A. W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. *Psychophysiology, 23,* 89-104.

Sherwood, A., & Turner, J. R. (1993). Postural stability of hemodynamic responses during mental challenge. *Psychophysiology, 30,* 237-244.

Schmidt, M. M., Lopes, R. D., Newby, L. K., Moura, M. R., Stochero, L., Gottschall, C. M., & Quadros, A. S. (2013). Anger control and cardiovascular outcomes. *International Journal of Cardiology, 9,* 168(4), 4338-9. doi: 10.1016/j.ijcard.2013.05.083.

Schmidt, N., Oh, L., & Hayes, T. (2009). Fixed- versus random-effects models in meta-analysis: Model properties and an empirical comparison of differences in results. *British Journal of Mathematical and Statistical Psychology, 62,* 97-128.

Schulz, A. J., Gravlee, C. C., Williams, D. R., Israel, B. A., Mentz, G., & Rowe, Z. (2006). Discrimination, symptoms of depression, and self-rated health among African American women in Detroit: Results from a longitudinal analysis. *American Journal of Public Health, 96*(7), 1265–1270. doi:10.2105/AJPH.2005.064543.

Schum, J. L., Jorgensen, R. S., Verhaeghen, P., Sauro, M., & Thibodeau, R. (2003). Trait anger, anger expression, and ambulatory blood pressure: A meta-analytic review. *Journal of Behavioral Medicine, 26,* 395–415.

Sherwood, A., Allen, M. T., Obrist, P. A., & Langer, A. W. (1986). Evaluation of beta-adrenergic influences on cardiovascular and metabolic adjustments to physical and psychological stress. *Psychophysiology, 23,* 89-104.

Sherwood, A., Dolan, C. A., & Light, K. C. (1990). Hemodynamics of blood pressure responses during active and passive coping. *Psychophysiology, 27*, 656-668.

Sherwood, A., & Turner, J. R. (1993). Postural stability of hemodynamic responses during mental challenge. *Psychophysiology, 30,* 237-244.

Sherwood, A., Johnson, K., Blumenthal, J. A., & Hinderliter, A. L. (1999). Endothelial function and hemodynamic responses during mental stress. *Psychosomatic Medicine, 61*, 365–370.

Siegman, A. W., Dembroski, T. M., & Ringel, N. (1987). Components of hostility and the severity of coronary artery disease. *Psychosomatic Medicine, 49*(2), 127-134.

Sigelman. L., & Welch, S. (1993). The contact hypothesis revisited: Black-White interaction and positive racial attitudes. *Social Forces, 71,* 781–95.

Singer, B., & Ryff, C. D. (1999). Hierarchies of life histories and associated health risks. In N. E. Adler, M. Marmot, B. S. McEwen, & J. Stewart (Eds.), *Socioeconomic status and health in industrial nations: Social, psychological, and biological pathways* (pp. 96–115). New York: Annals of the New York Academy of Sciences.

Singh. G. K., Kochanek, K. D., & MacDorman, M. F. (1996). Advance report of final mortality statistics, 1994. *Monthly Vital Statistics Report, 45*(suppl 3), 1–76.

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., 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 Psychology, 29,* 403-411.

Smedley, B. D., Stith, A. Y., & Nelson, A. R. (2003). *Unequal treatment: Confronting racial and ethnic disparities in health care.* Washington, DC: National Academies Press.

Somova, L. (1992). Parental history of hypertension in Zimbabwe and cardiovascular reactivity to psychological experimental stress. *Central African Journal of Medicine, 38*(6), 214-20.

Spielberger, C.D. (1988). *State–trait anger expression inventory professional manual.* Psychological Assessment Resources, Odessa, FL.

Spielberger, C.D. (1999). *Professional manual for the State-Trait Anger Expression Inventory-2 (STAXI-2)*. Odessa, FL: Psychological Assessment Resources.

Spielberger, C.D., Johnson, E.H., Jacobs, G.A., Krasner, S.E., Oesterle, S.E., & Worden, T.J. (1986). *The anger expression (AX) scale* (Unpublished manscript). University of South Florida, Center for Research in Community Psychology, Tampa, Florida.

Spielberger, C. D., Johnson, E. H., Russell, S. F., Crane, R. J., Jacobs, G. A., & de Worn, T. I. (1985). The experience and expression of anger: Construction and validation of an anger

expression scale. In M. A. Chesney & R. H. Rosenman (Eds.), *Anger and Hostility in Cardiovascular and Behavioral Disorders* (pp. 5-30). New York: McGraw-Hill and Hemisphere.

Spielberger, C.D. & Reheiser, E.C. (2009). Assessment of Emotions: Anxiety, Anger, Depression, and Curiosity. *Applied Psychology: Health and Well-Being, 1 (3),* 271–302. doi:10.1111/j.1758-0854.2009.01017.x.

Spielberger, C.D., Reheiser, E.C., Sydeman, S.J. (1995). Measuring the experience, expression, and control of anger. In H. Kassinove (Ed.), *Anger disorders: definition, diagnosis, and treatment*, Taylor & Francis: Washington, DC.

Spielberger, C. D., & Sydeman, S. S. (1994). State-Trait Anxiety Inventory and State-Trait Anger Expression Inventory. In C. D. Spielberger & S. J. Sydeman (Eds.), *The use of psychological testing for treatment planning and outcome assessment* (pp. 292-321). Hillsdale, NJ, England: Lawrence Erlbaum Associates, Inc.

Stam, R. (2007). PTSD and stress sensitization: A tale of brain and body. Part 1: Human studies. *Neuroscience, 31,* 530–557.

Statistics Canada. (2001). *Census of Population.* Ottawa, Ontario, Canada: Minister of Industry.

Statistics Canada. (2003). *Ethnic diversity survey: Portrait of a multicultural society.* Ottawa, Ontario, Canada: Minister of Industry.

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,* 746-750.

Stein, C. M., Lang, C. C., Singh, I., He, H. B., & Wood, A. J. (2000). Increased vascular adrenergic vasoconstriction and decreased vasodilation in Blacks. Additive mechanisms leading to enhanced vascular reactivity. *Hypertension*, *36*, 945–951.

Steptoe, A., Fieldman, G., Evans, O., & Perry, L. (1996). Cardiovascular risk and responsivity to mental stress: the influence of age, gender and risk factors. *Journal of Cardiovascular Risk, 3*, 83–93.

Steptoe, A., & Cropley, M. (2000). Persistent high job demands and reactivity to mental stress predict future ambulatory blood pressure. *Journal of Hypertension, 18,* 581–586.

Sternthal, M. J., Slopen, N., & Williams, D. R. (2011). Racial disparities in health: How much does stress really matter. *Du Bois Review: Social Science Research on Race, 8*(1), 95-113.

doi:10.1017/S1742058X11000087

Stewart, J. C., & France, C. R. (2001). Cardiovascular recovery from stress predicts longitudinal changes in blood pressure. *Biological Psychology, 58,* 105–120.

Summers, K. M., Martin, K. E., & Watson, K. (2010). Impact and clinical management of depression in patients with coronary artery disease. *Pharmacotherapy*, *30*(3), 304-322. doi: 10.1592/phco.30.3.304.

Suarez, E. C., & William, R. B. (1990). The relationship between dimensions of hostility and cardiovascular reactivity as a function of task characteristics. *Psychosomatic Medicine, 52*, 558-570.

Sue, D. W., Capodilupo, C. M., Torino, G. C., Bucceri, J. M., Holder, A. M. B., ... Esquilin, M. (2007). Racial microaggressions in everyday life: Implications for clinical practice. *American Psychologist, 62*, 271–286. doi:10.1037/0003-066X. 62.4.271.

Suls, J., & Bunde, J. (2005). Anger, anxiety and depression as risk factors for cardiovascular disease: the problems and implications of overlapping affective dispositions. *Psychological Bulletin, 131,* 260-300.

Sutherland, M. E., & Harrell, J. P. (1986). Individual differences in physiological responses to fearful, racially noxious, and neutral imagery. *Imagination, Cognition and Personality, 6,* 133-150.

Taherzadeh, A., Brewster, L. M., van Montfrans, G. A., & van Bavel, E. (2010). Function and structure of resistance vessels in black and white people. *The Journal of Clinical Hypertension, 12*(6), 431-438. doi: 10.1111/j.1751-7176.2010.00269.x.

Taylor, D.M., Wright, S.C., & Porter, P.E. (1994). Dimensions of Perceived Discrimination: The Personal/Group Discrimination Discrepancy. In The Psychology of Prejudice: The Ontario Symposium, eds. Mark P. Zanna and James M. Olson, vol. 7, 233–55. Hillsdale, NJ: Lawrence Erlbaum Associates, Inc

Taylor, J., & Grundy, C. (1996). Measuring Black internalization of White stereotypes about Blacks: The Nadanolitization Scale. In R. L. Jones (Ed.), *Handbook of Tests & Measurements for Black Populations*. Hampton, VA: Cobb & Henry.

Tersman, Z., Collins, A., & Eneroth, P.(1991). Cardiovascular responses to psychological and physiological stressors during the menstrual cycle. *Psychosomatic Medicine, 53*, 185–197.

Thomas, S. P. (ed.). (1993). *Women and Anger.* New York: Springer Publishing.

Thrasher, A. D., Earp, J. A., Golin, C. E., & Zimmer, C. R. (2008). Discrimination, distrust, and racial/ethnic disparities in antiretroviral therapy adherence among a national sample of HIV infected patients. *Journal of Acquired Immune Deficiency Syndromes* (JAIDS), *49*, 84–93. doi:10.1097/QAI.0b013e3181845589.

Thomas, M., Herring, C., & Horton, H. D. (1994). Discrimination over the life course: A synthetic cohort analysis of earnings differences between black and white males, 1940–1990. *Social Problems, 41,* 608-628.

Thomas, K. S., Nelesen, R. A., Malcarne, V. L., Ziegler, M. G., & Dimsdale, J. E. (2006). Ethnicity, perceived discrimination, and vascular reactivity to phenylephrine. *Psychosomatic Medicine, 68*(5), 692-697.

Thomas, K. S., Nelesen, R. A., Ziegler, M. G., Natarajan, L., & Dimsdale, J. E. (2009). Influence of education and neighborhood poverty on pressor responses to phenylephrine in African-Americans and Caucasian-Americans. *Biological Psychology, 82,* 18-24.

Thompson, H. S., Kamarck, T. W., & Manuck, S. B. (2002). The association between racial identity and hypertension in African Americans adults: Elevated resting and ambulatory blood pressure as outcomes. *Ethnicity and Disease, 12,* 20-28.

Thornhill, E. M. A. (2008) So Seldom for Us, So Often Against Us: Blacks and Law in Canada *Journal of Black Studies*, *38*(3), 321-337.

Thornhill, E. M. A. (Eds.). (2008) Blacks in Canada: Retrospects, Introspects, Prospects. *Journal of Black Studies* (special issue), *38*(3).

Todorova, I. L. G., Falcon, L. M., Lincoln, A. K., & Price, L. L. (2010). Perceived discrimination, psychological distress and health. *Sociology of Health & Illness, 32,* 843-861.

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.

Treiber, F. A., Musante, L., Braden, D., Arensman, F., Strong, W. B., Levy, M., & Leverett, S. (1990). Racial differences in hemodynamic responses to the cold face stimulus in children and adults. *Psychosomatic Medicine, 52,* 286-296.

Trieber, F. A., McCaffrey, F., Pflieger, K., Raunikar, R. A., Strong, W. B., & Davis, H. (1993). Determinants of left ventricular mass in normotensive children. *American Journal of Hypertension, 6*, 505-513.

Treiber, F. A. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65,* 46-62.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65,* 46–62.

Trivedi, A. N., & Ayanian, J. Z. (2006). Perceived discrimination and use of preventive health services. *Journal of General Internal Medicine, 21*, 553–558. doi:10.1111/j.1525-1497.2006.00413.x.

Tull, S., Wickramasuriya, T., Taylor, J., Smith-Burns, J., Brown, M., Champagnie, G., … Jordan, O. W. (1999). Relationship of internalized racism to abdominal obesity and blood pressure in Afro-Caribbean women. *Journal of the National Medical Association, 91,* 447-452.

Tull, E. S., Sheu, Y. T., Butler, C., & Cornelious, K. (2005). Relationships between perceived stress, coping behavior and cortisol secretion in women with high and low levels of internalized racism. *Journal of the National Medical Association, 97*, 206–212.

Turner, J. R., Sherwood, A., & Light, K. C. (1990). Generalizability of cardiovascular response: supportive evidence for the reactivity hypothesis. *International Journal of Psychophysiology, 11*, 207-212.

Turner, J. R., Sherwood, A., & Light, K. C. (1994). Intertask consistency of hemodynamic responses to laboratory stressors in a biracial sample of men and women. *International Journal of Psychophysiology, 17*, 159-164.

US Department of Health and Human Services. (2000). *Healthy people 2010: Understanding and improving health and objectives for improving health* (2nd ed.). Washington, DC, US: Department of Health and Human Services.

Verdecchia, P., Angeli, F., Gattobigio, R., & Porcellati, C. (2003). Ambulatory blood pressure monitoring and prognosis in the management of essential hypertension. *Expert Review of Cardiovascular Therapy, 1*, 79–89.

Verhaeghen P., Aikman, S. N., van Gulick, A. E. (2011). Prime and prejudice: Co-occurrence in the culture as a source of automatic stereotype priming. *British Journal of Social Psychology, 50*(3), 501– 518.

Victoroff, J. (2005). The Oppression Questionnaire [August 5, 2008]. Retrieved from http://www.humiliationstudies.org/documents/VictoroffOppressionQuestionnaire.pdf.

Vorauer, J., & Kumhyr, S.M. (2001). Is This About You or Me? Self-Versus Other-Directed Judgments and Feelings in Response to Intergroup Interaction. *Personality and Social Psychology Bulletin 27,* 706–19.

Watts-Jones, D. (2002). Healing internalized racism: The role of a within group sanctuary among people of African descent. *Family Process, 41,* 591-601.

White, K., Borrell, L. N., Wong, D. W., Galea, S., Ogedegbe, G., & Glymour, M. M. (2011). Racial/ethnic residential segregation and self-reported hypertension among US- and foreign-born Blacks in New York City. *American Journal of Hypertension, 24,* 904-910.

Wickrama, K. A. S., Conger, R. D., Wallace, L. E., & Elder, G. J., Jr. (1999). The inter-generational transmission of health-risk behaviors: Adolescent lifestyles and gender moderating effects. *Journal of Health and Social Behavior, 40*(3), 258–272. doi:10.2307/2676351.

Wilkinson, R. G., & Marmot, M. (Eds.). (2003). *Social determinants of health: The solid facts*. Copenhagen, Denmark: World Health Organization Publications.

Williams, D. R., Yu, Y., Jackson, J., & Anderson, N. (1997). Racial differences in physical and mental health: socioeconomic status, stress, and discrimination. *Journal of Health Psychology, 2*, 335–351.

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

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., & Jackson, P. B. (2005). Social sources of racial disparities in health. *Health Affairs, 24*(2), 325–334. doi: 10.1377/hlthaff.24.2.325.

Williams, D. R., Gonzalez, H. M., Williams, S., Mohammed, S. A., Moomal, H., & Stein, D. J. (2008). Perceived discrimination, race and health in South Africa. *Social Science & Medicine*, *67*, 441–452.

Williams, D. R., & Mohammed, S. A. (2009). Discrimination and racial disparities in health: Evidence and needed research. *Journal of Behavioral Medicine; 32*, 20-47.

Williams, K. D. (2001). *Ostracism: The power of silence.* New York, NY: Guilford Press.

Wilson, T. W., & Grim, C. E. (1991). Biohistory of slavery and blood pressure differences in

blacks today. *Hypertension, 17,* I122-I128.

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,* 1585-1592.

Wood, D. L., Sheps, S. G., Elveback, L. R., & Schirger, A. (1984). Cold pressor test as a predictor of hypertension. *Hypertension*, 6, 301–306.

Wyatt, S. B., Williams, D. R., Calvin, R., Henderson, F., Walker, E., & Winters, K. (2003). Racism and cardiovascular disease in African Americans: Evidence and implications for the Jackson Heart Study. *The American Journal of the Medical Sciences, 325*(6), 315–331. doi:10.1097/00000441-200306000-00003.

Xie, H. G., Kim, R. B., Stein, C. M., Gainer, J. V., Brown, N. J., & Wood, A. J. (1999). Alpha1A-adrenergic receptor polymorphism: Association with ethnicity but not essential hypertension. *Pharmacogenetics, 9,* 651–6.

Yehuda, R., Bryant, R., Marmar, C., & Zohar, J. (2005). Pathological Responses to Terrorism. *Neuropsychopharmacology, 30,* 1793–1805. doi:10.1038/sj.npp.1300816.

Ziegler, M. G., Nelesen, R. A., Mills, P. J., Ancoli-Israel, S., Clausen, J. L., Watkins, L., & Dimsdale, J. E. (1995). The effect of hypoxia on baroreflexes and pressor sensitivity in sleep apnea and hypertension. *Sleep, 18,* 859–65.