Risk Factors for Hypertension in a Black Canadian Population: Stress, Cardiovascular Reactivity, Cardiovascular Recovery, Gender, Dietary Sodium, Ambulatory Blood Pressure, Blood Pressure Dipping, Anger Expression, and Perceived Racism

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ABSTRACT

Risk Factors for Hypertension in a Black Canadian Population: Stress, Cardiovascular Reactivity, Cardiovascular Recovery, Gender, Dietary Sodium, Ambulatory Blood Pressure, Blood Pressure Dipping, Anger Expression, and Perceived Racism

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The present study investigated the effects of gender, sodium, anger-in, and perceived racism, on cardiovascular reactivity and recovery from laboratory stressors, ambulatory sleep and wake measures, and nocturnal blood pressure dipping. Fifty-three healthy Canadian Black male and female university students participated in a 21-day experimental protocol. Participants were twice exposed to three laboratory stressors tasks, and twice underwent 24-hr ambulatory blood pressure monitoring, at the end of a 10-day regular and a 10-day sodium-loaded diet period, respectively. Cardiovascular measures were recorded at baseline, pre-stress, during the stressor, and post-stress during the recovery period. Self- report measures on anger expression and perceived racism were collected. No effect of sodium on cardiovascular reactivity was found. Marginal findings implicate an interaction between sodium and gender on cardiovascular recovery from stress, and an effect of sodium on nocturnal blood pressure dipping. Gender differences were found for patterns in cardiovascular reactivity to stress, and for ambulatory heart rate measures. No significant findings emerged for effects of anger-in; however an interaction between gender and anger-out on blood pressure dipping is implicated. A significant interaction between gender, stressor type, and perceived racism was found for cardiovascular recovery from stress. Implications of these findings for future research on risk for development of hypertension in the Canadian Black population are discussed.

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Risk Factors for Hypertension in a Black Canadian Population: Stress, Cardiovascular Reactivity, Cardiovascular Recovery, Gender, Dietary Sodium, Ambulatory Blood Pressure, Blood Pressure Dipping, Anger Expression, and Perceived Racism

Hypertension, chronically elevated arterial pressure, is defined as untreated systolic blood pressure of 140 mmHg or higher, or diastolic blood pressure of 90 mmHg or higher, and is a primary risk factor in the development of cardiovascular diseases, including heart failure, coronary heart disease and stroke (Heart Disease and Stroke Statistics – 2009 Update, American Heart Association). Although hypertension in a small percentage of cases is traced to identifiable disorders (secondary hypertension), in the majority of cases the origin of elevated blood pressure is not readily determined (essential hypertension). The World Health Organization estimated that approximately 50% of cases of cardiovascular disease and 75% of strokes are caused by elevated blood pressure. In the U.S., the estimated prevalence for hypertension is approximately 1 in 3 adults (Heart Disease and Stroke Statistics - 2009 Update, American Heart Association). Moreover, a consistent finding in epidemiological literature is that hypertension is more prevalent, and is associated with greater morbidity and mortality, in Black compared to White Americans (Gillum, 1994).

The prevalence rate of hypertension among Black Americans, one of the highest in the world, is approximately two times greater than that for White Americans (Heart Disease and Stroke Statistics - 2009 Update, American Heart Association). Hypertension in Canada, similar to that in the U.S., is found to be present in approximately 21% of the population, with the highest prevalence rate among Black Canadians (Leenen et al., 2008). Additionally, hypertension in Blacks compared to Whites is more likely to develop at a younger age

(Berenson, Voors, Dalferes, Webber, & Schuler, 1979; Falkner, 1993), to be more severe, and to progress more rapidly with age (Hildreth & Saunders, 1991). Despite these findings, there is still insufficient research investigating risk factors for hypertension within Black populations in the Western Hemisphere. The present research aims to address this gap in research and contribute to research knowledge in the area.

Essential hypertension is generally agreed to be a complex and multifactorial disorder (Weiner & Shapira, 1987), with many etiological factors proposed, including the particularly well documented hypothesized risk factors of elevated cardiovascular reactivity to stress and dietary sodium (Anderson, McNeilly, & Myers, 1992; Harrell, Merritt, & Kalu, 1998; Krantz & Manuck, 1984; Umemura et al., 1992). More recently, research suggests the role of delayed cardiovascular recovery as a potentially important but under-investigated risk factor. Additionally, gender differences in cardiovascular reactivity and recovery have been implicated as a risk factor in the development of hypertension and cardiovascular diseases (Allen, Stoney, Owens, & Matthews, 1993; Girdler, Turner, Sherwood & Light, 1990; Hokanson, Burgess, & Cohen, 1963; Lai & Linden, 1992; Vogele, Jarvis, & Cheeseman, 1997), which may be linked to the higher prevalence for these conditions in men than in women in their reproductive years (Oparil & Miller, 2005). A risk factor of particular relevance to Black populations in the Western hemisphere is the proposed chronic psychosocial stressor, perceived racism (Thompson, Kamarck, & Manuck, 2002). Studies show that perceived racism in Blacks is positively associated with high blood pressure (Din-Dzietham, Nembhard, Collins, & Davis, 2004; Krieger & Sidney, 1996; Ryan, Gee, & Laflamme, 2006; Steffen, McNeilly, Anderson, & Sherwood, 2003; Sweet, McDade, Kiefe, & Liu, 2007). Finally, there is extensive literature linking certain psychological factors,

particularly anger, to the development of hypertension and cardiovascular diseases (Diamond, 1982; Light, 1987; Williams, 1987).

The above mentioned risk factors for hypertension have been predominantly investigated in experimental manipulations in laboratory protocols. There are fewer research studies investigating risk factors for hypertension involving the measurement of blood pressure in naturalistic settings (ambulatory blood pressure). Studies employing ambulatory blood pressure monitoring suggest that diurnal variations in blood pressure, particularly 'non-dipping' night-time blood pressure, are important risk factors for hypertension and cardiovascular disease (Kikuya et al., 2005; Murphy, Fumo, Gretler, Nelson, & Lang, 1991; Parati, Pomidossi, Albini, Malaspina, & Mancia, 1987). Non-dipping nocturnal blood pressure is indicated as a particularly important risk factor for Blacks given findings of increased prevalence of non-dipping night-time blood pressure in American Blacks compared to Whites (Profant & Dimsdale, 1999); however few studies have examined non-dipping within North American Black populations. Further research investigating blood pressure dipping within the Black population is clearly needed.

Research studies to date have looked at subsets of the above identified risk factors for hypertension to the exclusion of others; however no single study has investigated the combination of these risk factors in a single study. While these factors might be independently associated with risk for the development of hypertension, these factors might act in additive or interactive ways that conceivably represent increased risk. Thus, a comprehensive study looking at these multiple risk factors is warranted. The present study investigated associations between stress, dietary sodium, exaggerated cardiovascular reactivity, delayed cardiovascular recovery, gender, anger expression, perceived racism,

ambulatory sleep and wake blood pressure, and nocturnal blood pressure dipping, as they may moderate blood pressure and represent risk for hypertension within the Black Canadian population.

Laboratory Blood Pressure

Stress and Cardiovascular Reactivity. Cardiovascular reactivity is generally understood to reflect the physiological changes from a baseline state in response to some type of psychological or physical challenge or stressor (Manuck, Kasprowicz, Monroe, Larkin, & Kaplan, 1989). Findings from numerous studies provide supportive evidence that exaggerated cardiovascular response/cardiovascular hyper-reactivity to stressors may be a marker of, or contributor to development of essential hypertension or coronary heart disease (Chida & Steptoe, 2010; Falkner, 1984; Falkner, Kushner, Onesti, & Angelakos, 1981; Matthews et al., 2004; Matthews, Zhu, Tucker, & Wholley, 2006; Trieber et al., 2003). These studies employed diverse acute laboratory stressors including mental arithmetic tasks, the cold pressor task, and videogame challenges, among others. While the magnitude of stress exposure in the laboratory may be small compared with real life situations, it is posited that individuals who are reactive in the laboratory will also show exaggerated cardiovascular responses in daily life.

Faulkner and colleagues (1981) showed that reactivity to a 10-min mental arithmetic laboratory task predicted subsequent hypertension development in offspring of hypertensives. Likewise, in a study using the cold pressor task as a stressor, Menkes et al. (1989) found a relation between cardiovascular reactivity and future hypertension. Matthews et al. (2006) found that cardiovascular reactivity during a video game task was positively associated with coronary calcification 13-years later, even after adjusting for factors

including sex, age, family history of myocardial infraction, and body mass index. Such controlled experimental studies suggest that cardiovascular hyper-reactivity to stressors may be characteristic of individuals who go on to develop later hypertension or cardiovascular diseases. These findings are in line with those from epidemiological studies, which show an association among environmental stressors, higher blood pressure, and hypertension-related mortality (Harburg et al., 1973a; James & Kleinbaum, 1976).

More recent evidence demonstrating association between cardiovascular responses to laboratory mental stressors and subsequent cardiovascular risk status is provided in a comprehensive meta-analytical review study of prospective cohort studies, by Chida and Steptoe (2010). These authors found that greater responsivity to acute mental stress has an adverse effect on future hypertension status. In particular, the meta-analysis showed that both greater cardiovascular reactivity and poor cardiovascular recovery from stress were associated longitudinally with poor cardiovascular status. Specifically, incident hypertension was associated with greater stress reactivity while poorer recovery was associated increased carotid-media thickness. Additionally, both cardiovascular reactivity and recovery were associated with higher systolic and diastolic blood pressure at longitudinal follow-up. However, despite ample evidence suggesting that physiological responses to stress are implicated in the development of hypertension and cardiovascular disease, the mechanisms by which this process occurs remain speculative to date.

One mechanism linking physiological stress responses and hypertension/cardiovascular diseases involves the role of the sympathetic nervous system. It has been proposed that the links between stress/behavior and cardiovascular disease are mediated by alterations in the activation of the sympathetic nervous system

(e.g. Kamark et al., 2005), and there is evidence that essential hypertension is accompanied by sympathetic activation (Mancia, 1997). It has been postulated that such activation occurs because of exaggerated cardiovascular reactivity to environmental stressors, resulting in transient but repeated elevations in blood pressure (Kamark et al., 2005). Such stress responses may over time, lead to pathogenic structural adaptations of the cardiovascular system including vascular remodeling and endothelial dysfunction (Gibbons, 1998). These structural changes then go on to contribute to the development of hypertension (Lovallo & Gerin, 2003; Manuck, Kasprowicz, & Muldoon, 1990; Obrist, 1981; Schwartz et al., 2003).

Findings from a study by Flaa, Eide, Kjeldsen, and Rostrup (2008) support the link between sympathetic nervous system reactivity and risk for future hypertension and cardiovascular diseases. These researchers evaluated whether arterial catecholamine concentrations during rest and during laboratory stressor tasks were predictors of blood pressure at an 18-year follow-up. They found that norepinephrine and epinephrine concentrations during the mental arithmetic task significantly predicted future systolic blood pressure, implicating sympathetic activity as a possible causal factor in the development of hypertension and related cardiovascular diseases.

Given the differences in cardiovascular disease morbidity and mortality between Blacks and Whites, and the link between cardiovascular disease and cardiovascular reactivity to stressors, several laboratory studies have examined differences in patterns of reactivity between Blacks and Whites. As a result of such studies, it is now well documented that Blacks have augmented cardiovascular responses compared to Whites (e.g. Anderson, 1989; Kelsey, Alpert, Patterson, & Barnard, 2000; Light, Obrist,

Sherwood, James, & Strogatz, 1987). Kelsey and colleagues evaluated blood pressure, heart rate and impedance cardiography measures in healthy Black and White adolescents exposed to laboratory stressors. They found that Blacks exhibited both heightened myocardial and vasoconstrictive reactivity to a cold chamber stressor task compared to White individuals. Blood pressure reactivity during laboratory stressors has also been found to be more predictive of increased ambulatory blood pressure measured 3 years later, in Blacks compared to Whites. The latter was found in the CARDIA (coronary artery risk development in young adults) study, which included 316 Black and White men and women (Knox, Hausdorff, & Markovitz, 2002). These researchers concluded that greater hyper-responsivity to stress may be a risk factor for subsequent increased blood pressure in Blacks, which is indicated as one pathway leading to higher risk and prevalence of hypertension in North American Blacks compared to Whites.

Several factors have been proposed to be associated with the greater cardiovascular reactivity to stressors seen in Blacks, including higher levels of psychosocial stress and racial differences in physiological/hemodynamic responses to stressors (Calhoun, 1992). With regards to the link between higher levels of psychosocial stress and heightened cardiovascular reactivity to laboratory stressors, several potential stressors have been identified in the lives of Blacks in the United States. These include poverty, crowded housing, and exposure to racism (Anderson, Myers, Pickering, & Jackson, 1989; Calhoun, 1992; Pieterse & Carter, 2007; Wyatt et al., 2003). The influence of perceived racism, a chronic psychosocial stressor in the lives of Black individuals, and links with exaggerated cardiovascular responsivity and hypertension risk, is discussed in sections to follow. Such environmental stressors are proposed to lead

to chronically elevated levels of autonomic/sympathetic arousal in individuals dealing with these stressors (Krantz, Contrada, Hill, & Friedler, 1988). In turn, chronically high levels of sympathetic arousal are thought to interact with other risk factors leading to the higher prevalence of hypertension and cardiovascular disease found in the Black population (Kumanyika & Adams-Campbell, 1991).

Physiological differences in Blacks and Whites, particularly structural and functional differences of resistance vessels, may also contribute to greater cardiovascular reactivity and higher risk for hypertension in Blacks. In a review study of 31 articles on the topic, Taherzadeh, Brewster, van Montfrans, and VanBavel (2010) summarized evidence on racial differences in vascular reactivity and biochemical properties of resistance arteries in normotensive Black and normotensive Whites. From their findings, it is suggested that Black compared to White adults have enhanced vascular reactivity to sympathetic stimulation, attenuated responses to vasodilators, and a relatively narrow vascular lumen diameter. The authors conclude that physiological factors such as reduced vasodilation and reduced nitric oxide available in the vascular wall appear to be the most important differences in properties of resistance vessels in Black compared to White individuals, which may act in concert to contribute to higher risk for hypertension in Blacks.

Consistent with the above differences in physiological responses, Adamopoulos and colleagues (2009) found that the cold pressor task (hand immersed in iced water) provoked a higher increase in carotid-femoral pulse wave velocity in Blacks compared to Whites. This was attributed to greater increases in mean blood pressure for Blacks. Blacks compared to Whites also demonstrated greater skin micro-vascular response to the

cold pressor task. As such, the authors posit that increases in arterial stiffness and enhanced sympathetic skin vasoconstriction may be more pronounced in Blacks compared to Whites during exposure to stressors. Such differences in vascular responsivity to stress may contribute to the higher risk for development of future hypertension and cardiovascular diseases found in the Black population.

Stress and Cardiovascular Recovery. Cardiovascular recovery is defined as the extent to which blood pressure elevations persist following the termination of a stressor (Linden, Earle, Gerin, & Christenfeld, 1997), compared to cardiovascular reactivity (discussed above), which emphasizes the magnitude of the stress response. It is proposed that sustained elevated blood pressure, resulting from poorer or slower recovery, may lead to burden on the cardiovascular system, which has been linked to cardiovascular disease and end organ damage (Devereux & Pickering, 1991). Poor cardiovascular recovery from exposure to acute stressors is increasingly being recognized as an important marker of risk for hypertension and cardiovascular disease.

Research studies show that both current hypertension status and risk for hypertension have been associated with delayed cardiovascular recovery after exposure to acute stressors (Borghi, Costa, Boschi, Mussi, & Ambrosioni, 1986; Schuler & O'Brien, 1997; Steptoe & Marmot, 2005; Stewart & France, 2001). For example, in a longitudinal study with borderline hypertensive individuals, Borgi and colleagues found that poor diastolic blood pressure recovery from laboratory mental stress predicted hypertension at a 5-yr follow-up. In the study by Stewart and France, cardiovascular recovery was examined as a predictor of blood pressure change 3 years later. It was found that poor systolic blood pressure recovery from a cold pressor and a tourniquet ischemia task,

positively predicted systolic blood pressure at the 3-year follow-up. Likewise, the study by Steptoe and Marmot found that post-stress recovery from a colour/word and mirror tracing task predicted increased resting systolic blood pressure three years later, independent of control factors including age, body mass index, gender and socio-economic status. The above discussed studies suggest that cardiovascular recovery from various types of stressors are predictors of longitudinal changes in blood pressure linked to risk for development of hypertension

Interestingly, in a study employing repeated mental stress (four math task trials) with normotensive individuals, Schneider, Jacobs, Gevirtz, & O'Connor (2003) found that delayed cardiovascular recovery in heart rate and diastolic blood pressure occurred in participants even before the corresponding changes in cardiovascular reactivity were seen across trials. These authors concluded that delayed recovery may represent an early precursor to the development of hypertension, perhaps even earlier than cardiovascular reactivity. However, compared to cardiovascular reactivity, fewer research studies have focused on investigating delayed recovery as a risk factor for development of hypertension. Further research is clearly warranted in this area, especially in high risk populations. The need for such research is particularly indicated in the North American Black population, since African American ethnicity is a factor that has been found to be associated with poor cardiovascular recovery (Jackson, Trieber, Turner, Davis, & Strong, 1999), and as discussed before, Blacks have a higher prevalence rate of hypertension and related cardiovascular diseases. Research suggests that employing ecologically valid laboratory stressors that replicate real life situations may be important to understanding the link between cardiovascular recovery and hypertension risk. To this end, Krieger

(1990) found that social stress, especially discrimination/racism, may be linked to the high risk for cardiovascular diseases found in the Black population. Consistent with the later, it has been found that Blacks have a tendency towards responding with decreased cardiovascular recovery following racial versus nonracial stressors in the laboratory (Lepore et al., 2006). The current study investigates the effects of diverse laboratory stressors, including an ecologically valid social stressor (the Discrimination Recall task), on cardiovascular reactivity and recovery in a young adult Black Canadian population.

Gender, Stress, Cardiovascular Reactivity, and Cardiovascular Recovery. It is known that blood pressure and the prevalence of hypertension are higher in men than in women in their reproductive years (Oparil & Miller, 2005). While the mechanisms for gender differences in blood pressure are unclear, the effects of the sex hormones are implicated, with androgens contributing to higher blood pressure observed in males, and female ovarian hormones contributing to lower blood pressure in women in their premenopausal years (Oparil & Miller, 2005). Gender differences have also been found in physiological responses to laboratory stressors, although psychophysiological research in general have mostly focused exclusively on male participants, or have included both males and females but lump the data together in analyses. The scarcity of studies investigating gender differences in this area of research is largely due to the complexities of controlling for levels of female sex hormones across the various phases of the female menstrual cycle (Blair, 2007). None-the-less, significant findings for gender differences in both cardiovascular reactivity and cardiovascular recovery are indicated in the research. This literature is reviewed below.

Gender and Cardiovascular Reactivity. In general, it has been found that males tend to demonstrate greater cardiovascular reactivity than women across diverse stressor tasks in laboratory protocols (e.g. Girdler et al., 1990; Matthews, Gump, & Owens, 2001; Stoney, Matthews, McDonald, & Johnston, 1988; Vogele et al., 1997). For example, in a laboratory study with 62 men and women (ages 24 – 46), Matthews and colleagues found that relative to women, men demonstrated greater diastolic blood pressure reactivity to a mental arithmetic and public speaking stressor tasks. It has also been found that males tend to respond with a pattern of cardiovascular reactivity marked by greater blood pressure/vascular responses, while women tend to react with greater heart rate/myocardial responses (Allen et al., 1993; Fichera & Andreassi, 2000, Girdler et al., 1990; Vogele et.al., 1997).Vascular and myocardial patterns of reactivity are sympathetic response modes, which underlie blood pressure and heart rate response patterns.

Girdler et al. (1990) found that that males reacted with greater vascular reactivity, characterized by increased total peripheral resistance compared to women, whose reaction to stress was myocardially regulated, as evidenced by greater increases in heart rate and cardiac output. Similarly, Fichera and Andreassi (2000) found that men reacted with greater mean arterial blood pressure and greater diastolic blood pressure compared to women, who had greater increases in heart rate, during a public speaking stressor task. These differences between men and women may be linked to differences in gender physiology including hormonal differences (discussed above). Additionally, it has also been found that women have lesser concentrations of, and/or are less sensitive to peripheral alpha and beta-adrenergic receptors than men (Freedman, Sabharwal, & Desai, 1987), which may contribute to lower blood pressure reactivity compared to men.

Additionally, women may have decreased vagal activity, which may contribute to higher heart rate reactivity in women compared to men (Collins & Frankenhauiuser, 1978).

It is noteworthy, that some studies have reported mixed findings in gender differences in cardiovascular reactivity to laboratory stressors, that vary from the typical vascular/myocardial patterns discussed above (e.g. Stoney et al., 1988). These mixed findings, it has been suggested, may be understood in terms of gender-relevance of stressor tasks used (Kolk, & van Well, 2007). Kolk and van Well states that "a gender-relevant stressor is characterized by contexts associated with sociocultural norms about what is appropriate for women and men in regard to which it is more important to succeed for men and women, respectively, thereby having distinct effect on effort" (p. 197). These researchers manipulated gender relevance of the stressor task by administering the cold pressor task with masculine, feminine or gender-irrelevant introductions, to 169 normotensive individuals between the ages of 18 and 60 years. It was found that mismatch between gender and gender-relevance of stressor task produced high vascular reactivity on systolic and diastolic blood pressure, as well as for total peripheral resistance, along with low heart rate, stroke volume and cardiac output, across all participants (both males and females).

Another factor that may influence gender differences in cardiovascular reactivity to stressor tasks is the type of physiological responding typically triggered by task, i.e. alpha/vascular versus beta/myocardial responding. The present research study will examine gender differences in cardiovascular reactivity across three laboratory stressors that vary on task characteristics, with 53 Black Canadian adults. Preliminary analyses of task differences were examined in previous analyses with the first 17 participants tested

in this study, for the writer's master's thesis (Saunders, 2004). Gender differences were not examined at that time due to a very small sample size. The findings from those preliminary analyses are briefly discussed below.

Cardiovascular reactivity and task-relevant responses. Saunders' 2004 master's thesis investigated patterns of cardiovascular reactivity to different stressor tasks including a cold pressor task, known to elicit an alpha-adrenergic pattern of responding; a computerized math task, known to elicit a beta-adrenergic pattern of responding; and a discrimination recall task, known to elicit a mixed alpha/beta pattern of responding. The main findings showed significant diastolic blood pressure reactivity to all stressor tasks; however the hemodynamic profiles were strikingly different. While there were no significant differences in the magnitude of diastolic blood pressure for the cold pressor and discrimination recall tasks (both of which were greater than the math task), the underlying hemodynamic patterns were vastly different, with the former showing a vascular profile (large increase in total peripheral resistance) and the latter showing a predominantly cardiac/beta-adrenergic profile (large increase in heart rate and cardiac output, with a small decrease in total peripheral resistance) (Saunders, 2004).

The strong cardiac reactivity pattern observed for the discrimination recall task in Saunders' (2004) master's thesis was unexpected; however a possible reason for this reactivity pattern may be related to the speech characteristic of the task, which have been suggested to evoke blood pressure changes on its own (e.g. Kamarck, 1992). Additionally, the social-evaluative challenge of speaking to an audience is known to evoke significant cardiac activation (e.g. Bongard, al'Absi, & Lovallo, 1998). Moreover, gender differences, including physiological differences between men and women

affecting blood pressure responses, may have added to the patterns of cardiovascular reactivity found: Research shows that men are more likely to be vascular reactors and women are more likely to be myocardial reactors (e.g. Gregg, James, Matyas, & Morsteinsson, 1999; Turner, 1994; Allen et al., 1993).

The present study improves upon the writer's master's thesis by including gender as an independent variable, which is examined in multivariate analyses with other independent variables including sodium and psychosocial variables (anger expression and perceived racism), all known to influence cardiovascular reactivity to stressors. Additionally, the present study investigates the effects of the same independent variables on delayed cardiovascular recovery, another identified risk factor for hypertension and cardiovascular diseases. The present study also improves upon the writer's master's thesis with the addition of an ambulatory blood pressure protocol, investigating the effects of sodium, anger expression, and perceived racism, on ambulatory sleep and wake blood pressure measures, and on nocturnal blood pressure dipping. The above mentioned variables are discussed in relevant sections to follow.

Gender and Cardiovascular Recovery. A review of the literature found fewer studies looking at gender differences in cardiovascular recovery compared to cardiovascular reactivity to laboratory stressors. Again, this scarcity is likely due to deterrence of conducting psychophysiological research to examine gender differences because of complexities involved in controlling for levels of female sex hormones across phases of the menstrual cycle (Blair, 2007). In reviewing the limited research on gender and cardiovascular recovery, mixed findings emerge, which are discussed below.

A few studies have investigated the influence of chronic stress on cardiovascular reactivity and recovery from acute stressors in the laboratory (e.g. Chatkoff, Maier and Klien, 2010; Matthews et al., 2001). Matthews and colleagues investigated whether the latter relationship was gender specific, in a sample of 31 men and 31 women, ages 24 – 46 (mean age 34.6). Laboratory stressors included mental arithmetic and public speaking tasks. Chronic stress was measured with the perceived stress scale. Relative to women, men were found to have greater diastolic blood pressure reactivity to stressor tasks, consistent with other studies in the area (e.g. Girdler et al., 1990; Stoney, et al. 1988). Moreover, men also showed greater delayed systolic and diastolic blood pressure during cardiovascular recovery from laboratory stressors. Matthews et al. suggest that such delayed recovery may help to understand the increased risk of cardiovascular diseases seen in men compared to women, in young-middle adulthood.

Gender characteristics, particularly gender social roles, have also been shown to influence patterns of cardiovascular recovery from stress, particularly in research studies employing harassment/anger provocation stressor tasks, (e.g. Hokanson, Burgess, & Cohen, 1963; Lai & Linden, 1992). Hokanson and colleagues found that blood pressure recovered faster for males when they had the opportunity to express their anger compared to not having an opportunity for anger expression. Females on the other hand did not show differences in blood pressure recovery under the same two conditions. Similarly Lai and Linden found that opportunity to express anger after completing a math task under anger provocation, facilitated heart rate and diastolic blood pressure recovery in men but not in women. Hokanson and colleagues attributed the above discussed gender differences in cardiovascular recovery to differential socialization between men and

women, mainly the perceived need of women to confirm to social expectations to be peacekeepers and to remain non-aggressive.

It also appears that the relationship between gender and cardiovascular recovery is mediated by the degree to which individuals express anger to harassment. Along these lines Faber and Burns (1996) found that men who scored high on anger-out and who expressed much anger, and women who scored low on anger-out and who expressed little anger, showed sustained systolic blood pressure activity during the recovery period in a laboratory stress study. In another laboratory study utilizing emotional stressor tasks (but without a harassment component), Glynn, Christenfeld, and Gerin (2002) found that while both males and females demonstrated significant blood pressure reactivity and delayed recovery after exposure to stressor tasks, males demonstrated greater elevations in blood pressure than females across both periods. That is, males showed greater reactivity and greater delayed recovery than females.

Despite the inconsistencies, there is a trend in the research literature suggesting that in general, males tend to demonstrate greater delays in cardiovascular recovery compared to females after exposure to laboratory stressors. The present study investigates gender differences in cardiovascular reactivity and recovery across three diverse laboratory stressors, including a physical, a social, and a mental stressor task, in a young adult sample of Black Canadian men and women.

Sodium, Stress, Cardiovascular Reactivity, and Cardiovascular Recovery. From the literature reviewed thus far, several lines of evidence implicate cardiovascular reactivity to and recovery from stressful stimuli, as risk factors for hypertension development. Another factor thought to have an important controlling influence on blood pressure and subsequent

hypertension, is dietary sodium; an idea that was advanced many decades ago. There is a plethora of evidence from epidemiological, genetic, and animal studies showing that dietary sodium intake plays an important role in regulating blood pressure. Epidemiological studies suggest that variations in the prevalence of hypertension among different societies and cultures may be partly due to differences in salt intake. For example, societies where salt consumption is less than one gram per day have little or no hypertension (Dahl, 1972; Poep, 1976). On the other hand, societies such as those in regions of Japan, where the salt ingestion is more than 20 grams per day, have hypertension in 30 to 40 percent of the adult population (Freis, 1976; Sasaki, 1962). Additionally, randomized studies have shown that a reduction in salt intake lowers blood pressure in hypertensive and normotensive individuals (He & MacGregor, 2002).

Research shows that the effects of sodium intake vary among populations in the Western Hemisphere. For example, Weinberger, Miller, Luft, Grim, and Fineberg (1986) have found racial differences in blood pressure responses in studies involving sodium manipulations, with a greater prevalence of sodium sensitivity among Black individuals. Weinberger defined sodium sensitivity as a decrease in mean arterial pressure greater than or equal to 10 mmHg after sodium restriction/depletion, and defined sodium resistance as a decrease less than or equal to 5 mmHg, under the same conditions. Falkner and Kushner (1990) reported findings of sodium-sensitive increases in blood pressure in 18% of Whites compared with 37% of Black young adults. A greater proportion of sodium-sensitive individuals have been found in both normotensive and hypertensive Black populations (Sowers et al., 1988; Zemel, Gualdoni, & Sowers, 1988), leading to suggestion that greater retention of excess sodium and water causes volume overload, which may contribute to the

development of sustained high blood pressure, hypertension, and related cardiovascular diseases, in the Black population. In other words, significant differences in sodium homeostasis in Blacks and Whites have been linked to increased sodium sensitivity in Blacks. Consistent with the latter, it has been found that Blacks take longer to excrete a sodium-load compared to Whites (Luft et al., 1979). Likewise, in a more recent study with Black and White adolescents, it was found that Blacks had greater sodium retention compared to Whites, which may contribute to underlying racial differences in susceptibility to hypertension (Palacios et al., 2004). It has been found that sodium sensitivity not only increases the risk for essential hypertension, but also the risk of having blunted nocturnal blood pressure dipping, and risk for the development of left ventricular hypertrophy (Weinberger, Finebert, Finebert, & Wineberger, 2001).

As discussed in a recent article by Gleiberman (2009), sodium sensitivity is associated with several physiological factors including renal functioning and the renin-angiotensin-aldosterone system (a major mechanism for maintaining sodium and potassium homeostasis in the body). It has been found that plasma renin activity, one step in the pathway of the renin-angiotensin-aldosterone system, is lower in Blacks than in Whites (e.g. Adlin, Marks, & Channick, 1982; Creditor & Loschky, 1968), which may be linked to increased sodium sensitivity found in Blacks. Additionally, Blacks compared to Whites demonstrate a greater fall in systolic and diastolic blood pressure when changing from a high to a low salt diet, which may in part be due to a less responsive renin-angiotensin-aldosterone system in Blacks (He, Markandu, Sagnella, & MacGregor, 1998).

In addition to differences in physiological handling of sodium and effects on blood pressure discussed above, evidence suggests that there may be an interaction between dietary

sodium and behavioral stress in influencing exaggerated cardiovascular reactivity and related risk for hypertension development. For example, in a study by Anderson, Kearns, and Better (1983), where dogs were given continuous saline infusion (salt-loading) and shock-avoidance stress for two weeks, the combination of high salt and daily stress resulted in significant and progressive increases in blood pressure. Importantly, however, neither the saline infusion nor the avoidance stress alone was sufficient to raise blood pressure. Thus, sodium and stress appear to interact to influence cardiovascular reactivity and augment blood pressure levels. This is supported in studies such as that by Miller, Friese, and Sita (1995), who found that sodium-loading interacted with laboratory stress to increase cardiovascular reactivity in individuals with a family history of hypertension. Miller and colleagues suggest that the exposure to both stress and a high sodium intake may serve to potentiate sympathetic nervous system activity underlying cardiovascular reactivity.

While the mechanisms by which sodium affects blood pressure are not clear, studies implicate the role of the sympathetic nervous system (Somova, Khan, & Chetty, 1998). In a study with Dahl rats, Somova and colleagues showed that the effect of sodium-loading on blood pressure was mediated by increased activity of the sympathetic nervous systems while sodium restriction showed reduced sympathetic nervous system responsiveness. Several lines of evidence implicate sodium as a mediator of heightened blood pressure responses in Blacks. It has been shown that Blacks secrete less sodium in urine than Whites as well as exhibit greater cardiovascular reactivity to sodium-loading (Luft, Grim, & Weinberger, 1985). Studies also indicate that, in both humans and spontaneously hypertensive rats, sodium may augment blood pressure through heightened vasoconstriction than by increasing cardiac output (Nilsson, Fly, Fribert, Kalstrom, & Folkow, 1985). This is important, since

enhanced vascular reactivity is found to be characteristic of Black compared to White individuals, which may be linked to differential risk for development of hypertension (Falkner & Kushner, 1989; Fredrikson, 1986; Stein, Lang, Singh, He, & Wood., 2000; Thomas, Nelesen, Malcarne, Ziegler, & Dimsdale, 2006). Consistent with increased vascular reactivity in Blacks compared to Whites, research shows that under acute stress, Blacks demonstrate significantly higher plasma levels of the vasoactive endothelium peptide ET-1, a substance known to induce long-lasting vasoconstriction and modulate sympathetic system-mediated contractility (Ergul, 2000).

Taken together, the above discussed evidence suggests that sodium potentiates sympathetic nervous system-induced vascular reactivity to stress, which may be a principal physiological mechanism involved in racial differences in risk for hypertension. Fray and Douglas (1993) proposes an interesting conceptualization of the pathogenesis of hypertension that can be divided into three phases. The first phase likely involves an increase in stress response factors including catecholamines, which leads to increased vascular hemodynamics underlying blood pressure, mainly increased total peripheral resistance. In the second phase, it is proposed that genetic factors such as sodium sensitivity likely come into play, leading to intravascular volume overload with heightened vascular reactivity, which over time leads to the third phase of established hypertension.

While there have been studies looking at relationships between sodium, stress and cardiovascular reactivity as they may related to hypertension risk, there have been few if any studies, looking at relationships between sodium, stress and cardiovascular recovery. Given increasing evidence of the importance of cardiovascular recovery as a potential risk factor in the development of hypertension, it is important to investigate its association with other

prominent risk factors, namely stress and dietary sodium. The present study expands on the literature to date by examining relationships between these risk factors in the Black population.

Anger Expression and Cardiovascular Reactivity. Investigators have focused attention on examining the role of anger on cardiovascular reactivity to laboratory stressors, given that the emotion of anger is known to elicit strong cardiovascular arousal (Starner & Peters 2004), and exaggerated cardiovascular reactivity is identified as a potential risk factor for the development of hypertension and cardiovascular disease (Falkner, 1984; Falkner et al, 1981; Treiber et al., 2003). Research suggests that the style of anger expression (e.g. anger-in versus anger-out), as opposed to the mere experience of anger, is associated with heightened cardiovascular reactivity (e.g. Siegman & Boyle, 1992). While the experience of anger refers to subjective feelings of annoyance, irritation or rage, and is dependent on the appraisal and meaning given to events, anger expression refers to how individuals cope with angry feelings, or in other words, specific strategies or behaviours that individuals use in response to feeling angry. For example, anger-in or anger suppression occurs when angry feelings are experienced but not expressed, while anger-out occurs when an individual copes with the stress of an anger-provoking situation by venting the anger outwardly (Spielberger et al., 1985). Research suggests that anger expression is linked to cardiovascular disease through association with physiological reactivity (Engebretson, Matthews, & Scheier, 1989; Suarez, Kuhn, Schanberg, Williams, & Zimmermann, 1998).

The association between unexpressed anger or anger suppression and essential hypertension is longstanding, rooted in Franz Alexander's original hypothesis that

chronic inhibition of anger leads to sustained elevations in blood pressure (Alexander, 1939). Suppression of anger has been found to be associated with higher blood pressure at rest or elevated cardiovascular reactivity to stress (Cottington, Brock, House, & Hawthorne, 1985; Gentry, Chesney, Gary, Hall, & Harburg, 1982; Harburg et al. 1973b; Mills & Dimsdale, 1993; Jorgensen & Kolodziej, 2007; Poole, Snieder, Davis, Trieber, 2006). For example, Jorgensen and Kolodziej found that anger suppression was associated with the highest levels of diastolic and systolic blood pressure, as well as heart rate reactivity, in a sample of 74 college students exposed to a laboratory story telling stressor task. The suppression of anger is thought to induce exaggerated cardiovascular reactivity, thereby increasing the risk for hypertension.

Mills and Dimsdale (1993) found that individuals who routinely suppressed their anger had greater systolic blood pressure reactivity to a math stressor, as well as demonstrated increased beta-adrenergic receptor sensitivity. Similarly, Poole and colleagues (2006) found that Blacks who were carriers of the beta-2 adrenergic receptor gene (ADRB2) and had high anger-in, showed the highest levels of resting systolic blood pressure and total peripheral resistance (TPR), as well as the greatest TPR reactivity to the cold pressor task. As such, it is speculated that altered functioning of beta-adrenergic receptors is a potential mechanism by which anger-in has its effects on increased cardiovascular reactivity and associated risk for hypertension.

In prospective studies, research shows a link between anger expression and coronary heart disease (Chang, Ford, Meoni, Wang, & Klag, 2002; Davidson & Mostofsky (2010). For example, Davidson and Mostofsky found that anger expression, evaluated by the coding of videotaped interviews, was associated with incident coronary

heart disease over a 10-year follow-up period, in a randomly selected, population based sample of Canadian men and women. Additional supportive evidence implicating anger suppression as an underlying risk factor in the anger-blood pressure-heart disease association includes findings that suppression versus overt expression of anger is linked to increased carotid arterial stiffness and other subclinical indices of coronary heart disease (Anderson, Metter, Hougaku, & Najjar, 2006). It has also been found that suppressed anger is associated with higher all-cause mortality ((Julius, Harburg, Cottington, & Johnson, 1986). In a 12-year study, Julius and colleagues found that participants who suppressed their anger had twice the mortality risk compared to those who expressed their anger, even while controlling for age, sex and education. Moreover, suppressed anger measures significantly interacted with elevated blood pressure to predict the highest mortality risk. However, despite extensive research to date, the mechanisms underlying the association between anger expression, blood pressure responses, and cardiovascular disease, remain obscure at this time.

Racial differences are implicated in the association between anger expression and cardiovascular risk, which may contribute to the higher prevalence of hypertension and cardiovascular diseases found in Blacks compared to Whites. To date, most studies examining the association between anger expression and cardiovascular reactivity have been conducted among Whites, with fewer studies including Black participants. However, of the studies available to date, findings suggest that suppressed anger and hostility are more predictive of cardiovascular risk factors in Blacks compared to Whites (Finney, Stoney, & Engebretson, 2002, Harburg, Blakelock, & Roeper, 1979; Harburg et al., 1973b). Finney, and colleagues looked at relationships between anger expression and

hostility (two related concepts), and cardiovascular reactivity, to a laboratory speech stressor task, in a sample of 46 healthy American Blacks and Whites (mean age 20.15 years). These researchers found that Blacks with high anger-in and low cynicism had the greatest cardiovascular reactivity to the laboratory stressor. These findings suggest that anger suppression in coping with stress is an important risk factor linked to risk for hypertension and cardiovascular disease in the Black population. This is particularly relevant, given that Blacks compared to Whites are known to be exposed to more chronic stress in daily life, which may trigger frequent exaggerated cardiovascular responses associated with risk for hypertension (e.g. Anderson, McNeilly, & Myers, 1993). This risk is conceivably increased if Black individuals deal with chronic stressors through coping by anger suppression.

Further investigation of the anger expression-cardiovascular reactivity association in the Black population is clearly warranted. The present study investigates the relationship between anger expression (anger-in and anger-out) and cardiovascular reactivity (and recovery), in a Black Canadian population. Of specific importance, this study employs a Discrimination Recall stressor task, chosen to reflect salient features of the social environment associated with anger induction in the daily life of Western Blacks.

Anger Expression and Cardiovascular Recovery. As previously discussed, it is argued that delayed cardiovascular recovery may be a critical factor underlying the link between stress and hypertension/cardiovascular disease (Borghi et al., 1986; Schuler & O'Brien, 1997). Cardiovascular recovery appears to be particularly relevant for the emotion of anger, since research shows that delayed recovery from anger eliciting

stressors is greater than recovery from other laboratory stressors (Engebretson et al., 1989; Lai & Linden, 1992; Linden et al., 1997). Another interesting finding is that some individuals show delayed recovery to anger provoking stressors, despite insignificant cardiovascular reactivity to these stressors (e.g. Anderson, Linden, & Habra, 2005), suggesting that cardiovascular recovery from anger-related stressors may be the more important factor influencing cardiovascular health.

Given the link between anger and cardiovascular recovery, and the link between cardiovascular recovery and hypertension/cardiovascular disease risk, it would follow that factors influencing the relationship between anger and cardiovascular recovery are potentially implicated in the development of hypertension and cardiovascular diseases (Siegman, 1994). As such, it is important to investigate the relationship between anger coping strategies and cardiovascular recovery. To this end, research studies have shown that cardiovascular recovery is influenced by anger expression styles. For example, Lai and Linden (1992) found that anger-out subjects, given a chance to express frustration, showed blood pressure recovery more quickly than anger-in subjects who constrained expression of feelings. Such delayed recovery is proposed to be the underlying mechanism in the link between anger and cardiovascular disease (Brosschot & Thayer, 1998).

Brosschot and Thayer (1998) proposed that anger inhibition/suppression is influenced by low vagal tone, which is known to have a major regulatory role in cardiovascular recovery, and that excessive or frequent anger inhibition leads to a persistent decrease in cardiovascular recovery speed. The resulting chronic elevation of blood pressure is proposed to lead to changes where blood pressure control switches from

a cardiac to a primarily vascular profile (Amerena & Julius, 1995). Evidence of such changes have been noted in studies with extended lab stressors (e.g. Carroll, Cross, & Harris, 1990) and implicated in the developmental course of hypertension. Given the above, Brosschot and Thayer concluded that frequent or chronic anger inhibition may lead to shifts to long term vascular control of blood pressure and associated increased risk for hypertension and cardiovascular disease.

It has been argued that in social reality, incidences of anger inhibition (anger-in) outnumber incidences of anger expression (anger-out), given that our societal norms does not allow for ready expression of anger (Brosschot & Thayer, 1998). It follows that, populations exposed to frequent and/or chronic stressors, particularly those that are anger provoking, might be at particular risk for hypertension and cardiovascular risk. There is ample evidence of the presence of chronic stressors, including discrimination and racism, in the lives of Blacks in North America (Anderson et al., 1992; Clark, Anderson, Clark, & Williams, 1999; Williams, 1999). Anger linked to racial stressors is potentially especially detrimental to cardiovascular recovery in Black individuals. For instance, Lepore et al. (2006) evaluated cardiovascular responses to a racial/anger provoking stressor versus a non-racial stressor, in a sample of 40 Black and 40 White women (ages 16 – 41), and found that relative to Whites, Blacks exhibited greater diastolic blood pressure reactivity and marginally greater systolic blood pressure to the racial compared to the non-racial stressor. Additionally, Blacks exhibited marginally greater delayed systolic blood pressure recovery following the racial stressor compared to the non-racial stressor.

It is argued that anger inhibition and delayed cardiovascular recovery may be major contributing factors to the high prevalence of hypertension and related cardiovascular diseases found in the Black population. Indeed, studies have shown that suppressed anger/hostility in Blacks is associated with delayed cardiovascular recovery (Gentry, 1985; Dorr, Brosschot, Sollers, & Thayer, 2007). For example, Dorr and colleagues showed that Blacks who inhibited their anger in a laboratory stress protocol, had delayed total peripheral resistance recovery. Such a finding is consistent with research showing that Blacks have an increased tendency towards a vascular cardiovascular response profile which is implicated in risk for hypertension and cardiovascular diseases (e.g. Anderson, Lane, Muranaka, Williams, & Houseworth, 1988; Stein et al., 2000; Thomas et al., 2006). Despite research to date, the mechanisms underlying the association between anger expression, cardiovascular recovery, and hypertension and cardiovascular diseases, remain obscure. The present study contributes to this area of research by looking at the association between anger expression (anger-in and anger-out) and cardiovascular recovery, in response to an ecologically valid social stressor - a discrimination recall laboratory task - in a Black Canadian population.

Perceived Racism and Blood Pressure. As previously discussed, it is widely recognized that racial disparities in health, particularly higher risk for hypertension among Blacks, exist in the Western Hemisphere. It is also recognized that Blacks, as a minority population, share similar social experiences as a result of their social group status in the Western world, including chronic social and environmental stressors (Thompson et al., 2002). These include psychosocial stress associated with exposure to racism/discrimination, which has been proposed as a risk factor for elevated blood

pressure in Blacks (Clark et al., 1999; Krieger & Sidney, 1996; Roberts, Vines, Kaufman, & James, 2007). Racism has been defined as "the beliefs, attitudes, institutional arrangements, and acts that tend to denigrate individuals or groups because of phenotypic characteristics or ethnic group affiliation" (Clark et al., 1999, p. 805). Racial discrimination is a stressor for the individual who is on the receiving end of such behaviours. It has been suggested that the effects of racism can be understood within the framework of stress, appraisal and coping as proposed by Lazarus and Folkman (1984), which emphasizes that the effects of stress are mediated by a person's appraisals or coping responses to the stressor. As such, 'perceived' racism, rather than objective racism per se, is proposed to influence stress responses.

Several research findings suggest a positive association between perceived racism and high blood pressure (Din-Dzietham et al., 2004; Krieger & Sidney, 1996; Steffen et al., 2003). In a 7-year longitudinal study with young adults, Krieger and Sidney found that blood pressure was lower in those who reported little or no experience with racial discrimination. In the same vein, Din-Dzietham and colleagues found that Blacks who reported more stress from perceived racism at work were more likely to also report having a diagnosis of hypertension. Additionally, in a study by Steffen et al. (2003), it was found that perceived racism was related to higher ambulatory blood pressure during the day/wake period, in Black men and women with normal to mildly high blood pressure. There are therefore significant findings that implicate perceived racism as a critical chronic stressor in the lives of Black Canadians, which likely contributes to the higher prevalence rate of hypertension in this population.

Perceived Racism, Cardiovascular Reactivity, and Cardiovascular Recovery. Racial discrimination experienced by Black Americans has been shown to be associated with elevated cardiovascular reactivity (Clark et al., 1999; Fang & Myers, 2001), which (as previously discussed) is a proposed marker of mechanisms culminating in hypertension and related cardiovascular disease. Studies show that exposure to racist and other personally relevant psychosocial stimuli, compared with other types of stressors, are associated with significantly higher cardiovascular reactivity (Armstead, Lawler, Gordon, Cross, & Gibbons, 1989; Fang & Myers, 2001). It is suggested that perceived racism may result in such strong cardiovascular effects, because of its deep psychological/affective impact (Fang & Myers, 2001). Interestingly, in a study of adult Black men investigating the effect of exposure to audiotaped scenarios of blatant (racist) versus neutral (non-racist) discrimination provocations, Merritt, Bennett, Williams, Edwards, and Sollers (2006) found that the latter produced higher cardiovascular reactivity, particularly diastolic blood pressure, in participants who perceived higher levels of racism. Such findings suggest that heightened cardiovascular reactivity is likely to be experienced by individuals who generate racist attributions (perceived racism) to ambiguous provocative interpersonal situations. This is particularly relevant, given that in our present day society, older, more blatant forms of racism are increasingly being replaced by more subtle forms of racism, which are more ambiguous in nature (Dovidio, 2001). Merritt and colleagues note that, given the frequency with which Blacks in Western societies report ambiguous discriminatory situations, perceived racism may be a critical factor explaining some of the variance in Black-White differences in cardiovascular reactivity and hypertension risk.

Despite significant findings for the link between perceived racism and cardiovascular reactivity/blood pressure, and its implications for the risk of hypertension in the Black population, few studies have investigated these relationships within this population. Moreover, little has been done to address questions of the possible association between perceived racism and cardiovascular recovery. Given that perceived racism is likely an anger eliciting stressor, and given that anger eliciting stressors are positively associated with delayed cardiovascular recovery and possible subsequent risk for hypertension and cardiovascular disease (previously discussed), it is important to investigate the relationship between perceived racism and cardiovascular recovery. The current research looks at the association between perceived racism, and cardiovascular reactivity and recovery, during exposure to a discrimination recall laboratory task, within a Black Canadian population. This stressor task involved the re-experiencing through narrative recall, of a past personally stressful experience where the individual felt discriminated against.

Ambulatory Blood Pressure

Ambulatory blood pressure monitoring, compared to measurement of laboratory blood pressure changes, is unique in its ability to provide frequent assessments of blood pressure during routine activities and a range of situations across daily life, and allows for the assessment of diurnal variation of 24-hr blood pressure (Mancia et al., 1997). Research shows that laboratory blood pressure measures are poor predictors of 24-hr ambulatory blood pressure as well as diurnal variation in blood pressure (Kamarck & Lovallo, 2003; Langewitz, Ruddel, Schachinger, & Schmieder, 1989), therefore lacking in generalizabitity to real life situations. As such, it is argued that ambulatory blood

pressure monitoring is superior to laboratory procedures in investigating cardiovascular health risk, and critical gaps in research remain with studies that predominantly focus on measurement of blood pressure in laboratory stress protocols. Fewer studies have utilized a protocol looking at both cardiovascular responding to laboratory stressors, and 24-hr ambulatory sleep/wake blood pressure and blood pressure variability/nocturnal dipping, within the same sample population.

The present research addresses the above mentioned shortcomings in the current research literature by looking at cardiovascular activity in a broad and inclusive study involving both a laboratory stress protocol and a 24-hr ambulatory blood pressure monitoring protocol, in a sample of 53 Black Canadians. Although laboratory and ambulatory blood pressure measures are not compared in this study (following from research showing poor associations between the two, discussed above), this study aims to shed light on factors affecting blood pressure in a comprehensive manner by looking at laboratory (controlled) and ambulatory (naturalistic) findings within the same sample population. A homogeneous sample across both settings enhances control of extraneous factors that may influence variability in analyses and increases chances of identifying patterns that may characterize blood pressure and hypertension risk in this sample population.

Diurnal Variation of Blood Pressure. It is generally known, that for both males and females, blood pressure varies in a diurnal manner, being higher during the day/wake period and lower at night/sleep period. Examining differences in diurnal blood pressure variation is important as such variation have been found to be associated with higher rates of cardiovascular events and hypertension related target-organ damage (Kikuya et al.,

2005; Murphy et al., 1991; Parati et al., 1987). The ambulatory blood pressure protocol in the present study allows for investigation of factors influencing wake and sleep blood pressure, and nocturnal non-dipping blood pressure, which have been linked to risk for development of hypertension in the Black population. Associations between the various factors investigated in the ambulatory protocol in this study are discussed in sections to follow.

Nocturnal Blood Pressure Dipping. Investigations of day-night variations in blood pressure have distinguished various patterns, particularly 'dipper' and 'non-dipper' patterns. Individuals whose blood pressure falls normally at night are referred to as 'dippers'. Dippers have a typical pattern characterized by a 10% or greater drop in sleep/nighttime blood pressure compared to awake/daytime blood pressure. On the other hand, 'non-dippers' are individuals who experience less that a 10% drop in blood pressure at night (Routledge & McFetridge-Durdle, 2007). Approximately 25% of individuals with hypertension are characterized by a non-dipping profile (Pickering & Kario, 2001). Non-dippers, by definition, are exposed to a higher blood-pressure load over 24-hrs than dippers, which may contribute to increased risk for cardiovascular events (Sherwood, Steffen, Blumenthal, Kuhn, & Hinderliter, 2002). In addition, research studies show that a non-dipping blood pressure pattern is associated with hypertension related end organ damage (Parati et al., 1987). While the mechanisms underlying non-dipping blood pressure pattern are not clear, there is evidence to suggest that it may be related to a dysfunction of autonomic nervous system activity, particularly excessive activation of the sympathetic nervous system (Sherwood et al., 2002). There is also evidence suggesting an association between non-dipping blood pressure and various

demographic, physiological and psychosocial factors, including African American ethnicity (Profant & Dimsdale, 1999), sodium-sensitivity (Damasceno et al., 2000), anger (Thomas, Nelesen, & Dimsdale, 2004), and chronic stressors (Smith, Wentworth, Neaton, Stamler, & Stamler, 1996).

A consistent finding in the research literature is that non-dipping blood pressure is more prevalent in Western Blacks than Whites, both for Blacks with and without hypertension (Profant & Dimsdale, 1999). Studies have sought to examine whether such differences are true racial differences or consequences of environmental/psychosocial factors, by comparing blood pressure variation in American Whites, American Blacks and Blacks in South Africa, matched for daytime blood pressure (e.g. Fumo et al., 1992). It was found that American Whites and South African Blacks experienced a similar fall in nighttime blood pressure, while American Blacks had significantly higher nighttime blood pressure. Such findings suggest that differences in night-time blood pressure patterns between American Whites and Blacks may be related to psychosocial or environmental factors. Along these lines, Wilson, Kliewer, Teasley, Plybon, and Sica (2002) found a positive association between exposure to violence and non-dipping blood pressure in Black American adolescents. Exposure to chronic stressors and chronic activation of the sympathetic nervous system may influence the high prevalence of non-dipping blood pressure found in Western Blacks, and the associated high prevalence of target-organ damage and cardiovascular health risk observed in this population (Profant & Dimsdale, 1999). It is therefore important to investigate potential psychosocial factors linked to non-dipping blood pressure in the Black population.

Gender and Ambulatory Blood Pressure. Research shows that mean arterial blood pressure is higher in both normotensive and hypertensive men than in women, in all ethnic groups, as determined by findings of the Third National Health and Nutrition Survey with Americans 18 years and older (Burt et al., 1995), and other related studies (e.g. Stamler, Stamler, Reidlinger, Algera, & Roberts, 1976). Gender differences in blood pressure are typically first identified in adolescence and found to persist throughout adulthood until around the sixth decade of life (Himmelmann, Svensson, & Hansson, 1994; Yong, Kuller, Rutan, & Bunker, 1993). A corresponding higher prevalence of hypertension among men compared to women is found, with this pattern becoming reversed around age 60 when it is found that the prevalence of hypertension becomes higher in women than in men (Burt et al., 1995). While blood pressure is generally higher in men than in women, the reverse is found for heart rate, i.e. females tend to have higher heart rate than males (Stoney, Davis & Matthews, 1987). The reasons for such gender/sex based differences in normal physiology and/or in predisposition to hypertension are not known, however it has been suggested that genetic differences and/or effects of the sex steroid hormones (previously discussed) may be implicated (Blair, 2007). It is expected that these gender differences in blood pressure (higher in males and lower in females) and heart rate (higher in females and lower in males), will be observed in ambulatory measures for both wake and sleep periods, in the present study with Black Canadians.

Sodium, Ambulatory Blood Pressure, and Blood Pressure Dipping. As reviewed in the article by Meneton, Jeunemaitre, de Wardener, and Macgregor (2005), research data strongly supports a positive relationship between dietary sodium intake and blood pressure levels, especially in those susceptible/sensitive to sodium. Investigations

have found that normotensive adults on short-term sodium-loaded diets show an increase in ambulatory blood pressure (e.g. Rikimaru et al., 1988; Schorr, Turan, Distler, Sharma, 1997). The present study looks at the effect of short-term sodium-loading on ambulatory blood pressure during day/wake and night/sleep periods, in a Black Canadian population. Of specific importance, the present study investigates the effect of dietary sodium on nocturnal blood pressure dipping, which, as previously discussed, is strongly implicated in risk for development of hypertension in the Black population.

Dietary sodium has been identified as an important factor affecting nocturnal blood pressure dipping, particularly for individuals who have been identified as sodium-sensitive (blood pressure increases more than 10% with sodium-loading) compared to those identified as sodium-resistant (blood pressure increase is less than 10% with sodium-loading) (Uzu et al., 1996). It has been found that up to 73% of hypertensive and 36% of normotensive Blacks are sodium sensitive compared to 56% and 29% respectively in Whites (Weinberger et al., 1986). Uzu et al. (1996) found that for individuals with essential hypertension who are sodium-sensitive, blood pressure fails to fall at night. Uzu et al. (1997) further showed that sodium-sensitive hypertensive individuals shifted from a non-dipper to a dipper pattern when they were put on a sodium restricted diet.

Given the association between sodium-intake and reduced nocturnal blood pressure dipping, and the link between non-dipping blood pressure and cardiovascular complications (discussed previously), it is important to examine associations between these factors in at risk populations, particularly the high risk Black population. Along these lines, Wilson, Sica, and Miller (1999) found that that sodium sensitivity is

associated with non-dipper blood pressure status, in a study with Black normotensive adolescents. Supporting evidence for the link between sodium and non-dipping in Blacks was also found in a recent study by Bankir et al. (2008), where those who had poor sodium excretion during the daytime exhibited blunted blood pressure dipping at night. However, overall, studies investigating associations between sodium-intake and nocturnal blood pressure dipping in Black populations, are few and far in between. This study contributes to the research area by looking at nocturnal blood pressure dipping under regular and sodium-loaded diet conditions, in a Canadian Black population.

Anger Expression, Ambulatory Blood Pressure, and Blood Pressure Dipping. As previously discussed, research suggests that inhibition of anger, or anger-in style of coping, has been found to be related to higher blood pressure at rest, elevated cardiovascular reactivity to stressors (Cottington et al., 1985; Gentry et al., 1982; Mills & Dimsdale, 1993, Jorgensen & Kolodziej, 2007; Poole et al., 2006), and/or delayed cardiovascular recovery from stressors (e.g. Lai & Linden, 1992; Anderson et al., 2005) in the laboratory. Suppression of anger is implicated in risk for hypertension as it has been found to be associated with higher all-cause mortality (Julius et al, 1986) and clinical indices of coronary heart disease (e.g. Anderson et al., 2006). Based on the above, it is expected that suppression of anger will also be associated with higher blood pressure in the natural environment as measured by ambulatory blood pressure monitoring in individuals' daily lives. The available literature suggests an especially critical link between anger and non-dipping blood pressure, and associated risk for hypertension and cardiovascular diseases, particularly in the American Black population (Linden, Klassen, & Phillips, 2008; Steffen et al., 2003; Thomas et al., 2004).

Steffen et al. (2003) investigated links between anger inhibition and ambulatory blood pressure in 69 Black American men and women with normal or mildly elevated blood pressure. Anger expression was investigated with the Multidimensional Anger Inventory (MAI) (Siegel, 1986), with particular focus on the anger-in and anger-out subscales. Anger inhibition was found to be unrelated to daytime blood pressure levels but was significantly associated with nocturnal blood pressure and non-dipping blood pressure. Specifically, anger inhibition was found to be related to increased diastolic blood pressure during sleep, as well as a smaller drop in diastolic blood pressure from waking to sleeping hours. Other studies (discussed below) have found somewhat mixed results.

Thomas et al. (2004) investigated the link between anger expression and non-dipping blood pressure, in a 24-hour ambulatory blood pressure study with 34 Black and 52 White American men and women. These researchers found that Blacks compared to Whites, were both more likely to be classified as non-dippers, as well as scored higher on anger expression, as measured by the Spielberger Anger Expression Inventory (Spielberger et al., 1985). According to Spielberger and colleagues, high scores on anger expression reflect the experience of angry feelings which may be dealt with by suppression, expression through aggressive behaviour, or both. Anger expression was found to be associated with less dipping of blood pressure in both Blacks, and Whites. Interestingly, however while Blacks had higher scores on both anger experience and expression, and were more likely to deal with anger by directing it inwards (suppression of anger), anger-in measures was not found to be related to non-dipping in this study. Notwithstanding, this study by Thomas and colleagues demonstrate that anger

expression is indicated as an important factor in the link between anger and non-dipping blood pressure, and risk for hypertension in the Black population.

Linden et al. (2008), sought to investigate links between anger/hostility, chronic stress, and non-dipping blood pressure in a sample of 62 drug-free hypertensive patients. Anger coping preferences were measured with the Behavioral Anger Response Questionnaire (BARQ) (Linden et al., 2003). It was found that the extent of systolic blood pressure dipping was positively associated with high anger diffusion scores. Given that anger diffusion is conceptualized as a proactive way of coping with angry feelings, Linden and colleagues (2008) proposed that perhaps nondippers have a hard time letting go of angry feelings, which may have a negative impact on blood pressure dipping. An interesting, though non-significant finding was that the effect sizes associated with 'perceived stress' and the 'avoidant coping' subscale of the BARQ were in the low to moderate range, which the authors suggest may have reached significance with a larger sample. It is noteworthy, that coping with anger by avoidance of angry feelings is potentially similar to an anger-in coping style, which has been found to be detrimental to blood pressure recovery from stress in laboratory research, as shown in the previously discussed study by Dorr et al. (2007).

Further research is needed to elucidate clearer associations between anger expression and ambulatory blood pressure/blood pressure dipping, particularly with respect to effects of anger inhibition coping styles, in efforts to shed light on the disproportionate rate of hypertension in the North American Black population. The current study contributes to the scarce data in this research area by investigating the

association between anger-in and ambulatory sleep and wake blood pressure, and blood pressure dipping at night, in a Canadian Black population.

Perceived Racism, Ambulatory Blood Pressure, and Blood Pressure Dipping. As previously discussed, perceived racism has been proposed as a chronic daily psychosocial stressor in the lives of Western Blacks, and a potential risk factor for the development of hypertension in this population (Clark et al., 1999). Along these lines, perceived racism has been found to be associated with elevated cardiovascular reactivity to stressors in laboratory studies (e.g. Clark et al., 1999; Fang & Myers, 2001). It follows that perceived racism would also be associated with individuals' blood pressure responses in their natural social environments during daily life, as measured by 24-hr ambulatory blood pressure monitoring. Indeed, studies have found that greater perceived racism was related to greater ambulatory blood pressure during the wake period, in an adult Black American sample (Steffen et al., 2003), and to greater sleep ambulatory measures, in an adult American Black and Hispanic sample (Brondolo et al., 2008). Additionally, a study by Hill, Kobayashi, and Hughes (2007) showed that greater perceived racism was associated with both higher sleep and wake blood pressure in a study with Black American college students. Oddly, no association was found between perceived racism and non-dipping blood pressure in the study by Steffen and colleagues. Alternatively, the study by Brondolo and colleagues found that perceived racism was associated with increased risk of being categorized as a non-dipper, which is in line with research suggesting an important link between chronic stress and non-dipping blood pressure, and risk for development of hypertension (e.g. Fallo et al., 2002).

Research on the association between perceived racism and ambulatory blood pressure, particularly non-dipping blood pressure, is sparse. Further investigations are clearly warranted in order to shed light on potential important relationships between these variables and the high prevalence of hypertension in the Black population. Although not specific to perceived racism, a related study by Tomfohr, Cooper, Mills, Nelesen and Dimsdale (2010) looked at the effect of 'everyday discrimination' on nocturnal blood pressure dipping in Black and White Americans, and consistent with the proposal of a link between chronic stress and non-dipping blood pressure, found that discrimination was associated with less diastolic and systolic blood pressure dipping. An important difference between the above mentioned study and the present study is that the former allowed for inclusion of multiple types of discrimination while the latter looked at a specific type of discrimination – racism (perceived). None-the-less, the study by Tomfohr and colleagues offers support for this study's hypothesis for an association between high perceived racism and decreased nocturnal blood pressure dipping in the Black population. The present study contributes uniquely to the research literature by being the first to investigate the association between perceived racism, and ambulatory sleep and wake blood pressure and blood pressure dipping, in a Black Canadian population.

Summary, Rationale, and Hypotheses

Research shows a disproportionately high prevalence rate of hypertension and related cardiovascular diseases in Black populations in North America. As such, there is an identified need for a focus in research investigating potential high risk factors within the Black population. Many risk factors contributing to development of essential hypertension have been proposed to date. These include dietary sodium, gender

differences in cardiovascular responses, exaggerated cardiovascular reactivity to stress, delayed cardiovascular recovery from stress, and diurnal variations in ambulatory blood pressure, including sleep, wake, and non-dipping blood pressure. In addition, psychological factors particularly anger/anger expression, and effects on cardiovascular responses, have been prominently linked to the development of hypertension and cardiovascular diseases. Also, of specific importance to Blacks living in a predominantly White North American society, the proposed chronic psychosocial stressor, perceived racism, and effects on cardiovascular responses, have been linked to the high risk for essential hypertension in the Black population. The present study investigates the above mentioned risk factors within the Black Canadian population, using a joint protocol involving measurement of cardiovascular responses under controlled laboratory conditions, as well as in the naturalistic environment via ambulatory blood pressure monitoring.

In the laboratory protocol, male and female participants were exposed to three stressor tasks (a mental, a physical, and a social stressor), in two dietary conditions (regular diet compared to a sodium-loaded diet). It has been shown that Black individuals respond to various stressors with increased cardiovascular reactivity and decreased cardiovascular recovery, which might contribute to the higher risk for development of hypertension and cardiovascular diseases found in this population. Research also shows that males tend to react with greater blood pressure/vascular responses compared to females, who tend to respond with greater heart rate/myocardial responses when exposed to stress. Additionally, males have been found to have higher cardiovascular activation/decreased cardiovascular recovery during the recovery period following

exposure to stress, compared to females. These gender differences are investigated in the present research study. Research also implicates higher levels of dietary sodium in facilitating exaggerated cardiovascular responses to stressors, which are linked to the development of hypertension and cardiovascular diseases. As such, it was expected that cardiovascular reactivity to and recovery from various stressors would be different under conditions involving different levels of sodium consumption. Furthermore, psychological and psychosocial factors such as anger-in and perceived racism, have also been associated with increased cardiovascular reactivity to and decreased recovery from, laboratory stressors. These associations are investigated in the present study.

In the ambulatory blood pressure protocol, blood pressure measures were collected over a 24-hr period, in each of the two diet conditions (regular versus sodium-loaded diet), to allow for evaluation of blood pressure during day/wake and night/sleep periods, and percentage blood pressure dipped at night, as influenced by sodium. This follows from research showing a positive relationship between dietary sodium intake and blood pressure levels, research implicating non-dipping blood pressure as an important risk factor in the development of hypertension and cardiovascular disease, and findings that high sodium diet is associated with greater non-dipping blood pressure. As such, it was expected that ambulatory wake and sleep blood pressure, and percentage blood pressure dipped at night, would differ under different diet conditions involving different levels of sodium consumption. Psychosocial factors including anger-in and perceived racism have also been associated with ambulatory blood pressure and non-dipping nocturnal blood pressure in the Black population; these associations are also investigated in the present study. Additionally the presents study looks at the effect of gender on

ambulatory blood pressure measures, following established findings of male/female differences in resting and ambulatory blood pressure and heart rate.

Research investigating the above discussed risk factors for hypertension in the Black population has been conducted predominantly in the United States. Limited research exists on investigation of hypertension risk factors within the Black Canadian population. Moreover, no study has examined the effects of perceived racism on risk for hypertension among Canadian Blacks. Given different socio-cultural contexts of American and Canadian societies, it is conceivable that the effects of perceived racism on American Blacks may not be wholly generalizable to Canadian Blacks. Specifically, it is possible that the effects of perceived racism may differ on the extent to which it affects psychological and physiological variables linked to development of essential hypertension within the Canadian Black population. The present research study was developed to add to and address gaps in current literature, for factors that potentially contribute to the high risk for essential hypertension among Canadian Blacks, including perceived racism, anger expression, dietary sodium, gender, cardiovascular reactivity, cardiovascular recovery, ambulatory sleep and wake blood pressure levels, and nocturnal non-dipping blood pressure.

The following hypotheses were proposed for the present study with a Canadian Black population. (1) Individuals will have greater cardiovascular reactivity in response to laboratory stressor tasks in the sodium-loaded diet condition compared to the regular diet condition. (2) Individuals will have decreased cardiovascular recovery after exposure to laboratory stressors in the sodium-loaded diet condition compared to the regular diet condition. (3) Males will have greater blood pressure/vascular reactivity compared to

females, in response to laboratory stressors. (4) Females would have greater heart rate/myocardial reactivity compared to males, in response to laboratory stressors. (5) Males will have decreased cardiovascular recovery compared to females, after exposure to laboratory stressors. (6) Individuals with high compared to low perceived racism scores will have greater cardiovascular reactivity in response to laboratory stressors. (7) Individuals with high compared to low perceived racism scores will have decreased cardiovascular recovery after exposure to laboratory stressors. (8) Individuals with high compared to low anger-in scores will have greater cardiovascular reactivity in response to laboratory stressors. (9) Individuals with high compared to low anger-in scores will have decreased cardiovascular recovery after exposure to laboratory stressors. (10) Males will have higher ambulatory blood pressure measures compared to females during wake and sleep periods, while females will have higher heart rate measures than males during these periods. (11) Individuals will have higher ambulatory wake and sleep blood pressure measures, and less nocturnal blood pressure dipping, in the sodium-loaded diet compared to the regular diet condition. (12) Individuals with high perceived racism scores compared to those with low perceived racism scores will have higher ambulatory wake and sleep blood pressure measures, and less nocturnal blood pressure dipping. (13) Individuals with high anger-in scores compared to those with low anger-in scores will have higher ambulatory wake and sleep blood pressure measures, and less nocturnal blood pressure dipping.

Method

Participants

Fifty-three Black individuals (20 males; 33 females) between the ages of 18 and 30 years participated in the study. Participants were recruited from the Concordia student population. All individuals completed a screening Health Questionnaire (see Appendix A) developed in our lab and used in similar studies in the past. Those who reported any physical (see health questionnaire) or psychological health problems and/or regularly used medication that affected blood pressure were excluded from the study. Individuals who were accepted to participate in the study had to have been born in Canada or had lived in Canada at least since the age of 12 years. This is necessary to control, to some degree, for the social climate experienced by Black individuals in Canada. The social climate of minority Black individuals living in a predominantly White society is a factor, which in combination with other factors, is thought to influence the development of hypertension (Clark et al., 1999). Participants who met the above criteria were invited for an in-person interview and information session providing details of the study. After full disclosure of the study details, individuals who were interested in participating were asked to read and sign a detailed Informed Consent form (see Appendix B), which was approved by the Human Subjects Ethics Committee of Concordia University. Each individual was paid $100 for participating upon the completion of the 21-day testing protocol.

Medical screening. After giving written informed consent and prior to inclusion in the study, individuals were required to undergo a 30-minute general medical exam by an appointed physician to further assess physical and mental health. Individuals gave

additional written consent for release of their medical exam information from the physician to the research team (see Appendix C). Individuals were accepted as participants and included in the study upon recommendation of the physician in a completed medical report (see Appendix D). Females who were pregnant at screening were excluded from the study, and in addition, females who participated in the study were required to undergo a urine based pregnancy screen immediately prior to the beginning of the testing protocol. If pregnant at that point, they were excluded from the study.

Physiological Measures and Apparatus

During laboratory procedures, Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) measurements (in mmHg) were obtained at 1-min intervals using an IBS Model SD-700A Automatic Blood Pressure and Pulse Rate Monitor (IBS Corporation, Waltham, Mass, USA). The blood pressure cuff was placed on the participant's non-dominant arm. Additional cardiovascular measures taken include Heart Rate (HR) (in bpm), Stroke Volume (SV) (in ml), Cardiac Output (CO) (in l/min.), and Total Peripheral Resistance (TPR) (in dyne-sec.cm $^{-}$5). SBP is the arterial pressure during the contraction phase and DBP is the arterial pressure during the relaxation phase of the ventricles. HR is the number of heartbeats each minute. SV is the quantity of blood ejected by the heart during a single cardiac cycle. CO is the total volume of blood ejected by the heart during a period of time, typically, as is the case in the present study, a 1 min period. TPR is the resistance to blood flow throughout the entire cardiovasculature.

Values for the measures of HR, SV, CO, and TPR were obtained non-invasively using a Minnesota Impedance Cardiograph (Model 304A, Instrumentation for Medicine,

Greenwich, Conn, USA), an IBM compatible personal computer, EKG spot electrodes, and the Cardiac Output Program (COP Version 2.1, Bio-impedance Technology, Chapel Hill, North Carolina, USA). The impedance cardiography utilized a tetrapolar electrode-band configuration. The inner two recording electrode-bands were placed around the base of the participant's neck and around the thorax over the tip of the xiphoid process. The outer two electrode-bands were placed around the neck and the thorax at least 3 cm apart from each of the inner electrode bands. The ECG signal was recorded independently using three spot electrodes. Two electrodes were placed on either side of the torso below the ribcage and a ground electrode was positioned on the right hipbone. The ECG signal was filtered through a Coulbourn Instruments bandpass filter (Coulbourn Instruments, Allentown, Penn, USA) and then routed to the Minnesota Impedance Cardiograph. Within every measurement minute, 55 s of recordings were obtained and processed by the COP system, yielding ensemble averaged values for HR, SV, CO, and TPR.

24-hr ambulatory blood pressure monitoring was accomplished by using a programmed portable Suntech Accutraker II ambulatory blood pressure monitor. The monitor was programmed to obtain 4 blood pressure measures per hour (on a variable schedule, but no less than 15 minutes apart) between the hours of 7 AM to 11 PM, and 2 measures per hour between the hours of 11 PM to 7 AM so as to minimize sleep disturbance. Participants were instructed to drop their arms to their side as soon as they felt the cuff inflating, and to keep the arm relaxed until a few seconds after the cuff had deflated. The monitor was programmed to make one retry if a reading yielded

unacceptable values, inflated to a level not high enough to obtain a systolic reading, or if a reading occurred during arm movement.

Psychological Measures

State Affect Questionnaire. A state affect questionnaire, the Visual Analog Mood Scale (VAMS) (See Appendix E) was employed to determine the participant's current state affect at six times (pre-task and post-task for each of the three tasks) during the cardiovascular reactivity testing session. This measure consists of 13 affective terms including, nervous, agreeable, happy, tense, anxious, relaxed, discouraged, irritated, annoyed, sad, angry, depressed, and guilty. Subjects rated their current affective state on the visual analogue scale by marking a vertical stroke at a point along a 12-cm line with endpoints labeled 'not at all' and 'very'. Points on the line were measured and given numerical values for data analysis.

Perceived Racism Scale. The Perceived Racism Scale (See Appendix F) was employed to assess participants' perceived racial experiences. This questionnaire has face validity and reliably distinguishes amongst subgroups of African-Americans that vary in their experience of racial discrimination (McNeilly et. al., 1996). It assesses how often individuals are exposed, in the past year and during their lifetime, to racially discriminatory experiences in (i) the workplace (ii) academic settings (iii) public life, and (iv) to racist statements. Scores in the above domains were calculated for each participant, for the past year and for their lifetime. An overall perceived racism score was computed, totaling scores from all domains. Participants were also asked to rate how angry, frustrated, sad, powerless, hopeless, ashamed, and strengthened they felt in

response to discrimination. Averages for each of the above affective states were calculated.

Spielberger Anger Expression Scale. The Spielberger Anger Expression Scale (Spielberger et al., 1985) (see Appendix G) was completed by all participants, to assess anger expression. This is a 41-item Likert scale which yields a total score as well as subscale scores for Anger-in and Anger-out, two modes of anger expression. Anger-in is defined as the tendency to suppress anger whereas Anger-out refers to the tendency to direct anger outwards to others. Test-retest reliability for this measure ranges from .58 to .75 over 8-10 week intervals (Spielberger et al.) Cronbach alphas have ranged from .70 to .84 for total and subscale scores (Johnson, Spielberger, Worden, & Jacobs, 1987). Good convergent and divergent validities have also been indicated (Spielberger et al.).

Laboratory Stressor Tasks

Computerized Math Subtraction Task. The mathematical subtraction task (Math task) consisted of the Computerized Subtraction Task Version 1.21 computer program (Turner, Sherwood, & Lutz, 1989), an IBM PC computer, and a computer mouse. The Math task was 3 min long, and consisted of a series of mathematical subtraction equations presented with either correct or incorrect solutions. During the task, 60 equations were sequentially presented on the monitor. Each equation was presented for a duration of 3 s, first appearing as white characters against a black background and switching to yellow characters if the participant did not respond within the first 2 s. Participants used the right or left computer mouse buttons to indicate whether the solution on the screen was correct or incorrect, respectively. Auditory feedback, either a high- or low-pitched tone, informed the participant whether he/she had answered

correctly or incorrectly, respectively. No tone was emitted if the participant failed to respond within the allotted time and that trial was scored as incorrect. The Math task was designed so that each participant attained a 50 to 60 percent correct response rate. That is, the equations become easier or more difficult depending on each participant's performance. See Appendix H for Math task instructions.

Cold Pressor Task. The Cold Pressor stimulus consisted of a cold gel pack (temperature approximately 4 ºC), which was positioned across the forehead of the participant while the participant was reclining, with his/her head comfortably supported against the back of the padded armchair. The cold pack was held to the participant's forehead for 2 min, taken off for 1 min, and then followed by the application of a similar cold pack of the same temperature for another 2 min. See Appendix I for Cold Pressor task instructions.

Discrimination Recall Task. The participant was asked to recall and talk about an incident in his/her past when he/she felt discriminated against. The participant was asked to re-experience, as much as possible, the situation and his/her feelings at the time. The task lasted for 3 min. The experimenter used prompts to elicit feelings and details of the event for the duration of the 3 min, as needed. Prompts used included, "How did that make you feel?"; "What did you say?"; "What did you do?" See Appendix J for Discrimination Recall task instructions.

Dietary Manipulation

Dietary Sodium. Participants were given a 10-day supply of dietary sodium materials to be consumed with their regular diet. Identical daily portions were packaged separately in sealed zip-locked plastic bags. Each zip-locked bag contained a packet of

Lipton Noodle Soup (84 g) with NaCl content of 10.6 g, and a smaller sealed plastic bag with 4.4 g of Nacl (table sodium). This dietary manipulation amounted to daily sodium-loading of 15 g Nacl in addition to regular sodium intake. Participants were given a detailed instruction sheet for this sodium-loaded diet period (see Appendix K).

Dietary Compliance Verification

Urine Collection. Participants were given a 3-L brown translucent urine collection jar for overnight urine collection, and two 12-ml labeled test-tubes (one for each diet period) to collect and return urine samples to the lab for analysis of dietary compliance. Participants were also given detailed written instructions on the urine collection procedure (see Appendix L). Urine samples were analyzed for sodium content at the Central Laboratory of the Montreal General Hospital.

Setting

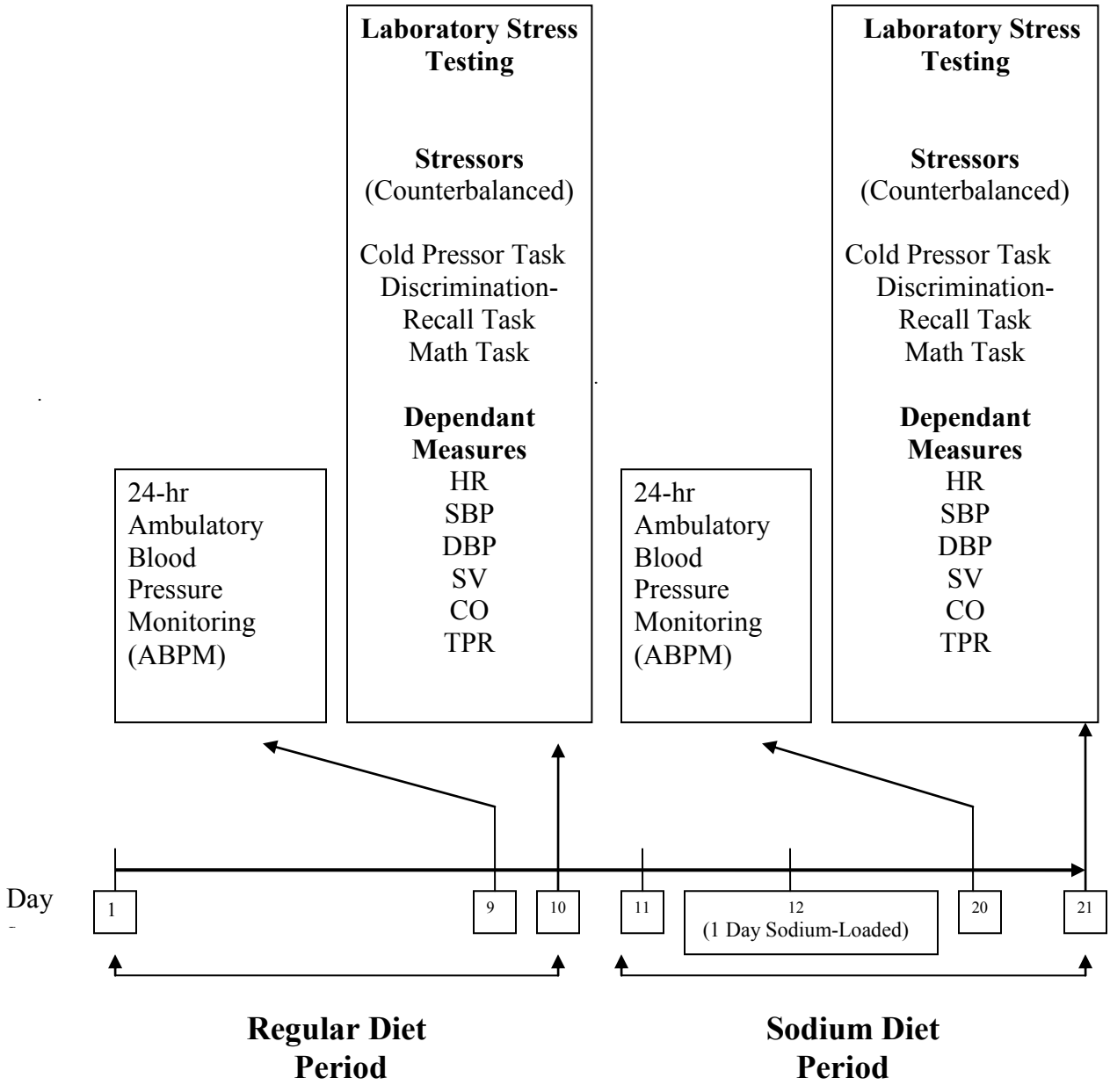
The lab testing procedure was carried out in a quiet room, with a comfortable, reclining armchair. Two-way mirrors and the use of a "walkie-talkie" baby monitor (900 MHz Grow-With-Me-Monitor) enabled communication and synchronization of the physiological recording of cardiovascular measures with the laboratory stress procedure, which was carried out in adjacent rooms by two experimenters, one in each room. Experimenter A, a female, who interacted with the participants, remained constant throughout the 21-day protocol. Experimenter B, who monitored the physiological equipment (the COP system) in the adjacent room, varied, and had no interaction with the participants.

Experimental Procedure

Overview. The experiment was a within-subject design. Each individual participated in a 21-day experimental protocol, which consisted of two counterbalanced periods: a 10-day regular diet period and an 11-day sodium-loaded diet period (given the initial 24-hr period needed to become sodium-loaded, participants are 10 days sodium-loaded on the 11-day of the sodium-loaded period). Participants who were approved by the physician presented themselves to the laboratory where instructions were given for the first diet period, and four appointments to complete the protocol were scheduled on specific days within the 21-day experimental period: Appointments were scheduled on the ninth and tenth days of the regular diet period, and on the tenth and eleventh days of the sodium-loaded diet period. Participants were asked to refrain from consuming caffeine, cigarettes, alcohol, or recreational drugs for approximately 14 hours prior to laboratory testing sessions. They were further given a package of questionnaires, which they were required to complete at home and return by the end of the 21-day testing protocol. A diagram of the study protocol is presented in Figure 1. This study employed no deception.

Details of the Procedure. Participants adhered to a diet that did not deviate from their usual lifestyle pattern for the 10 days of the regular diet period. During the sodium-loaded diet period, participants were instructed to consume the sodium-loaded food provided as a supplement to their regular diet. The dietary sodium materials were given to the participants just prior to beginning the sodium-loaded phase of the protocol, and female participants were required to undergo a urine-based pregnancy screen in the laboratory at that time. The participant was allowed to proceed with the protocol if the

Experimental Design



Dependent Measures: Heart Rate (HR); Systolic Blood Pressure (SBP); Diastolic Blood Pressure (DBP); Stroke Volume (SV); Cardiac Output (CO); Total Peripheral Resistance (TPR)

Figure 1. Details of the 21-day research study protocol.

pregnancy screen was negative. On the ninth day of the regular diet phase and on the tenth day of the sodium-loaded diet phase, participants came to the lab for approximately 30 min, between 7 and 9 a.m. Individuals were seated in an armchair and resting blood pressure levels were taken prior to being instrumented with a programmed ambulatory blood pressure monitor to be worn for 24 hr, after which the monitor would be removed by the experimenter at the scheduled visit the next morning. Participants were given a plastic bag with the urine collection materials and verbal and written instructions for collecting urine for a 12-hr period that night (see Appendix L). They were also given an activity diary to fill out during waking hours for the next 24-hr period.

Participants returned to the lab on the tenth day of the regular diet period and on the eleventh day of the sodium-loaded period for a testing session, which lasted approximately 90 min. The urine samples, any completed questionnaires, the activity diary, and the urine instruction sheet with the completed data collection information, were collected, and the ambulatory blood pressure monitor was removed from the participant's arm. The urine sample was placed in the freezer. The principal experimenter (Experimenter A) reminded the participant of the procedure for that day and proceeded to instrument the participant with the physiological equipment for cardiovascular measurement recording. The participant was then seated in an armchair, which was reclined to an approximately, 60°- angled position. Experimenter A instructed the participant regarding the order of administration of the stressor tasks (counterbalanced across participants), explained the instructions for each task, and showed the participant a sample of the State Affect Questionnaire which was to be filled out before and after each task. Calibration of the COP system, the computerized program used in recording

physiological measurements from participants, was carried out simultaneously by Experimenter B in the adjacent room. The two experimenters communicated via a two way walkie-talkie monitor in executing the stress testing protocol with simultaneous recordings on the COP system. Upon verbal cue from Experimenter B that the procedure was to begin, Experimenter A dimmed the lights and left the testing room, instructing the participant to relax, but not to fall asleep.

The participant rested for 10-min, then cardiovascular measures were taken for 4 min for the pre-task baseline period. Experimenter A then re-entered the testing room, turned the lights up, and handed the participant the State Affect Questionnaire to complete (approximately 1 min), then administered the first stressor task. Experimenter B communicated by monitor to Experimenter A when to begin and end the task. A post-task State Affect Questionnaire was completed by the participant immediately upon task completion, and then Experimenter A left the room, dimming the lights again. Cardiovascular measures were recorded throughout this period. Cardiovascular measures continued to be taken for a 4-min recovery period. The participant then rested for 5 minutes. Following this, an identical procedure was completed for the second and third tasks.

At the end of the testing procedure, the physiological recording apparatus was removed from the participant. The participant was also asked if he/she had any questions. The participant was given instructions and/or dietary materials for the next diet period, if applicable. If it was the end of the 21-day protocol, participants were paid $100 for their participation. This study was approved by the Human Subjects Ethics Committee of Concordia University.

Data Reduction and Analyses

Laboratory Cardiovascular Measures. Cardiovascular data during the laboratory testing sessions were reduced in the following manner: for each cardiovascular measures (SBP, DBP, HR, SV, CO, and TPR), values collected during the last 4 min of baseline testing (rest period) were averaged to obtain a mean baseline value. All values collected during the Math task were averaged across the 3 min of the task yielding a mean math task value. A mean Discrimination Recall task value was obtained in the same way. A mean Cold Pressor task value was obtained from the 4 min of active cold pressure application (measures from the 1-min break in the middle of this task were not included). All values collected for a 4 min period immediately following each of the above laboratory tasks were averaged yielding a mean recovery value for each respective task. Change scores were used in all cardiovascular analyses given the uncertainty regarding validity of impedance-derived volume measures when absolute values are employed (Sherwood et al., 1990). For Cardiovascular Reactivity, change scores were obtained by subtracting from the task mean, the mean of the immediately preceding baseline period for each laboratory task. For Cardiovascular Recovery, change scores were obtained by subtracting from the recovery mean, the baseline mean of the preceding stressor task. Analyses of cardiovascular responses to stressor tasks were carried out on task minus baseline change scores (reactivity measures) and recovery minus baseline change scores (recovery measures). Change scores for cardiovascular measures were calculated in both the regular diet and the sodium-loaded diet periods.

Ambulatory Blood Pressure Measures. Measures obtained during 24-hr ambulatory blood pressure monitoring were used to calculate averaged values of MAP,

SBP, DBP and HR for the total monitoring period, as well as averaged values for during the night (sleep period) and day (wake period) respectively. Sleep and wake periods were defined by each individual's report of the time he/she went to bed at night and the time that he/she awoke the next morning. Measures of MAP for sleep and wake periods were used to calculate the percentage blood pressure dipped during sleep. Averaged measures for MAP, SBP, DBP and HR, and percentage of blood pressure dipped, were calculated in both the regular diet and the sodium-loaded diet periods. Analyses were conducted on ambulatory means of MAP, SBP, DBP, and HR for the total ambulatory period, the sleep period, and the wake period. Analyses were also conducted on percentage blood pressure dipped at night.

Questionnaire Measures. For the State Affect Questionnaire, values collected prior to each stressor task for each of the following affective measures (nervous, tense, anxious, annoyed, sad, irritated, angry, and depressed), were considered baseline-affect values. Baseline minus stress affect change scores values were calculated by subtracting baseline-affect values from the affect values obtained immediately following the stressor task. Analyses were carried out on these change scores. For the Perceived Racism Questionnaire, analyses were carried out on median-split values for Total Perceived Racism for the past year (i.e. high perceived racism versus low perceived racism). For the Anger Expression Questionnaire, analyses were similarly carried out on median-split values for measures of Anger-in and Anger-out (i.e. high anger-in versus low anger-in, and high anger-out versus low anger-out).

The above laboratory, ambulatory, and questionnaire data were analyzed using the following procedures: Repeated Measures MANCOVAs were first performed, given the

presence of multiple dependent measures, and the within-subject variables of Diet and Stressor Tasks. Gender/Sex and median-split questionnaire measures - Perceived Racism and Anger Expression - were entered as between-subject variables in the MANCOVAs. Covariates included Body Mass Index (BMI) and Family History of Hypertension, which were entered to control for possible association with cardiovascular measures. Significant multivariate results of the MANCOVAs were followed by univariate ANCOVAs, and investigation of post-hoc contrasts of interest utilizing Fisher's LSD test.

Results

Dietary Compliance

Dietary compliance was assessed by comparing sodium levels in the regular diet condition versus the sodium-loaded diet condition. The criterion for assessing compliance required that participants' sodium level in the high-sodium diet condition be higher than their sodium level in the regular diet condition; no set criteria for amount of difference in sodium level was used, given a within-subject design where each participant's sodium level is compared against their own across conditions, and given inter-individual differences in sodium metabolism. Participants' sodium levels in each diet condition are presented in Table 1. It was found that 9 of the 53 participants who completed the study were not compliant with the dietary protocol. To determine whether urine sodium content was significantly higher in the sodium-loaded condition versus the regular diet condition for the 44 diet compliant participants, paired *t* tests were conducted with measures of the sodium content of urine for each dietary condition. A significant effect was found for dietary compliance, $t(44) = 9.16$, $p < .01$.

Participant Characteristics

Participants in this study included 20 males and 33 females. Descriptive statistics of participants' demographic characteristics of age, height, weight and body mass index (BMI) are presented in Table 2.

Analysis Plan

Testing of each of the 13 hypotheses was conducted in the following manner. First, MANCOVA analyses, with covariates, body mass index, and family history, were conducted. These were followed by relevant ANCOVA analyses, including the same

Table 1

Participants' Urine Sodium Content in the Regular Diet versus the Sodium-loaded Diet

	Sodium Content (mmol/L)	
Participant #	Regular Diet	Sodium Diet
101	138	199
102	16	222
*103	*177	*52
105	94	197
107	146	242
108	63	100
109	42	126
110	161	196
112	56	118
113	182	260
*114	*181	*74
115	107	164
116	144	243
117	155	190
118	215	310
*119	*172	*160
120	102	284
121	132	201
122	173	207
123	32	117
*124	*119	*111
125	41	134
126	187	210
127	168	284
*128	*218	*173
*129	*117	*63
130	63	125

Note. * represents data for participants who were not compliant with the sodium diet.

Table 1 (continued)

Participants' Urine Sodium Content in the Regular Diet versus the Sodium-loaded Diet

	Sodium Content (mmol/L)	
Participant #	Regular Diet	Sodium Diet
131	43	72
132	140	244
133	87	312
134	143	244
135	137	298
136	83	93
137	178	291
138	79	88
139	122	321
140	231	235
141	300	357
142	130	224
*143	*116	*110
144	144	145
145	52	181
146	94	126
*147	*264	*230
148	63	152
*149	*148	*104
150	137	245
151	179	273
152	24	35
153	12	67
154	137	183
155	134	143
156	184	200

Note. * represents data for participants who were not compliant with the sodium diet.

Table 2

Demographic Characteristics of Participants

	Minimum	Maximum	Mean	SD
Height (ins)				
Male	67	74	70.7	2.2
Female	60	70	65.6	2.6
Weight (lbs)				
Male	134	240	177.5	25.0
Female	110	225	138.3	22.3
BMI				
Male	20.4	32.5	24.9	2.9
Female	16.9	35.2	22.6	3.4
Age (yrs)				
Male	18	30	23.3	3.9
Female	18	28	22.1	2.4

covariates, and with the syntax modified to look only at effects found in the MANCOVA analyses. For hypotheses including diet, namely hypotheses 1, 2 and 11, analyses were conducted only on data for participants who had been compliant with the sodium diet; hence a reduced sample excluding dietary noncompliant participants was used. For all other hypotheses, the full sample of participants was used in analyses, which were conducted on measures obtained during the regular diet condition.

Laboratory Protocol Analyses. *Hypotheses 1 and 2*, which proposed differences in cardiovascular reactivity and cardiovascular recovery, respectively, based on diet condition (sodium-loaded versus regular), were tested with 2(diet conditions) x 2(sex) x 3(stressor tasks) repeated measures MANCOVAs, followed by 2 x 2 x 3 repeated measures ANCOVAs. *Hypotheses 3 and 4,* which proposed differences between genders for pattern of cardiovascular reactivity (vascular versus myocardial), were tested with 2(sex) x 3(stressor tasks) repeated measures MANCOVAs, followed by 2 x 3 repeated measures ANCOVAs. *Hypothesis 5,* which proposed differences between genders on cardiovascular recovery was tested with a 2(sex) x 3(stressor tasks) repeated measures MANCOVA, followed by 2 x 3 repeated measures ANCOVAs. *Hypotheses 6 and 7,* which proposed differences on cardiovascular reactivity and recovery, respectively, based on perceived racism scores, were tested with 2(sex) x 3(stressor tasks) x 2(perceived racism) repeated measures MANCOVAs, followed by 2 x 3 x 2 repeated measures ANCOVAs. *Hypotheses 8 and 9* which proposed differences in cardiovascular reactivity and recovery, respectively, based on anger-in scores, were tested with a 2(sex) x 3(stressor tasks) x 2(anger-in) repeated measures MANCOVA, followed by 2 x 3 x 2 repeated measures ANCOVAs.

Ambulatory Protocol Analyses. *Hypothesis 10*, which proposed differences in wake and sleep blood pressure, based on gender, was tested with a one-way MANCOVA followed by one-way ANCOVAs. *Hypothesis 11*, which proposed differences in wake and sleep blood pressure, and percentage blood pressure dipped at night, based on diet condition (sodium-loaded versus regular), was tested with 2(diet conditions) x 2(sex) repeated measures MANCOVAs, followed by 2 x 2 repeated measures ANCOVAs. *Hypothesis 12,* which proposed differences in wake and sleep blood pressure, and percentage blood pressure dipped at night, based on perceived racism scores, was tested with a 2(sex) x 2 (perceived racism) MANCOVA, followed by 2 x 2 ANCOVAs. *Hypothesis 13*, which proposed differences in wake and sleep blood pressure, and percentage blood pressure dipped at night, based on anger-in scores, was tested with a 2(sex) x 2 (anger-in) MANCOVA, followed by 2 x 2 ANCOVAs.

Additional Analyses. Additional analyses were conducted to supplement the study's main analyses described above. These included analyses on baseline and resting cardiovascular measures during the laboratory protocol. Additionally, baseline versus stress analyses, were conducted to demonstrate that the stressor tasks had a stressful effect in the laboratory protocol. All analyses involving anger-in as an independent variable in hypotheses (in both the laboratory and ambulatory protocol) were repeated with anger-out measures, in order to identify potential patterns of interest. Analyses with state affect measures (administered before and after lab stressors) were also conducted to investigate relevant effects during the laboratory stress protocol.

Laboratory Protocol Analyses

Resting and Baseline Analyses. To assess whether participants differed in cardiovascular measures at rest prior to beginning the laboratory stress testing procedure, in the regular versus sodium-loaded diet conditions, and as a function of sex, a 2(diet) x 2(sex) MANCOVA analysis followed by 2 x 2 ANCOVA analyses, were conducted on average resting scores for cardiovascular measures, taken as baseline before the first of the three (counterbalanced) stressor tasks for participants. MANCOVA analysis found a multivariate main effect for Sex, $F(6,34) = 4.9$, $p < .002$, and ANCOVA analyses found significant Sex effects for HR, $F(1,39) = 21.62$, $p < .001$, and SV, $F(1,39) = 6.35$, $p < .017$. Females had significantly higher resting HR than males, and males had significantly higher resting SV than females.

To assess whether participants differed on cardiovascular measures at baseline prior to the various stressor tasks, in the regular versus sodium-loaded diet conditions, and as a function of sex, a 3(stressor task) x 2(diet) x 2(sex) MANCOVA analysis, followed by 3 x 2 x 2 ANCOVA analyses, were conducted on average baseline scores for cardiovascular measures before each of the three stressor tasks. Significant MANCOVA effects were found for Sex, $F(6,33) = 4.6$, $p < .003$, and for Task x Diet x Sex , $F(12,27) = 2.9$, $p < .011$. ANCOVA analyses looking at Sex, found significant effects for HR, $F(1,38) = 17.37$, $p < .001$, and SV, $F(1,38) = 9.84$, $p < .004$. Females had significantly higher resting HR than males, and males had significantly higher resting SV than females. ANCOVA analyses looking at the interaction of Task x Diet x Sex, did not produce any significant findings for cardiovascular measures. Means and standard

deviations for baseline cardiovascular measures before each stressor task, for males and females, in each diet condition, are presented in Table 3.

Baseline versus Stress Analyses. To assess whether there were significant changes from baseline (period 1) to stress (period 2) for each cardiovascular measure for the various stressor tasks, a series of 2(period) x 3(stressor tasks) repeated measures ANOVAs were conducted on average baseline and stress measures, for each cardiovascular measure, in the regular diet condition. The purpose of these analyses was to demonstrate the effectiveness of stressor tasks used in the laboratory protocol. Means and standard deviations for baseline and stress cardiovascular measures, for each stressor task, are presented in Table 4. Significant differences found between baseline and stress cardiovascular measures are discussed below.

For HR, main effects for Task and Period, and a significant interaction between Task and Period, were found; $F(2, 42) = 39.82$, $p < .001$; $F(1, 43) = 87.18$, $p < .001$; and $F(2, 42) = 58.63$, $p < .001$, respectively. Post-hoc analyses revealed significant effects for Period, for the Discrimination Recall and Math tasks, $F(1, 43) = 103.21$, $p < .001$ and $F(1, 43) = 51.17$, $p < .001$, respectively. The findings indicated that participants had significantly higher HR measures in the stress period compared to the baseline period, for both tasks.

For CO, main effects for Task and Period, and a significant interaction between Task and Period, were found; $F(2, 42) = 11.01$, $p < .001$; $F(1, 43) = 31.60$, $p < .001$; and $F(2, 42) = 19.17$, $p < .001$, respectively. Post-hoc analyses revealed significant effects for Period, for the Discrimination Recall and Math tasks, $F(1, 43) = 26.17$, $p < .001$ and $F(1, 43) = 33.18$, $p < .001$, respectively. The findings indicated that participants had

Table 3

Means and Standard Deviations of Baseline Cardiovascular Measures as a Function of Sodium, Stressor Task, and Gender

	Regular Diet						Sodium Diet					
	CP		DR		M		CP		DR		M	
CVR Measures	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
HR *Male*	60.39	9.88	60.30	9.30	58.80	9.67	57.57	7.81	57.63	8.06	57.30	8.00
Female	69.52	8.54	69.91	8.89	69.28	7.98	67.32	7.50	67.44	8.88	67.02	7.75
SV *Male*	129.57	35.48	132.86	37.32	133.29	38.93	142.46	41.09	138.08	38.47	137.81	43.20
Female	105.14	29.06	103.91	26.67	103.12	28.79	108.11	32.15	108.02	31.50	109.44	33.01
CO *Male*	7.89	2.65	8.03	2.60	7.88	2.69	8.16	2.36	7.91	2.28	7.84	2.48
Female	7.25	1.90	7.21	1.77	7.09	1.85	7.26	2.22	7.26	2.20	7.28	2.16
SBP *Male*	115.53	8.58	113.88	10.6	114.22	10.82	114.35	8.75	112.60	10.60	116.22	7.63
Female	109.49	9.81	110.57	9.88	110.04	10.97	110.96	8.79	111.54	8.69	110.69	9.95
DBP *Male*	70.08	8.70	70.16	7.53	69.20	8.10	71.34	9.40	72.09	9.27	71.46	7.62
Female	73.43	8.93	72.67	8.37	75.00	8.28	74.17	7.65	74.74	7.06	75.48	8.84
TPR *Male*	976.99	345.75	969.82	401.50	974.30	374.18	955.82	419.65	959.31	348.21	1008.49	442.33
Female	1008.70	344.02	1021.27	367.80	1058.21	382.39	1084.61	484.26	1092.71	474.40	1008.54	463.20

Table 4

Means and Standard Deviations of Baseline and Stress Cardiovascular Measures as a Function of Stressor Task

		Regular Diet					
		CP		DR		M	
CVR Measures		Mean	*SD*	Mean	*SD*	Mean	*SD*
HR	Baseline	65.85	10.07	66.10	10.08	65.12	9.96
	Stress	65.31	9.43	78.07	12.63	71.13	11.27
SV	Baseline	113.57	32.89	113.31	33.25	113.43	35.08
	Stress	112.62	32.55	108.33	27.57	112.45	32.18
CO	Baseline	7.42	2.20	7.41	2.13	7.31	2.20
	Stress	7.28	2.02	8.45	2.40	7.95	2.34
SBP	Baseline	112.40	10.56	112.55	11.07	112.34	11.92
	Stress	116.36	10.44	120.22	11.70	115.09	10.20
DBP	Baseline	77.60	9.22	72.10	8.34	73.22	8.86
	Stress	77.98	9.51	80.96	8.43	74.41	8.37
TPR	Baseline	1008.67	340.75	1019.56	377.31	1040.74	375.94
	Stress	1103.13	413.79	990.60	384.33	986.29	395.96

significantly higher CO measures in the stress period compared to the baseline period, for both tasks.

For SBP, main effects for Task and Period, and a significant interaction between Task and Period, were found; $F(2, 42) = 3.82$, $p < .031$; $F(1, 43) = 30.15$, $p < .001$; and $F(2, 42) = 6.93$, $p < .003$, respectively. Post-hoc analyses revealed significant effects for Period, for the Cold Pressor, Discrimination Recall, and Math tasks, $F(1, 43) = 10.23$, $p < .004$, $F(1, 43) = 31.54$, $p < .001$, and $F(1, 43) = 7.25$, $p < .011$, respectively. The findings indicated that participants had significantly higher SBP measures in the stress period compared to the baseline period, for all tasks.

For DBP, main effects for Task and Period, and a significant interaction between Task and Period, were found; $F(2, 42) = 8.85$, $p < .002$; $F(1, 43) = 44.37$, $p < .001$; and $F(2, 42) = 21.31$, $p < .001$, respectively. Post-hoc analyses revealed significant effects for Period, for the Cold Pressor and Discrimination Recall tasks, $F(1, 43) = 18.93$, $p < .001$ and $F(1, 43) = 58.43$, $p < .001$, respectively. The findings indicated that participants had significantly higher DBP measures in the stress period compared to the baseline period, for both tasks.

For TPR, a main effect for Task and a significant interaction between Task and Period, were found; $F(2, 42) = 6.08$, $p < .006$; and $F(2, 42) = 15.47$, $p < .001$, respectively. Post-hoc analyses revealed significant effects for Period, for the Cold Pressor and Math tasks, $F(1, 43) = 12.21$, $p < .002$ and $F(1, 43) = 8.24$, $p < .007$, respectively. The findings indicated that participants had significantly higher TPR measures in the stress period compared to the baseline period for the Cold Pressor task,

and significantly lower TPR measures in the stress period compared to the baseline period for the Math task.

In summary, significant changes in cardiovascular measures from baseline to stress were found for all stressor tasks (i.e., the stressor tasks administered had significant stressful effects). HR and CO increased for the Discrimination Recall and Math tasks. SBP increased for all stressor tasks. DBP increased for the Cold Pressor and Discrimination Recall tasks. TPR increased for the Cold Pressor task and decreased for the Math task.

Sodium and Cardiovascular Reactivity. To assess whether participants differed on cardiovascular reactivity across stressor tasks in the regular versus sodium-loaded diet conditions, and as a function of sex, a 2(diet) x 2(sex) x 3(stressor tasks) repeated measures MANCOVA, followed by 2 x 2 x 3 repeated measures ANCOVAs, were conducted on change scores for measures of cardiovascular reactivity. The analyses revealed no significant findings for the effect of diet on cardiovascular reactivity. Means and standard deviations for cardiovascular reactivity measures as a function of diet, as a function sex, and as a function of stressor task, are presented in Table 5.

Sodium and Cardiovascular Recovery. To assess whether participants differed on cardiovascular recovery across stressor tasks in the regular versus sodium-loaded diet conditions, and as a function of sex, a 2(diet) x 2(sex) x 3(stressor tasks) repeated measures MANCOVA, followed by 2 x 2 x 3 repeated measures ANCOVAs, were conducted on change scores for measures of cardiovascular recovery. Significant MANCOVA interactions were found for Diet x Task, $F(12,27) = 2.3$, $p < .032$, and Diet x Task x Sex, $F(12,27) = 2.2$, $p < .042$, and a marginally significant interaction

Table 5

Means and Standard Deviations of Cardiovascular Reactivity Change Scores as a Function of Sodium, Stressor Task, and Gender

	Regular Diet						Sodium Diet					
	CP		DR		M		CP		DR		M	
CVR Measures	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
HR *Male*	-1.33	3.25	11.80	6.54	5.83	4.49	-0.74	2.32	10.28	5.46	4.65	3.98
Female	-0.19	3.27	12.01	8.34	5.74	6.03	-0.28	3.13	11.57	9.04	6.76	7.48
SV *Male*	1.13	9.64	-15.60	11.90	-3.50	11.01	-7.97	14.50	-11.35	19.98	0.14	12.34
Female	-1.57	9.83	0.38	17.27	1.34	8.21	-1.78	8.75	-4.57	10.93	0.79	12.31
CO *Male*	-0.17	0.84	0.50	1.27	0.49	0.68	-0.57	0.93	0.67	1.42	0.78	1.07
Female	-0.10	0.72	1.31	1.30	0.75	0.78	-0.15	0.58	0.86	1.20	0.83	1.09
SBP *Male*	3.34	7.19	7.89	9.95	2.03	6.94	4.14	7.97	7.15	8.85	1.76	4.76
Female	4.70	7.94	8.07	8.39	3.22	6.71	5.15	6.88	8.51	9.80	2.27	6.45
DBP *Male*	5.03	7.03	9.36	8.66	2.88	5.32	4.01	6.72	7.51	4.84	2.98	4.64
Female	5.62	8.83	8.38	7.29	-0.15	4.70	7.27	7.36	9.65	6.56	1.56	6.47
TPR *Male*	56.94	90.94	53.18	219.88	-51.26	101.91	127.67	145.10	3.8	184.84	-78.59	176.52
Female	71.81	130.96	-86.73	178.84	-96.16	115.91	89.75	166.42	-31.71	153.22	-97.10	207.76

was found for Diet x Sex, $F(6,33) = 2.14$, $p < .075$. Subsequent ANCOVA analyses investigating the above mentioned MANCOVA interactions resulted in a significant finding for the Diet x Sex interaction only. The latter interaction had a significant effect on DBP recovery, $F(1,39) = 6.20$, $p < .017$, where males demonstrated significantly less DBP recovery than females on the regular diet, and females demonstrated significantly less DBP recovery than males on the sodium-loaded diet. Means and standard deviations for cardiovascular recovery measures as a function of diet, as a function of sex, and as a function of stressor task, are presented in Table 6.

Gender and Cardiovascular Reactivity. To assess whether males and females differed in cardiovascular reactivity across laboratory stressor tasks, a 2(sex) x 3(stressor tasks) repeated measures MANCOVA, followed by 2 x 3 repeated measures ANCOVAs, were conducted on change scores for measures of cardiovascular reactivity in the regular diet condition. MANCOVA analysis found a multivariate main effect for Sex, $F(6,43) = 2.5$, $p < .039$, and an interaction for Task x Sex $F(12,33) = 2.2$, $p < .034$. ANCOVA analyses looking at Sex, found significant effects for SV, $F(1,48) = 9.4$, $p < .004$; CO, $F(1,48) = 7.4$, $p < .010$, and TPR, $F(1,48) = 5.0$, $p < .032$. ANCOVA analyses looking at the interaction between Task and Sex found significant effects for SV, $F(2,47) = 4.3$, $p < .020$. Post-hoc analyses found significant Sex differences in SV reactivity on the Discrimination Recall and Math tasks, $F(1,48) = 9.4$, $p < .005$ and $F(1,48) = 5.8$, $p < .020$, respectively, but not on the Cold Pressor task.

The above results revealed that males had substantial decreases in SV measures on the Math and Discrimination Recall tasks, which were significantly different from measures for females, who showed a minimal increase in SV on the former task and a

Table 6

Means and Standard Deviations of Cardiovascular Recovery Change Scores as a Function of Sodium, Stressor Task, and Gender

	Regular Diet						Sodium Diet					
	CP		DR		M		CP		DR		M	
CVR Measures	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*	Mean	*SD*
HR *Male*	0.10	2.66	0.78	3.99	2.72	2.98	0.44	2.01	1.34	2.60	1.53	2.72
Female	0.71	2.69	1.15	3.56	1.16	3.27	0.24	2.86	0.45	3.10	1.23	3.08
SV *Male*	-2.03	4.94	-1.07	14.63	-4.53	8.92	-7.15	9.02	0.03	11.63	-1.68	6.92
Female	-1.46	6.65	-0.51	13.65	-0.28	4.80	-0.38	5.92	0.23	9.69	-1.11	7.61
CO *Male*	-0.16	0.55	0.04	0.87	-0.02	0.48	-0.39	0.66	0.15	0.59	0.12	0.49
Female	-0.08	0.71	0.12	0.91	0.14	0.34	-0.02	0.45	0.07	0.72	0.08	0.63
SBP *Male*	-0.16	5.66	3.38	8.28	-0.03	5.68	2.85	5.54	4.60	6.50	-2.02	6.54
Female	3.44	5.73	3.21	4.65	0.81	5.41	2.44	5.82	3.32	5.61	2.38	6.31
DBP *Male*	1.74	3.94	3.77	4.89	1.93	4.13	2.40	3.86	0.55	3.85	0.15	4.03
Female	1.49	4.31	2.90	2.94	-0.62	3.72	2.95	4.22	2.27	3.33	1.17	3.16
TPR *Male*	24.19	37.66	31.19	148.49	-12.08	94.46	78.73	85.90	-0.02	80.66	-26.06	57.78
Female	31.69	114.36	26.24	148.62	-29.79	75.75	22.22	106.99	16.10	120.29	25.90	120.69

minimal decrease in SV on the latter task. For CO, females showed a significantly greater increase in reactivity compared to males. For TPR, males and females demonstrated opposite directions of reactivity, with males showing an increase and females showing a decrease on this measure. Differences in cardiovascular reactivity as a function of sex, are shown in Figure 2.

Gender and Cardiovascular Recovery. To assess whether males and females differed in cardiovascular recovery across laboratory stressor tasks, a 2(sex) x 3(stressor tasks) repeated measures MANCOVA was conducted on the change scores for measures of cardiovascular recovery in the regular diet condition. No significant gender differences were found for cardiovascular recovery following laboratory stressor tasks.

Anger-in and Cardiovascular Measures. ***At Rest***. To assess whether participants differed on cardiovascular measures at rest prior to beginning the laboratory stress testing procedure, as a function anger-in, and as a function of sex, a 2(anger-in) x 2(sex) MANCOVA was conducted on average resting scores for cardiovascular measures, taken at baseline before the first of the three (counterbalanced) stressor tasks for participants. No effects of anger-in on resting cardiovascular measures were found.

Cardiovascular Reactivity. To assess whether participants differed on cardiovascular reactivity across laboratory stressor tasks, as a function of anger-in, and as a function of sex, a 2(anger-in) x 2(sex) x 3(stressor tasks) repeated measures MANCOVA was conducted on cardiovascular reactivity change score measures. No significant effects for anger-in on cardiovascular reactivity were found.

Cardiovascular Recovery. To assess whether participants differed on cardiovascular recovery across laboratory stressor tasks as a function of anger-in, and as a

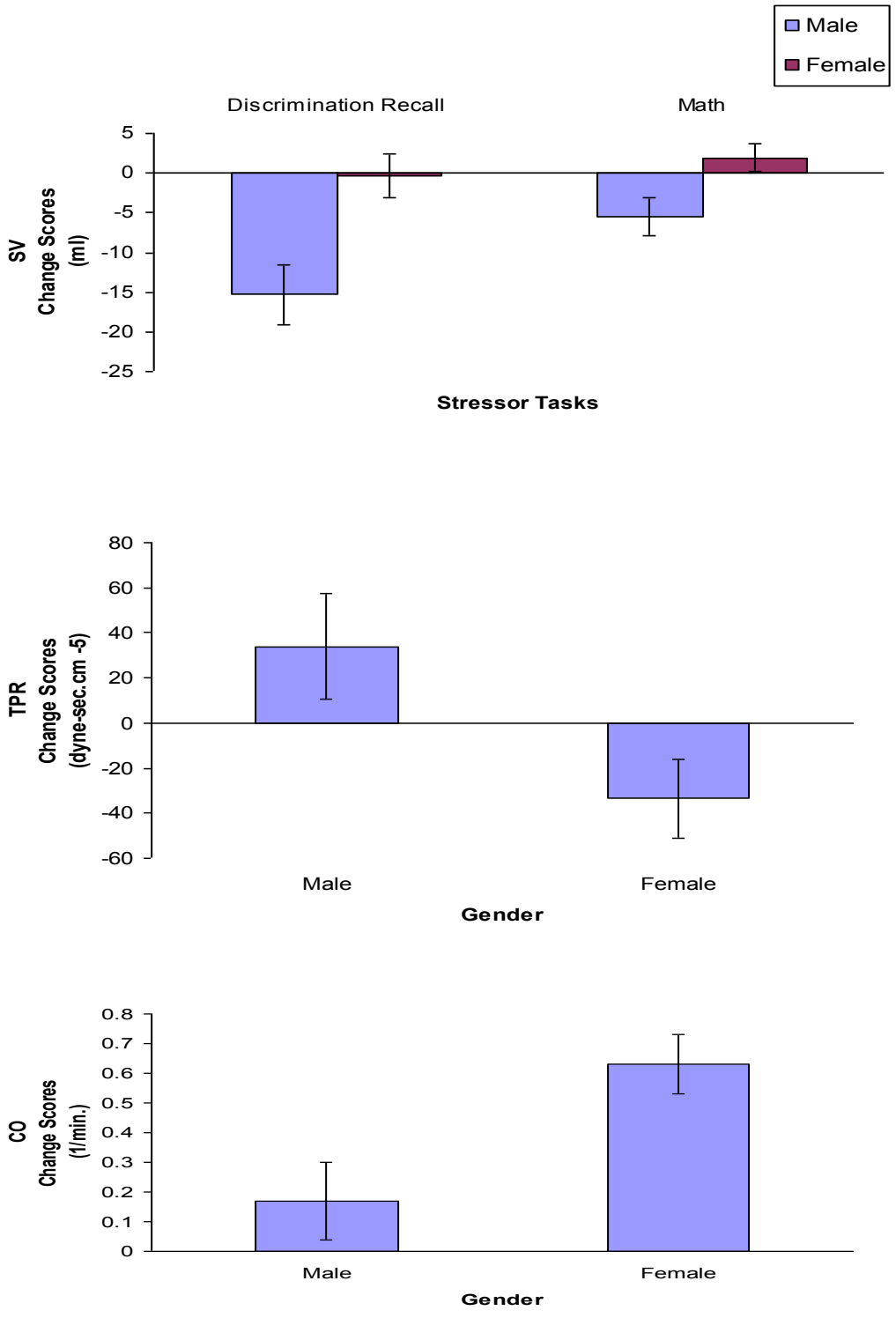


Figure 2. Stroke Volume (SV), Total Peripheral Resistance (TPR), and Cardiac Output (CO) Reactivity change scores as a function of Gender.

function of sex, a 2(anger-in) x 2(sex) x 3(stressor tasks) repeated measures MANCOVA was conducted on cardiovascular recovery change score measures. No significant effects for anger-in on cardiovascular recovery measures were found.

Anger-out and Cardiovascular Measures. MANCOVA analyses, similar to those described above for anger-in, were conducted to test the effects of anger-out on resting cardiovascular measures, cardiovascular reactivity measures, and cardiovascular recovery measures, respectively. No significant effects for anger-out on laboratory cardiovascular measures were found.

Perceived Racism and Cardiovascular Measures. ***At Rest.*** To assess whether participants differed on cardiovascular measures at rest prior to beginning the laboratory stress testing procedure, as a function perceived racism, and as a function of sex, a 2(perceived racism) x 2(sex) MANCOVA was conducted on average resting scores for cardiovascular measures, taken at baseline before the first of the three (counterbalanced) stressor tasks for participants. No significant effects for perceived racism on resting cardiovascular measures were found.

Cardiovascular Reactivity. To assess whether participants differed on cardiovascular reactivity across laboratory stressor tasks, as a function of perceived racism, and as a function of sex, a 2(perceived racism) x 2(sex) x 3(stressor tasks) repeated measures MANCOVA was conducted on cardiovascular reactivity change score measures. No significant effects for perceived racism on reactivity were found.

Cardiovascular Recovery. To assess whether participants differed on cardiovascular recovery across laboratory stressor tasks as a function of perceived racism, and as a function of sex, a 2(perceived racism) x 2(sex) x 3(stressor tasks) repeated

measures MANCOVA was conducted on cardiovascular recovery change score measures. Interactions were found for Task x Perceived Racism, $F(12,33) = 2.4$, $p < .026$; and for Task x Sex x Perceived Racism, $F(12,33) = 2.2$, $p < .041$. ANCOVA analyses looking at Task x Perceived Racism found a significant effect for SBP, $F(2,44) = 3.9$, $p < .029$. ANCOVA analyses looking at Task x Sex x Perceived Racism found significant effects for SV, $F(2,43) = 4.4$, $p < .020$; CO, $F(2,43) = 5.22$, $p < .010$; and DBP, $F(2,44) = 3.31$, $p < .047$.

Post hoc analyses for the Task x Perceived Racism interaction found for SBP recovery revealed that participants had significantly less recovery on the math task in the high perceived racism condition, $F(1,47) = 4.4$, $p < .041$. Post-hoc analyses for the Task x Sex x Perceived Racism found for SV and CO were not significant. Post-hoc analyses for the Task x Sex x Perceived Racism found for DBP, revealed that males had significantly less recovery on the Math task in the high perceived racism condition, $F(1,23) = 5.07$, $p < .035$. SBP recovery as a function of perceived racism, and DBP recovery as a function of sex and perceived racism, for the math stressor task, is shown in Figure 3.

Laboratory Analyses with State Affect Measures. ***Resting and Baseline Analyses.*** To assess whether participants differed on state affect measures at rest prior to beginning stressor tasks, in the regular versus sodium-loaded diet conditions, and as a function of sex, the following analyses were conducted on average resting affect ratings taken as baseline before the first of three stressor tasks for participants, in each diet condition. First, 2(diet) x 2(sex) MANOVAs were conducted, followed by 2 x 2 ANOVAs with syntax modified to look only at the significant effects found in the MANOVA analyses. Similarly, 2(diet) x 2(sex) x 3(stressor tasks) MANOVA and

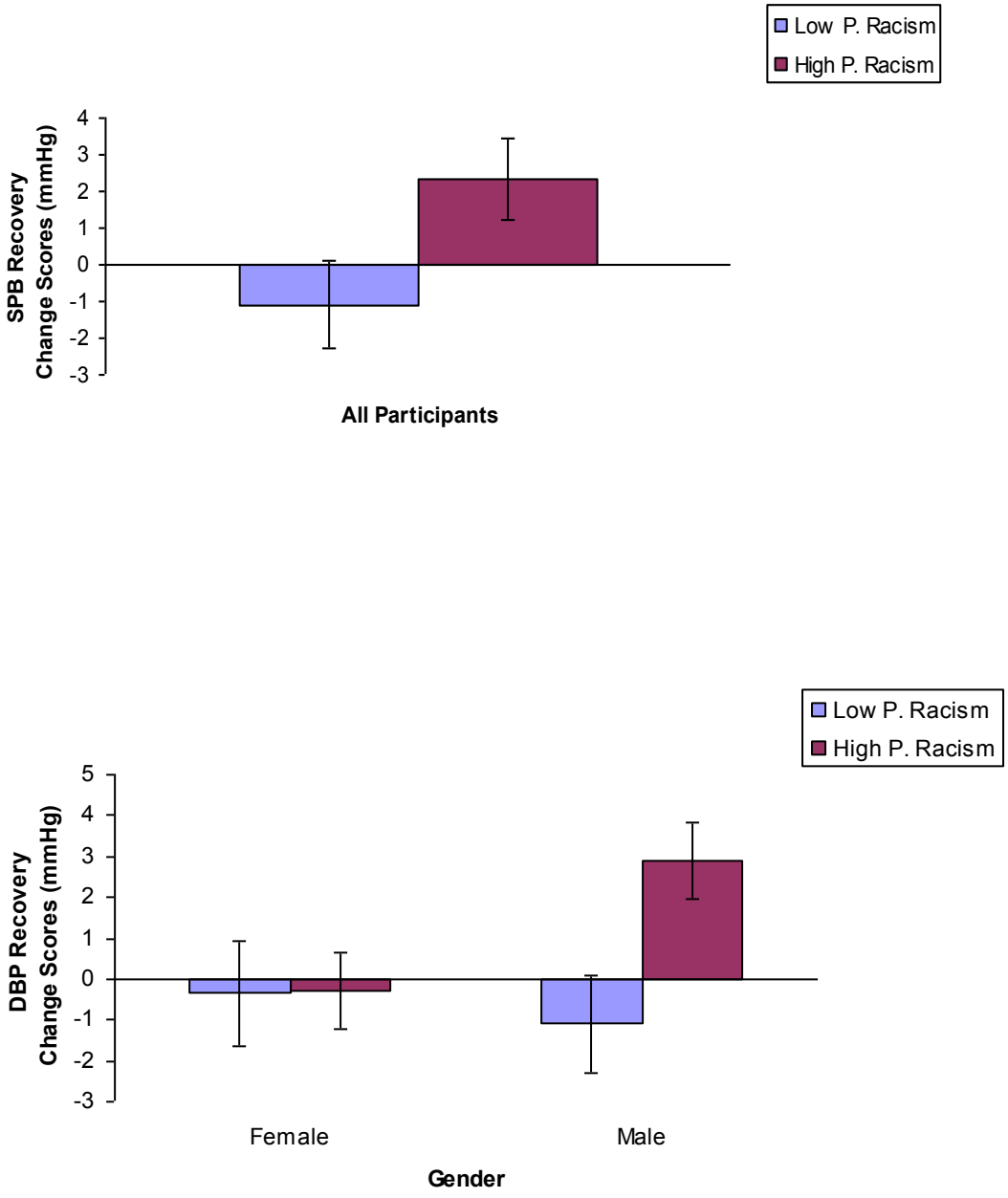


Figure 3. Cardiovascular Recovery on the Math Task, as a function of Gender, and as a function of Perceived Racism.

ANOVA analyses were conducted to assess whether participants differed on state affect at baseline before each of the stressor tasks, in each diet condition. No significant effects were found in the above analyses. Means and standard deviations for baseline state affect measures as a function of dietary sodium and sex are presented in Table 7.

Baseline to Stress Change in State Affect Measures. To assess whether participants differed on state affect changes in response to stressor tasks, in the regular versus sodium-loaded diet conditions, and as a function of sex, the following analyses were conducted on change scores for state affect measures. First, a 2(diet) x 2(sex) x 3(stressor tasks) MANOVA was conducted, followed by 2 x 2 x 3 ANOVAs with syntax modified to look at the significant effects found in the MANOVA analyses. Results of these analyses are reported below. Means and standard deviations for state affect measures as a function of diet, sex, and stressor tasks are presented in Table 8.

MANOVA analysis revealed a significant multivariate effect for Task, $F(16,20) = 4.3$, $p < .002$. ANOVA analyses looking at Task found significant effects for the following state affect measures: Nervous, $F(2,40) = 13.0$, $p < .001$; Anxious, $F(2,41) = 7.4$, $p < .003$, Sad, $F(2,39) = 4.5$, $p < .018$; and Angry, $F(2,40) = 9.0$, $p < .002$; Participants were most nervous and anxious on the Math task, while they were most sad and angry on the Discrimination Recall task. No significant changes were found for state affect measures of tense, annoyed, irritated, and depressed.

Association between Baseline State Affect and Baseline Cardiovascular Measures. Correlation analyses between each baseline state affect measure and each baseline cardiovascular measures were conducted for each diet condition separately. In

Table 7

Means and Standard Deviations of Baseline State Affect Measures as a Function of Sodium, Stressor Task, and Gender

	Regular Diet						Sodium Diet					
	CP		DR		M		CP		DR		M	
State Affect Measures	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
NER												
Male	2.88	2.0	3.24	2.4	2.20	1.7	2.47	1.9	2.79	2.0	3.15	2.7
Female	4.09	2.9	2.89	1.7	3.20	2.5	4.06	3.0	3.17	2.9	3.24	3.0
TEN												
Male	2.47	1.4	3.06	2.4	2.81	2.0	2.84	1.9	3.91	3.0	3.88	3.2
Female	4.87	3.4	4.41	3.1	3.89	2.7	4.82	3.4	3.80	3.2	4.44	3.7
ANX												
Male	5.29	3.8	4.41	3.5	3.91	3.1	4.21	2.7	4.27	3.3	4.03	3.3
Female	5.43	3.8	4.65	2.9	4.70	3.3	5.43	3.9	5.15	3.7	5.35	4.3
ANN												
Male	2.56	1.5	2.63	1.5	2.59	1.5	2.72	2.1	3.69	2.9	2.88	2.1
Female	3.56	3.1	3.48	2.8	3.20	2.8	4.09	3.7	3.20	2.7	3.50	2.8
SAD												
Male	2.13	1.9	2.44	2.1	2.00	1.9	1.69	0.6	1.91	0.9	1.75	0.8
Female	3.15	2.6	2.78	2.0	2.91	2.0	3.09	2.8	2.67	2.2	2.93	2.8
IRR												
Male	2.29	1.6	2.44	1.6	2.35	1.6	2.77	2.3	3.91	3.2	2.88	2.1
Female	3.48	3.0	3.06	2.4	3.00	2.4	3.89	3.5	2.70	2.2	3.89	3.1
ANG												
Male	1.84	1.0	1.84	0.9	1.69	0.7	1.72	0.9	1.97	0.8	2.13	1.4
Female	2.87	2.4	2.57	1.9	2.98	2.6	2.24	2.0	2.00	1.3	2.44	1.8
DEP												
Male	2.40	2.1	2.23	1.8	2.40	2.2	1.77	0.7	2.07	1.0	1.83	1.0
Female	2.78	2.0	2.59	1.8	2.82	2.2	2.85	2.6	2.76	2.3	2.74	2.5

Table 8

Means and Standard Deviations of State Affect Change Scores as a Function of Sodium, Stressor Task, and Gender

	Regular Diet						Sodium Diet					
	CP		DR		M		CP		DR		M	
State Affect Measures	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
NER												
Male	-0.47	2.3	-0.35	2.5	2.50	3.1	0.59	2.2	1.03	4.0	0.85	3.1
Female	-0.42	2.7	1.81	2.7	2.37	3.1	-0.15	2.9	0.75	3.2	1.48	2.7
TEN												
Male	1.28	2.4	1.47	2.9	3.88	4.2	1.97	3.7	1.50	3.7	1.53	2.1
Female	0.79	3.1	1.54	2.7	2.39	2.7	0.54	3.1	1.35	3.2	1.30	4.1
ANX												
Male	-0.21	2.9	-0.79	3.7	2.15	3.4	1.35	3.8	0.06	1.8	1.68	4.0
Female	-0.11	2.9	1.31	2.4	2.07	2.6	0.35	2.5	0.61	2.8	0.67	4.0
ANN												
Male	1.34	2.7	1.19	2.5	1.34	2.6	0.41	1.8	-0.13	2.4	0.44	1.8
Female	1.31	3.1	1.48	3.1	1.81	3.9	0.46	2.6	1.71	3.7	1.00	4.6
SAD												
Male	0.03	0.6	0.31	1.7	0.41	0.8	-0.06	0.5	0.41	1.3	0.06	0.5
Female	-0.27	1.0	1.77	2.5	0.67	1.7	-0.48	2.0	1.56	2.6	0.29	2.1
IRR												
Male	1.59	3.0	1.26	2.2	1.21	2.5	1.59	2.2	-0.18	3.3	0.85	2.5
Female	1.70	2.7	2.19	3.7	2.37	3.7	0.96	3.2	2.44	3.7	0.56	3.7
ANG												
Male	0.91	2.5	2.19	2.6	0.53	1.0	1.13	2.1	1.28	2.2	0.41	1.3
Female	0.33	1.7	2.24	3.1	0.44	2.9	0.69	2.0	2.69	3.5	0.89	2.6
DEP												
Male	-0.17	1.1	0.47	1.0	0.23	1.1	0.20	1.5	0.00	1.1	0.70	1.8
Female	0.17	1.7	1.07	2.2	0.67	1.7	0.06	1.2	0.78	1.8	0.20	1.8

the regular diet condition, significant correlations were found for the following: On both the Cold Pressor and Discrimination Recall tasks, DBP was positively correlated with Anxious, $r(44) = .354$, $p < .019$ and $r(44) = .420$, $p < .006$, respectively.

In the sodium-loaded diet condition, significant correlations were found for the following: On the Cold Pressor task, DBP was positively correlated with Annoyed, $r(42) = .325$, $p < .032$. On the Discrimination Recall task, CO was positively correlated with Nervous, $r(42) = .421$, $p < .007$ while TPR was negatively correlated with Nervous, $r(44) = -.310$, $p < .047$. On the Math task, CO was positively correlated with Nervous, $r(44) = .334$, $p < .028$, while DBP was positively correlated with Annoyed, $r(43) = .382$, $p < .011$.

In summary higher baseline DBP measures was associated with participants being more anxious in the regular diet condition, and with participants being more annoyed in the sodium-loaded diet condition. Additionally, higher baseline CO measures and lower baseline TPR measures were associated with participants being more nervous in the sodium-loaded diet condition.

Association between State Affect Change and Cardiovascular Reactivity Measures. Correlation analyses between each state affect change measures and cardiovascular reactivity measure were conducted for each diet condition separately. In the regular diet condition, significant correlations were found for the following: On the Cold Pressor task, HR was positively correlated with Irritated, $r(44) = .376$, $p < .013$ and Depressed, $r(44) = .325$, $p < .032$, while CO was positively correlated with Irritated, $r(44) = .390$, $p < .010$ and Angry, $r(44) = .372$, $p < .014$. On the Discrimination Recall

task, HR was also positively correlated with Depressed, $r(44) = .347$, $p < .021$. On the Math task, SBP was also positively correlated with Depressed, $r(44) = .300$, $p < .049$.

In the sodium-loaded diet condition, significant correlations were found for the following on the Discrimination Recall task: HR was negatively correlated with Tense, $r(42) = -.375$, $p < .015$, Anxious, $r(42) = -.316$, $p < .042$, and Angry, $r(41) = -.404$, $p < .010$; CO was negatively correlated with Anxious, $r(42) = -.378$, $p < .015$, Annoyed, $r(42) = -.386$, $p < .013$ and Angry, $r(41) = -.450$, $p < .004$; and SBP was negatively correlated with Anxious, $r(43) = -.315$, $p < .041$. No significant correlations were found for the Cold Pressor and Math tasks.

In summary, in the regular diet condition, greater HR, CO, and SBP reactivity were associated with participants feeling more irritated, angry, and depressed, while the opposite relationship was found in the sodium-loaded diet condition; i.e. lower HR, CO and SBP reactivity were associated with participants feeling more tense, anxious, angry and annoyed.

Association between State Affect Change and Cardiovascular Recovery Measures. Correlation analyses between each state affect change measure and each cardiovascular recovery measure were conducted for each diet condition separately. In the regular diet condition, a significant positive correlation was found between HR measures and Irritated, $r(43) = .334$, $p < .029$, on the Cold Pressor task. In the sodium-loaded diet condition, significant correlations were found for the following: On the Cold Pressor task, SBP measures was positively correlated with Depressed, $r(43) = -.405$, $p < .007$, and on the Discrimination Recall task, HR measures were negatively correlated with Irritated, $r(42) = -.315$, $p < .043$. In summary, participants who endorsed being more

irritated at the end of stressor tasks showed greater HR (lesser recovery) in the regular diet condition and lower HR (greater recovery) in the sodium-loaded diet condition, while those who endorsed feeling more depressed after stressor tasks demonstrated higher SBP (lesser recovery) in the sodium-loaded diet condition.

Ambulatory Protocol Analyses

Sodium and Ambulatory Sleep and Wake Measures. To assess whether participants differed on ambulatory blood pressure measures as a function of diet (sodium-loaded versus regular), and as a function of sex, 2(diet) x 2(sex) repeated measures MANCOVAs were conducted on mean ambulatory measures for MAP, SBP, DBP, and HR, in the sleep and wake periods. This was followed by 2 x 2 ANCOVAs looking at significant effects found in the MANCOVA analyses. Means and standard deviations for ambulatory measures as a function of dietary condition and sex are presented in Table 9. MANCOVA analyses found an effect for Diet in the sleep period, $F(4,26) = 2.8$, $p < .047$, but found no effect in the wake period. ANCOVA analyses looking at Diet found a significant effect for HR, $F(1,29) = 10.5$, $p < .004$, with participants having higher sleep heart rate in the regular diet compared to the sodium-loaded diet. No effect of diet on blood pressure measures was found.

Gender and Ambulatory Sleep and Wake Measures. To assess whether participants differed on ambulatory blood pressure measures as a function of sex, one-way MANCOVAs were conducted on mean ambulatory measures for MAP, SBP, DBP, and HR, in the sleep and wake periods, followed by one-way ANCOVAs to look at significant effects found in the MANCOVA analyses. MANCOVA analyses found effects for Sex in both the sleep and wake periods, $F(4,37) = 8.42$, $p < .001$,

Table 9

Means and Standard Deviations of Ambulatory Blood Pressure Measures as a Function of Diet and Gender

		Wake				Sleep			
		Regular Diet		Sodium Diet		Regular Diet		Sodium Diet	
		Mean	SD	Mean	SD	Mean	SD	Mean	SD
MAP									
	Male	93.17	7.48	93.06	7.63	82.24	10.51	83.78	6.85
	Female	89.33	7.37	89.34	7.65	78.91	8.55	82.19	10.28
SBP									
	Male	133.69	13.88	132.60	12.27	124.85	15.03	127.99	13.62
	Female	128.20	12.67	128.68	12.93	121.35	17.40	125.20	15.09
DBP									
	Male	73.41	6.02	73.56	6.30	63.12	9.33	62.24	6.68
	Female	70.31	6.60	70.24	6.06	58.29	6.11	61.74	9.26
HR									
	Male	76.28	10.56	76.29	9.81	61.40	9.54	57.84	5.08
	Female	86.42	7.66	83.10	9.12	72.67	10.35	71.47	11.67

and $F(4,39) = 6.95$, $p < .001$, respectively. ANCOVA analyses looking at Sex found significant effects for HR in both the sleep and wake periods, $F(1,46) = 22.49$, $p < .001$ and $F(1,44) = 14.47$, $p < .001$, respectively. Females were found to have greater heart rate in comparison to men, in both the sleep and wake periods. No effect of sex on blood pressure measures was found.

Sodium and Percentage Blood Pressure Dipped at Night. To assess whether participants differed on percentage ambulatory blood pressure dipped as a function of diet (sodium-loaded versus regular), and as a function of sex, a 2(diet) x 2(sex) repeated measures MANCOVA was conducted on percentage dipped calculations for ambulatory MAP, SBP, DBP, and HR measures. This was followed by 2 x 2 ANCOVAs looking at significant effects found in the MANCOVA analysis. Means and standard deviations for percentage blood pressure dipped as a function of diet condition, and as a function of sex, are presented in Table 10. MANCOVA analysis revealed a marginally significant effect for Diet, $F(4, 24) = 2.5$, $p < .067$. ANCOVA analyses found a significant effects for MAP, $F(1,27) = 5.1$, $p < .033$, and HR, $F(1,27) = 4.7$, $p < .041$, and marginally significant effects for SBP, $F(1,27) = 4.2$, $p < .051$ and DBP, $F(1,27) = 3.7$, $p < .065$. As shown in Figure 4, participants demonstrated a trend towards less blood pressure dipping on the sodium-loaded diet compared to the regular diet condition.

Anger-in and Ambulatory Sleep and Wake Measures. To assess whether participants differed on ambulatory measures as a function of sex, and as a function of anger-in, 2(sex) x 2(anger-in) MANCOVA analyses was conducted on mean ambulatory measures for MAP, SBP, DBP, and HR, in the sleep and wake periods. Significant effects were followed up by 2 x 2 ANCOVA analyses. MANCOVA analysis revealed a

Table 10

Means and Standard Deviations of Percentage Blood Pressure Dipped as a Function of Diet and Gender

		Regular Diet		Sodium Diet	
ABP Percentage Dipped		Mean	SD	Mean	SD
MAP					
	Male	11.33	6.67	9.50	8.12
	Female	10.65	8.83	8.85	6.24
SBP					
	Male	7.58	4.66	3.30	9.49
	Female	4.62	9.59	2.63	6.80
DBP					
	Male	14.23	9.91	14.75	8.95
	Female	15.85	8.81	13.61	8.36
HR					
	Male	18.12	9.59	22.18	7.24
	Female	15.16	9.93	15.55	8.84

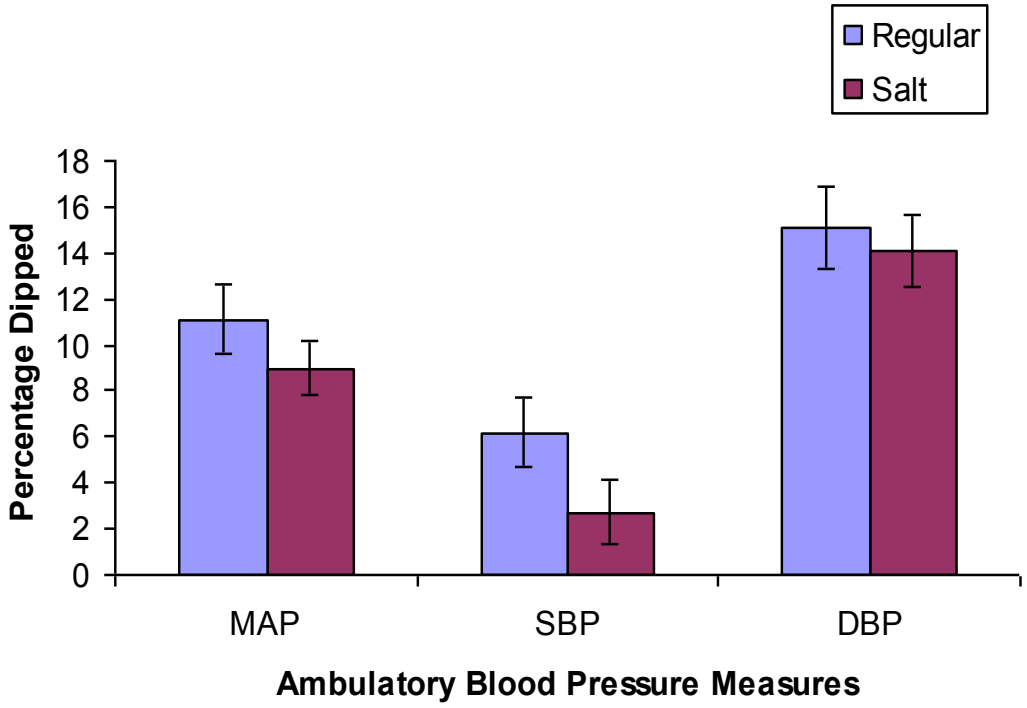


Figure 4. Percentage Blood Pressure Dipped as a function of Diet.

significant effect for Anger-in, $F(4, 33) = 3.1$, $p < .029$, in the sleep period. Subsequent ANCOVA analyses looking at Anger-in did not find any significant effects. MANCOVA analysis revealed a significant effect for Sex, $F(4, 35) = 6.3$, $p < .002$, for the wake period. Subsequent ANCOVA analyses maintained an effect for Sex, $F(1, 38) = 20.5$, $p < .001$, with females having higher HR than males in the wake period.

Anger-in and Percentage Blood Pressure Dipped at Night. To assess whether participants differed on percentage blood pressure dipped at night as a function of sex, and as a function of anger-in, a 2(sex) x 2(anger-in) MANCOVA analysis was conducted on percentage dipped calculations for MAP, SBP, and DBP ambulatory measures. No significant findings emerged for the effect of anger-in on percentage blood pressure dipped at night.

Anger-out and Ambulatory Sleep and Wake Measures. To assess whether participants differed on ambulatory blood pressure measures as a function of sex, and as a function of anger-out, a 2(sex) x 2(anger-out) MANCOVA analysis was conducted on mean ambulatory measures for MAP, SBP, DBP, and HR. This was followed by 2 x 2 ANCOVAs, to look at significant effects found in the above MANCOVA analysis. These analyses were conducted separately for the sleep and wake periods. MANCOVA analyses revealed a significant effect for Sex, $F(4, 33) = 8.0$, $p < .001$, for the sleep period, as well as for the wake period, $F(4, 35) = 6.7$, $p < .001$. Subsequent ANCOVA analyses looking at Sex found significant effects for MAP, $F(1, 36) = 4.3$, $p < .046$, and HR, $F(1, 36) = 19.4$, $p < .001$, in the sleep period, and for HR, $F(1, 38) = 22.0$, $p < .001$, in the wake period. Males had higher MAP than females during the sleep period, and females had higher HR than males during both the sleep and wake periods.

Anger-out and Percentage Blood Pressure Dipped at Night. To assess whether participants differed on percentage blood pressure dipped at night as a function of sex, and as a function of anger-out, a 2(sex) x 2(anger-out) MANCOVA analysis was conducted on percentage dipped calculations for MAP, SBP, and DBP ambulatory measures. A significant interaction for Sex x Anger-out was found, $F(4, 32) = 2.9$, $p < .040$. ANCOVA analyses looking at the latter interaction found significant effects for MAP, $F(1, 35) = 7.0$, $p < .013$, and SBP, $F(1, 35) = 8.7$, $p < .007$, and a marginally significant effect for DBP, $F(1, 35) = 4.0$, $p < .055$. Post-hoc analyses revealed that males had significantly greater dipping on MAP, $F(1, 10) = 9.2$, $p < .014$; SYS, $F(1, 10) = 10.9$, $p < .009$; and DBP, $F(1, 10) = 5.4$, $p < .044$, in the high anger-out compared to the low anger-out condition, as shown in Figure 5.

Perceived Racism and Ambulatory Sleep and Wake Measures. To assess whether participants differed on ambulatory blood pressure measures as a function of sex, and as a function of perceived racism, a 2(sex) x 2(perceived racism) MANCOVA analysis was conducted on mean ambulatory measures for MAP, SBP, DBP, and HR. This was followed by 2 x 2 ANCOVAs to look the significant effects found in the MANCOVA. These analyses were conducted separately for the sleep and wake periods. MANCOVA analyses revealed a significant effect for Sex, $F(4, 34) = 8.4$, $p < .001$, for the sleep period, as well as for the wake period, $F(4, 36) = 6.8$, $p < .001$. ANCOVA analyses looking at Sex found a significant effect for HR in both the sleep and wake periods, $F(1, 37) = 25.0$, $p < .001$, and $F(1, 39) = 24.5$, $p < .001$, respectively, with females having higher HR than males.

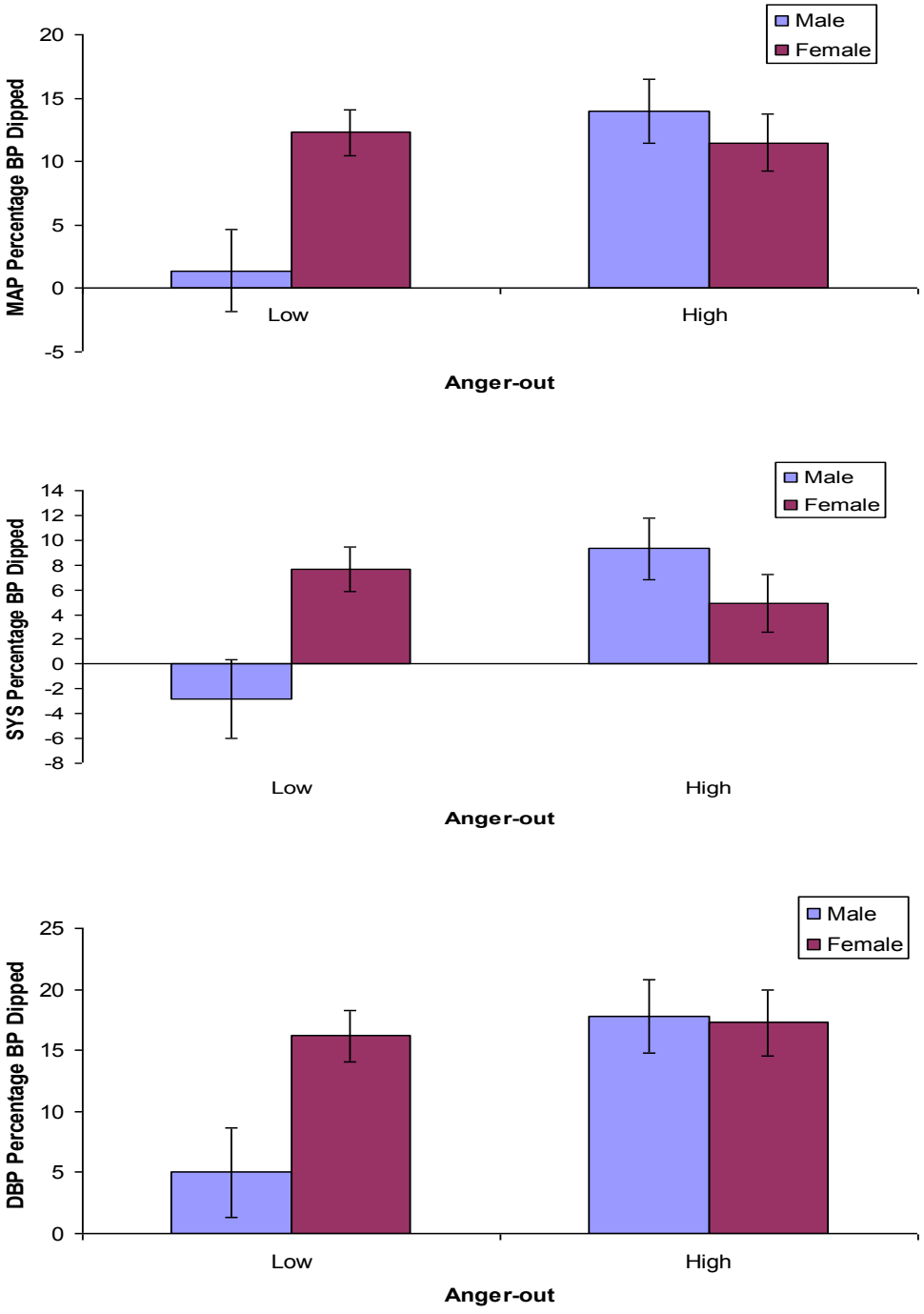


Figure 5. Percentage Blood Pressure Dipped as a function of Gender, and as a function of Anger-out.

Perceived Racism and Percentage Blood Pressure Dipped at Night. To assess whether participants differed on percentage blood pressure dipped at night, as a function of sex, and as a function of perceived racism, a 2(sex) x 2(perceived racism) MANCOVA analysis was conducted on percentage dipped calculations for MAP, SBP, and DBP ambulatory measures. No significant findings emerged for the effect of perceived racism on percentage blood pressure dipped at night.

Discussion

The goal of the present study was to examine the effects of sodium, stress, gender, anger-in, and perceived racism, on cardiovascular reactivity to and recovery from laboratory stress, on ambulatory sleep and wake blood pressure, and on nocturnal blood pressure dipping, and how these associations may moderate hypertension risk in the Black Canadian population. These selected factors were chosen for investigation from among the numerous biopsychosocial variables implicated in hypertension risk, given evidence suggesting their particular importance for risk within the Black population. This study, employing a joint laboratory and ambulatory blood pressure protocol, is the first to investigate the above mentioned combination of proposed risk factors in a Black population, and more so, within a Black Canadian population. Given that most previous studies have been conducted with American Black populations, the present study contributes to, and addresses gaps in the research literature, for factors contributing to high risk for hypertension among Canadian Blacks.

Thirteen hypotheses were tested with relevant multivariate and univariate analysis of variance procedures. The main findings in the laboratory protocol include significant gender differences in cardiovascular reactivity to stress, a significant interaction between gender, stressor type, and perceived racism for cardiovascular recovery from stress, and a marginally significant interaction between sodium and gender for cardiovascular recovery from stress. In the ambulatory blood pressure protocol, the main findings include a marginally significant effect of sodium on nocturnal blood pressure dipping, an interaction between gender and anger-out on blood pressure dipping, and gender differences in ambulatory sleep and wake heart rate measures.

Sodium, Stress, Cardiovascular Reactivity, and Cardiovascular Recovery

The first two hypotheses involved cardiovascular responses to laboratory stressor tasks under different diet conditions. The first hypothesis stated that Black individuals would have greater cardiovascular reactivity to stressor tasks in the sodium-loaded condition compared to the regular diet condition, while the second hypothesis stated that Black individuals would have decreased cardiovascular recovery in the sodium-loaded compared to the regular diet condition. These hypotheses are based on numerous studies showing that exaggerated cardiovascular reactivity to acute stress is linked to the development of essential hypertension or coronary heart disease (e.g. Chida & Steptoe, 2010; Matthews et al., 2004; Matthews et al., 2006; Trieber et al., 2003); the prominent theory that racial disparities in hypertension risk may be explained by heightened cardiovascular responses to stress in Black individuals (Anderson, 1989) and supportive evidence from studies showing the same (e.g. Kelsey et al., 2000; Light et al., 1987); evidence showing that hypertension status and risk for hypertension, have been associated with delayed cardiovascular recovery after exposure to acute stressors (Borghi et al., 1986; Schuler & O'Brien, 1997; Steptoe & Marmot, 2005; Stewart & France, 2001); and evidence suggesting that interaction between stress and dietary sodium influences cardiovascular responses (e.g. Anderson et al., 1983; Nilsson et al., 1985). Unexpectedly, no main effect of sodium on cardiovascular reactivity or cardiovascular recovery was found in this study.

In review of the literature, while the bulk of the evidence to date supports increased cardiovascular reactivity to sodium-loading, a few prominent studies in the literature have also found a lack of support for the effect of sodium on cardiovascular

responses (e.g. Dimsdale, Ziegler, Mills, Delehanty & Berry, 1990; Falkner & Kushner, 1989), similar to what was found in this study. This will be discussed below, as it may relate to lack of confirmation for this study's hypotheses regarding the effects of sodium on cardiovascular reactivity and recovery.

Consistent with results of the present study, Falkner and Kushner (1989) failed to find a main effect for sodium in a study looking at long-term sodium loading (14 days) on stress-induced cardiovascular responses in normotensive and marginally hypertensive young adults. In that study, high sodium intake did not augment blood pressure or heart rate response to a Math stressor task. The researchers found however, that individuals classified as sodium-insensitive had a higher rate of urinary sodium excretion than sodium-sensitive individuals. They further found a significant negative correlation between blood pressure change and sodium excretion for sodium-sensitive individuals - i.e. as sodium excretion decreased, blood pressure increased. Such findings strongly implicate sodium-sensitivity as an important factor moderating cardiovascular responses to stressful stimuli under high sodium conditions.

The importance of sodium-sensitivity among Black populations is strongly suggested in studies that show a higher rate of sodium-sensitivity among Blacks compared to Whites in North America (e.g. Falkner & Kushner, 1990; Weinberger et al., 1986). As well, studies show that a greater proportion of sodium sensitive individuals is present in both normotensive and hypertensive Black populations (Sowers et al., 1988; Zemel et al., 1988). Additionally, the wider literature shows that across both sexes, there is heterogeneity in blood pressure response to sodium-loading, with sodium-sensitive individuals responding to sodium-loading with an increase in blood pressure, whereas the

blood pressure of sodium-insensitive/resistant individuals remain unchanged under the same dietary conditions (Weinberger, 1996). Unfortunately sodium-sensitivity was not measured in the present study. Thus, it is possible that differences in sodium-sensitivity among Black individuals might have contributed to the lack of findings for the effect of sodium-loading on cardiovascular reactivity and recovery in this study. Future studies would need to control for sodium-sensitivity in order to elucidate true associations between sodium, stress, and cardiovascular reactivity and recovery, and implications for hypertension risk in the Canadian Black population.

In addition to variations in sodium-sensitivity between individuals, male/female differences in terms of sex hormones may have also contributed to the lack of findings for a main effect of sodium on cardiovascular responses in the present study. As discussed in the review article on sex hormones and hypertension (Dubey, Oparil, Imthurn, & Jackson, 2002), studies suggest that male testosterone is a pro-hypertensive hormone (e.g. Chen & Meng; 1991; Reckelhoff, Zhang, Granger, 1998; Reckelhoff, Zhang, & Srivastava, 2000). There is evidence that testosterone may have its effects on blood pressure through modulating vascular tone (e.g. Ceballos et al., 1999; Webb, McNeill, Hayward, de Ziegler & Collins, 1999). On the other hand, studies suggest that the female hormone estradiol, may be protective against hypertension in premenopausal women (e.g. Chapman et al., 1997; Dunne, Barry, Ferriss, Grealy, & Murphy, 1991; Karpanou, Vyssoulis, Georgoudi, Toutouza, & Toutouzas, 1993). Some researchers have found that female hormones influence the relationship between sodium and cardiovascular reactivity. For example, Pechere-Bertschi and Burnier (2004) found that

the blood pressure of pre-menopausal women (in both phases of their menstrual cycle) was insensitive to sodium-loading compared to post-menopausal women.

The above discussed evidence is consistent with the differential prevalence of hypertension observed for the genders; i.e. hypertension risk for premenopausal women is less than that of men, but this pattern becomes reversed for women in their postmenopausal years where prevalence in women exceeds that of men (Burt et al., 1995; Stamler et al., 1976; August & Oparil, 1999). It would follow, that if premenopausal women's blood pressure is not responsive to sodium-loading, but blood pressure of age similar men is likely to vary with sodium-loading, then a main effect for sodium-loading on blood pressure (across the genders) may turn out to not be significant. It might also follow that an interaction between gender and sodium would be indicated. Along these lines, a notable diet x gender interaction (though statistically insignificant; $p < .076$), was found in the MANCOVA analysis for cardiovascular recovery from stress. Follow-up with ANCOVA analyses found a significant effect on DBP recovery. Specifically, it was found that males showed less DBP recovery compared to females in the regular diet condition, while females showed less DBP recovery compared to males in the sodium-loaded diet condition.

The above mentioned findings in the sodium condition are puzzling, since the opposite would be expected. That is, males would be expected to show less DBP recovery under the sodium-loaded condition compared to females, given evidence as previously discussed, for the protective effects of the female hormone, estradiol, on blood pressure (e.g. Chapman et al., 1997), including the relative insensitivity of females to sodium-loading (Pechere-Bertschi & Burnier, 2004), and for the pro-hypertensive

(vascular) effects of male hormone, testosterone (e.g. Reckelhoff, Zhang, & Srivastava, 2000). A possible explanation for the unexpected findings in the sodium-condition may be that these are chance findings, especially given that these are linked to marginally significant MANCOVA results. Alternatively, it is possible that these findings were influenced by the significant differences in sample sizes; i.e. a small number of males (n = 15) compared to a significantly larger number of females (n = 27) who were compliant with the sodium diet and included in the analyses. None-the-less, these findings implicate complex gender x sodium/sodium-sensitivity interactions influencing cardiovascular recovery from stress that warrant investigation in future studies. Future research would benefit from conducting studies with each gender separately, or from having larger sample sizes in mixed gender studies in order to allow for sufficient power to investigate interactions and/or allow for separate analyzes of male and female data. Such research would help to elucidate important patterns influencing cardiovascular recovery which may contribute to risk for development of hypertension in the Black population.

Gender, Stress, Cardiovascular Reactivity, and Cardiovascular Recovery

The third and fourth hypotheses for this study involved gender differences in cardiovascular reactivity to laboratory stressors. The third hypothesis stated that males would have greater blood pressure/vascular reactivity than females when exposed to laboratory stressors, while the fourth hypothesis stated that females would have greater heart rate/myocardial reactivity than males when exposed to such stress. This follows from research showing that males typically exhibit greater cardiovascular reactivity to stressors than women (e.g. Girdler et al., 1990; Matthews et al., 2001; Stoney et al., 1988; Vogele et al., 1997), with males tending to respond with greater blood pressure responses

and women tending to react with greater heart rate responses (Allen et al., 1993; Fichera & Andreassi, 2000; Girdler et al., 1990; Vogele et al., 1997).

Consistent with this study's hypothesis for vascular reactivity in Black males, it was found that males demonstrated significant increases in TPR reactivity (indicative of vasoconstriction) compared to females who showed decreases in TPR response to stress (indicative of vasodilatation). Additionally, it was found that males demonstrated significantly greater SV reactivity compared to females in this study; specifically, males showed significant decreases in SV on the math and discrimination recall tasks. While SV (the amount of blood pumped out by the heart per beat) response is not typically defined as a vascular response, decreased SV as a result of stress could be a result of a reduction in return of blood to the heart, which may be due to vascular factors such as venous vasoconstriction resulting from effects of catecholamine or other vasoconstrictor agents (Light, Turner, Hinderliter, & Sherwood, 1993).

The observed increase in vascular tone/vasoconstriction (TPR) observed for males is consistent with research underlying the importance of these hemodynamics in the risk for development of hypertension (Folkow, 1956). Folkow hypothesized that over time, repeated stress affects peripheral vasculature by causing a structural change in the wall-to-lumen ratio of arterioles, ultimately resulting in sustained increase in TPR and future hypertension. The findings of this study suggest that young Black adult males may be particularly vulnerable to the above described mechanism underlying risk for future hypertension, which is consistent with research supporting the classification of Black males as "vascular responders", compared to Black females and White males and females (e.g. Llabre, Klein, Saab, McCalla, & Schneiderman, 1998). Greater vascular reactivity

and associated risk for hypertension in Black males, is consistent with epidemiological research showing a higher risk of hypertension in males compared to females during young-middle adulthood, and a higher prevalence of hypertension in Blacks compared to Whites in North America. It is suggested that these disparities in hypertension risk may be linked to differences in underlying physiological mechanisms related to ethnicity, as well as differences in underlying physiology of the sexes.

Research suggests that underlying ethnic physiological differences contributing to greater vascular reactivity in Blacks compared to Whites are associated with both structural and functional differences of the resistance vessels. This includes greater vascular reactivity to sympathetic stimulation, attenuated responses to vasodilators, and relatively narrow vascular lumen diameter, in Black compared to Whites (Taherzadeh et al., 2010) With regards to underlying sex differences in physiological differences contributing to greater vascular reactivity in males compared to females, it has been found that the female hormone, estrogen, acts to stimulate nitric oxide in the endothelium (the thin layer of cells that lines the interior surface of blood vessels) leading to vasodilatation in women while the male hormone testosterone acts to stimulate the renin-angiotensin system, leading to increased vasoconstrictor tone in men (Liberman et al., 1994; Orshal & Khalil, 2004; Reckelhoff, 2001). Alternatively, decreased TPR and resultant vasodilatation in females may be due to predominance of beta receptor activation to stress-related sympathetic nervous system activity, while increased TPR and resultant vasoconstriction seen in men may be due to predominance of alpha receptor activation to sympathetic activity. As previously mentioned, there is evidence that males and females have differential concentrations of and/or sensitivities to alpha and beta

adrenergic receptors (Freedman et al., 1987). Following from the above, it is possible ethnic and sex differences contribute in additive or interactive ways to increase vascular reactivity and associated hypertension risk in Black males compared to Black females, which is supported by findings in this study.

The fourth hypothesis stated that females would have greater HR/myocardial reactivity than males when exposed to laboratory stressors. It was found that females demonstrated greater CO reactivity compared to men in this study. This is consistent with the hypothesis for greater myocardial reactivity in females, given that CO represents the volume of blood pumped by the heart per minute (a product of HR and SV). It is noteworthy, that despite the increase in CO in response to stress, there were no significant increases in blood pressure for women. This may be explained by the decrease in TPR (vasodilatation) that was observed for women, potentially due to beta-adrenoceptor activation to stress-related sympathetic nervous system activity. Additionally, as previously discussed, female sex hormones are known to facilitate vasodilatation compared to men (Liberman et al., 1994; Orshal & Khalil, 2004; Reckelhoff, 2001), which may be protective in reducing risk for hypertension in young-middle adulthood females.

The fifth hypothesis stated that males will have lesser cardiovascular recovery than females after exposure to laboratory stressor tasks. This follows from research showing that in general, males demonstrate greater elevations in blood pressure than females during the recovery period after exposure to laboratory stress (e.g. Glynn et al., 2002; Matthews et al., 2001). This hypothesis was not confirmed as a main effect for gender on cardiovascular recovery was not found in this study. However, interestingly, an

interaction between gender and perceived racism (discussed in the section below) was found in this study, indicating that psychosocial factors may moderate the relationship between gender and cardiovascular recovery.

Perceived Racism, Cardiovascular Reactivity, and Cardiovascular Recovery

The sixth and seventh hypotheses examined relationships between perceived racism and cardiovascular reactivity, and perceived racism and cardiovascular recovery, respectively. These hypotheses build on research literature suggesting that perceived racism is a significant chronic social stressor affecting cardiovascular responses and hypertension risk in Black Americans (Brondolo, Rieppi, Kelly & Gerin, 2003). The sixth hypothesis stated that individuals with high versus low perceived racism scores will have greater cardiovascular reactivity to laboratory stressors. This hypothesis was unexpectedly not confirmed. This is surprising as it differs from studies done in the United States with American Blacks that have demonstrated a positive association between high perceived racism and high cardiovascular reactivity. This is the first study to investigate effects of perceived racism on Black Canadians; therefore no comparative research exists to offer explanations for the above findings. However, it might be speculated that the effects of racism/perceived racism may be different in the extent to which it affects psychological and physiological variables in Black Canadians compared to Black Americans. This could be based on differences in race-related history and associated social-economic-political influences on Canadian society compared to that of the United States.

A significant difference between the histories of Blacks in the United States and Canada is that American Blacks are more homogenous in their historical descent

compared to Canadian Blacks who are more diverse in this respect. Specifically, the majority of American Blacks are direct descendants of African Blacks who were brought by forced migration to the United States during the slavery period spanning the1600's to the 1800's (Franklin & Moss, 2001), while relatively few Canadian Blacks are direct descendants of African slaves brought to Canada during those early times (Fong, 1996). Instead, as discussed in the Fong article, most Canadian Blacks are immigrants, the majority of whom have voluntarily moved to Canada from the Caribbean in more recent times. Research shows that more than 60% of Canadian Blacks have immigrated since the 1970's (Fong, 1996). A smaller subset of the Canadian Black population include immigrants from Africa, also in recent times, and those who are descendents from American Blacks who immigrated to Canada during significant American historical events (Fong, 1996). The effects of intense social, political, and economic upheavals during the history of slavery and movements that eventually led to the abolishment of slavery in the United States, have left lasting effects on American society, including the significant problems of racism and discrimination faced by American Blacks (Franklin & Moss, 2001). While Canada is not free of racism, the extent to which racism and/or perceived racism affects Canadian Blacks (psychologically and physiologically) is likely mitigated to some extent by the unique history and related social-political-cultural experiences of Blacks in Canadian society, compared to that of Blacks in American society. No previous study in Canada has looked at cardiovascular responses to perceived racism in the Black population. Further studies are needed to investigate the effects of racism/perceived racism and cardiovascular reactivity in a Canadian context, and how

these factors might be linked to risk for developing hypertension within the Canadian Black population.

Another possible reason for the null findings for the effect of perceived racism on cardiovascular reactivity may be that potential moderating/mediating variables not measured in this study could be involved in explaining the relationship between the former two variables. Along these lines, an important body of research suggests that the way in which a Black individual identifies with his/her race, i.e. racial identity, affects that person's perception of racial discrimination (Hall & Carter 2006; Jefferson & Caldwell, 2002). Further, the research suggests that racial identity mediated perceptions of racism, contribute in complex ways to psychological and/or physiological responses and functioning (Clark, Cobb, Hopkins, & Smith 2006; Sellers & Shelton, 2003; Pieterse & Carter, 2010). For example, Sellers and Shelton found that the more African Americans endorsed Black-oriented racial identities (e.g. oppressed minority) as measured by the Multidimensional Inventory of Black Identity (MIBI) (Sellers, Rowley, Chavous, Shelton, & Smith, 1997), the more they reported perceived racial discrimination; however positive feelings about their race appeared to buffer/protect these individuals from psychological distress. On the other hand, the more individuals endorsed non-Black oriented racial identities (e.g. humanist), the less perceived racial discrimination they reported. Sellers and Shelton suggest that these findings may be explained by consistency or inconsistency of racial discrimination with one's worldview. That is, African Americans with Black-oriented racial identities may expect to experience racial discrimination and be more prepared to deal with it, and thus be buffered from negative psychological and physiological consequences of such discrimination. On the

other hand, those with non-Black oriented racial identities are less likely to think that others will treat them negatively because of their race, and as such may experience psychological distress with associated physiological responses when faced with racial discrimination, as a result of their experience being inconsistent with their worldview (Sellers and Shelton, 2003).

Consistent with the above conceptualization by Sellers and Shelton (2003), it was found that Blacks who were high on a number of Black-oriented racial identities (oppressed minority; nationalist; centrality; and public-regard) as measured by the MIBI, failed to show significantly elevated cardiovascular reactivity to a racial profiling scene (Clark et al., 2006). In contrast, those who were high on non-Black oriented identities (assimilationist and humanist), and interestingly also high on one-Black oriented identity (private regard), showed significantly elevated cardiovascular reactivity to the racial profiling stressor. Therefore, it appears that complex relationships exist between various dimensions of racial identity and psychological and physiological responses to perceived racism. Such relationships were not examined in the present study, which may explain why we failed to find a main effect for perceived racism on cardiovascular reactivity. It is important for future studies to include racial identity status variables, in order to elucidate true associations between perceived racism and cardiovascular responses, and determine how such associations might contribute to risk for later development of hypertension in Black Canadians.

The seventh hypothesis stated that individuals with high versus low perceived racism scores will have less cardiovascular recovery after exposure to laboratory stressors. Analyses revealed a task x perceived racism interaction for SBP recovery, and a

task x sex x perceived racism interaction for DBP recovery from stress. Participants were found to have less SBP recovery on the math stressor task in the high perceived racism condition, while males, but not females, were found to have significantly less DBP recovery on the math task in the high perceived racism condition. As such, it appears that while high perceived racism has a detrimental effect on recovery from stress for both Black males and females (as seen in decreased SBP recovery for both), high perceived racism may be especially detrimental to cardiovascular recovery for males, (as seen in decreased DBP recovery for males but not for females).

The above mentioned gender differences in DBP recovery are consistent with epidemiological research that shows a higher risk for hypertension in young adult Black males compared to Black females, and is consistent with research implicating impairments in recovery of DBP as one of the earliest precursors for hypertension development (e.g. Schneider et al., 2003). Delays in recovery from stressors are thought to contribute to allostatic load, or the total time blood pressure is elevated, which is potentially damaging to the cardiovascular system and may contribute to later development of hypertension and cardiovascular disease (McEwen & Stellar, 1993). The results of this study suggest that Black males are potentially more susceptible to allostatic burden associated with negative effects of high perceived racism. Interestingly, this difference between genders was only found for the math task, which is a cognitive/performance-based task. It has been argued that men and women have different coping strategies in dealing with stress, shaped by socialization, and associated differential cognitive appraisals of stressors, which may determine physiological responding and cardiovascular risk (e.g. Frankenhaeuser, 1993). A speculative

explanation of the finding for decreased DBP recovery for men on the math task is that males possibly continued to think about/ruminate about their performance on the task during the recovery period, which may have contributed to sustained physiological activity and decreased cardiovascular recovery after the stressor ended. Along these lines, Brosschot, Pieper, & Thayer (2005) posit that perseverative cognition and associated physiological activation, is an important pathway through which stress may continue to exercise deleterious effects on the cardiovascular system.

It is noteworthy, that in the present study, measures of state affect collected immediately before and after each stressor task, indicated that participants were overall significantly nervous and anxious on the Math task, however measures of state affect (nor measures of cognitions) during/after the recovery period, were not obtained in this study. Instead, as per the research protocol, recovery measures for each task were collected in the 4-min period following administration of the after-task state affect measure and after the experimenter had left the testing room. It is likely that participants/males engaged in ruminative cognitions during this 4-min period of time after the math stressor task ended, which may have negatively impacted their cardiovascular recovery from that stressor.

A growing body of literature suggests that dwelling on the stressor experience, or rumination, may be an important factor contributing to the association between sustained cardiovascular arousal/delayed cardiovascular recovery and hypertension risk (Gerin, Davidson, Christenfeld, Goyal & Schwartz, 2006; Glynn et al., 2002; Key, Campbell, Bacon, & Gerin 2008). These researchers suggest that rumination may play a role in the latter association by prolonging the psychological and physiological arousal that accompanies stress exposure. Gerin et al. (2006) proposed a rumination arousal model

which posits that rumination sets into motion an intertwined set of process whereby cognition leads to negative emotions, and the emotion in turn produces elevated autonomic activation and blood pressure elevation. In particular, poor DBP recovery was found for those who ruminated after termination of a stressor, in the study by Key and colleagues (2008). It is thus reasonable to speculate that rumination processes contributed to the observed delay in DBP recovery for males observed in study.

Given that poor DBP recovery in the study was only observed for males in the high perceived racism condition, it can be further speculated that an interaction between high perceived racism, rumination, and gender, contributed to poor DBP recovery observed in males. Future studies are warranted to examine interactions between perceived racism and rumination, and differential effects on cardiovascular responses in males and females. Such studies would benefit from the inclusion of measures designed to capture ruminative cognitions during the recovery period, in order to establish how these may be linked to delayed cardiovascular recovery and risk for hypertension within the Black Canadian population.

Anger-In, Cardiovascular Reactivity, and Cardiovascular Recovery

The eighth and ninth hypotheses looked at associations between suppression of anger (anger-in) and cardiovascular responses. This follows from the several decades long association between anger and blood pressure, particularly evidence showing that anger suppression is associated with heightened cardiovascular reactivity (e.g. Jorgensen & Kolodziej, 2007; Mills & Dimsdale, 1993; Poole et al., 2006) and delayed cardiovascular recovery (e.g. Dorr et al., 2007; Lai & Linden, 1992). Hypotheses predicting similar relationships were proposed in the present study. The eighth hypothesis

stated that individuals with high compared to low anger-in would have greater cardiovascular reactivity to laboratory stress, while the ninth hypothesis stated that individuals with high compared to low anger-in would have less cardiovascular recovery from laboratory stress. These hypotheses were unexpectedly not confirmed as no main effect for anger-in on cardiovascular reactivity nor recovery, was found in this study.

It is possible that effects for anger-in on cardiovascular responses were not found in this study due to inherent limitations of self-report data on anger expression styles, and/or the possibility that trait measures of anger expression (as measured by the Spielberger Anger Expression Scale in this study) did not accurately represent anger expression as it occurs/varies in daily life situations for participants. Several researchers have challenged the conceptualization of anger expression as a trait versus a state that fluctuates according to situational characteristics or demands (e.g. Porter, Stone, & Schwartz, 1999; al'Absi & Bongard, 2006). Findings from these studies, as might be applicable to findings in the present research, are discussed below.

The study by Porter et al. (1999) compared trait and state measures of anger expression to examine associations between anger expression and situational variables among 100 college students. Students completed the state version of the Spielberger Anger Expression Scale, and they additionally completed state measures of anger expression in response to anger-provoking situations as they occurred in real life, over a 7-day period, collecting an average of 7.3 anger incidents per participant over the week. Significant associations were found between various situational variables and state anger expression. In particular, the participant's relationship to the target of anger, the location, and the presence of the target were all significant predictors of anger expression.

Specifically, participants used anger-out more often in situations where they knew and liked the other person (target) compared to when the anger was directed towards a stranger, and also when the location was private compared to public. Additionally, the power in the relationship was a significant predictor for anger-in, with lower anger-in demonstrated when the target of anger was a subordinate compared to a peer. As well, the presence of the target was a significant predictor for both anger-in and anger-out, with scores on both scales being higher when the target was present. In total, anger expression, measured as a trait with the Spielberger Anger Expression Scale, accounted for only 19% of the variance in the state measures collected across the week.

Researchers, al'Absi and Bongard (2006), also suggested that people express their anger differently in different domains, and that the Spielberger Anger Expression Scale, which measures anger-in and anger-out as stable personality traits, may not capture the variability in anger expression across situations in real life. Their research showed that people modified their anger expression according to situational demands/locations, with anger at home being associated with more overt expressions of anger while anger at work was associated with less overt expressions of anger. Furthermore, the research showed differences in patterns for males and females, with females reporting more anger-out at home then males, and males reporting more anger-out at work than females.

Overall, the research described above indicated that trait and state measures of anger expression differ significantly, and that situational factors play an important role in anger expression, which is not reliably captured in trait measures of anger expression. It is thus reasonable to speculate the Spielberger Anger Expression Scale used to measure anger-in and anger-out in this study, may not have captured state expressions of anger

expression in day-to-day response to stressors, which may have contributed to null findings for the relationship between anger-in and blood pressure responses to laboratory stressors in this study. Future research would benefit from addressing such methodological issues, in order to account for assessment of state/situational variability of anger expression that influences cardiovascular responses and risk for hypertension. As stated by Porter and colleagues (1999), "increasing the complexity of research designs in this area is likely to be the only way to achieve definitive answers concerning the relationship between anger expression and blood pressure".

Ambulatory Sleep and Wake Blood Pressure, and Blood Pressure Dipping

The tenth, eleventh, twelfth, and thirteenth hypotheses looked at factors including sodium, gender, perceived racism, and anger-in, which may contribute to ambulatory day/wake and night/sleep blood pressure, and nocturnal blood pressure dipping, in the Black population. This follows from research showing the prognostic value of ambulatory blood pressure in assessing risk for hypertension (Verdecchia, 1999); gender differences in blood pressure regulation (Oparil & Miller, 2005); findings that a large percentage of Black individuals are characterized by a non-dipping blood pressure profile (Pickering & Kario, 2001); the negative implications of higher blood pressure load of non-dippers on risk for cardiovascular events (Sherwood et al., 2002); the association between sodium intake and blood pressure dipping (Uzu et al. 1996); and the association between psychological/psychosocial stress and ambulatory blood pressure and non-dipping blood pressure (e.g. Steffen et al., 2003). The mixed findings in the present study for the influence of the above mentioned factors on ambulatory sleep and wake blood pressure, and non-dipping blood pressure, are discussed below.

Gender, Ambulatory Blood Pressure, and Heart Rate Measures. The tenth hypothesis, which proposed that males will have higher blood pressure compared to females during wake and sleep periods, and that females will have higher heart rate compared to males during these periods, was partially confirmed. No gender differences in wake or sleep blood pressure were found; however, as expected, females demonstrated significantly greater heart rate compared to males, in both the sleep and wake periods. The significantly greater number of females (n = 31) compared to males (n = 15) whose data were included in analyses for the ambulatory protocol in this study, likely enhanced the strong confirmatory findings for higher heart rate in females; on the other hand, the imbalance in sample sizes and the likelihood of differential variability, may have contributed to null findings for gender differences in ambulatory sleep and wake blood pressure measures in this study. Additionally, a multitude of unmeasured factors during ambulatory blood pressure monitoring, could have contributed to the lack of findings for the proposed gender differences in ambulatory sleep and wake blood pressure. Possible confounding factors include variability in length of wake period and sleep period (which were individually determined by participants), variability in exposure to daily stressors and in coping with stressors, variable activity levels during the wake period, etc. Future studies with larger sample sizes and a more balanced male/female ratio, as well as repeated collection of measurements through multiple 24-hour ambulatory blood pressure monitoring periods are needed, in order to elucidate true ambulatory blood pressure differences in Canadian Black males and females.

Sodium, Ambulatory Blood Pressure, and Non-Dipping. The eleventh hypothesis proposed that participants will have higher wake and sleep blood pressure

during the sodium-loaded diet compared to the regular diet condition, as well as less nocturnal blood pressure dipping in the sodium-loaded condition. No effect of diet was found for ambulatory blood pressure levels, in the wake nor sleep periods; however participants were found to have higher heart rate measures in the sleep period during the regular diet compared to the sodium-loaded diet condition. Such mixed results could be linked to differential sympathetic and parasympathetic influences on blood pressure and heart rate during the two diet periods. It is known that heart rate changes reflect the integration of the effects of both sympathetic and parasympathetic activity, while the latter is only indirectly involved in blood pressure control, especially during sleep (Sayk et al., 2007). Variable sympathetic/parasympathetic balance during the different diet periods could potentially be influenced by various uncontrolled factors, including sodium sensitivity, differential types/levels of activities engaged in, and/or differential exposure to stressors during each diet period. Such variability may have contributed to the mixed pattern of findings described above. Future studies with appropriate controls and larger sample sizes are needed to elucidate clearer patterns for the effects of sodium on ambulatory wake and sleep blood pressure.

With regards to the proposed relationship between sodium and nocturnal blood pressure dipping, although the hypothesis was not confirmed at a statistically significant level, there was a trend in the right direction for all measures of blood pressure dipping - that is, participants consistently showed higher levels of MAP, SBP and DBP (less blood pressure dipping) in the sodium-loaded diet compared to the regular diet condition. These findings are consistent with the pressure-natriuresis hypothesis, which states that under conditions of high sodium intake, increased blood pressure at night promotes sodium

excretion to compensate for insufficient sodium excretion during the day (Guyton, 1980). According to Guyton, the latter phenomenon is a means to reestablish sodium balance and homeostasis in the system. Blood pressure remaining high at night to facilitate excess sodium excretion, results in individuals showing a non-dipping blood pressure profile, i.e. their blood pressure does not show the typical 10% or more decrease at night. The sustained higher blood pressure during the night is thought to contribute to allostatic load, which is deleterious to the cardiovascular system (Sherwood et al., 2002). The capacity to excrete sodium during the daytime was recently confirmed as a significant determinant of nocturnal blood pressure and blood pressure dipping within the Black American population (Bankir et al., 2008). It can thus be reasonably speculated that greater non-dipping blood pressure during the high sodium diet as demonstrated for Black individuals in the present study, is potentially linked to insufficient sodium excretion during the day, and compensatory high blood pressure to promote sodium excretion at night.

Further to the above, in a comprehensive review article looking at blood pressure dipping and sodium excretion, Harshfield, Don, Kapuku, Zhu, and Hanevold (2009) suggest that the variation in circadian rhythms of sodium excretion may be due to stress-related sodium-retention during the day. These researchers found that individuals had diminished natriuretic response/retained sodium, in response to laboratory mental stress, with resultant increased blood volume and pressure. They further found that blood pressure remained elevated after the stressor ceased, until blood volume expansion diminished. It is possible that this stress-induced pattern of daytime sodium retention contributes to higher blood pressure (non-dipping) at night to facilitate sodium-excretion in order to regain system homeostasis. With regards to the present study, it is possible

that chronic stressors experienced during daily life of Black individuals contribute to imbalance in circadian rhythm of sodium-excretion while on a high sodium diet, and the resultant trend observed for greater non-dipping blood pressure during sodium-loading. This higher load of 24-hour elevated blood pressure, or allostatic burden, may contribute to the increased risk of hypertension found in the Black population.

As discussed above, a trend only (marginally significant findings) was found for the effect of sodium-loading on non-dipping blood pressure, in this study. A possible reason why these results failed to reach statistically significant levels might be due to the effects of confounding factors influencing the association between sodium and non-dipping blood pressure. Along these lines, research shows that the relationship between sodium and non-dipping is influenced by participants' sodium-sensitivity status. Uzu et al. (2006), found that those who were classified as sodium-resistant showed dipping on MAP on both a high and low sodium diet, whereas those who were classified as sodium-sensitive did not show nocturnal dipping on MAP in neither diet condition. This suggests that only sodium-sensitive patients are likely to manifest as non-dippers. Furthermore, Sachdeva and Weder (2006), posit that perhaps sodium-sensitive individuals respond with insufficient daytime sodium-excretion and resultant non-dipping blood pressure, because there may be differential limits to sodium excretion capacity between individuals that are revealed only when sodium intake increases. While the underlying mechanism remains unclear, sodium-sensitivity appears intricately linked to the non-dipping blood pressure phenomenon. As such, future research should focus on including measurement of sodium-sensitivity and interactions with other factors influencing blood pressure

dipping, in order to elucidate potentially important relationships contributing to risk for development of hypertension in the Black population.

Perceived Racism, Ambulatory Blood Pressure, and Non-Dipping. The twelfth hypothesis, which proposed that participants with high perceived racism scores would have higher wake and sleep blood pressure, and less blood pressure dipped at night, compared to those with low perceived racism scores, was not confirmed. This was unexpected, given that perceived racism is considered a chronic stressor in the lives of Black individuals in North America (e.g. Clark et al., 1999; Clark, 2004), and research shows a positive relationship between perceived racism and sleep and wake blood pressure (e.g. Hill et al., 2007), and a negative relationship between stress and blood pressure dipping (e.g. Thomas et al., 2004). Exploring the lack of support for the hypothesis proposing associations between perceived racism and ambulatory blood pressure/ blood pressure dipping is made difficult because of limited research to date in this area. Additionally, the few studies that have investigated these relationships have used widely variable samples, and have shown inconsistent results (reviewed below), with no clear patterns emerging to date.

In Steffen et al. (2003) study with Black American adults (25-33 years), it was found that perceived racism was associated with higher awake SBP and DBP ambulatory blood pressure but was not associated with sleep or non-dipping blood pressure. On the other hand, in a study with Black college students, Hill et al. (2007), found a positive association with perceived racism and higher DBP during both awake and sleep periods. Another study with a community sample of 357 American Blacks and Latinos (ages 24-65), found that perceived racism was associated with nocturnal SBP and DBP, and

predicted nocturnal non-dipping blood pressure, but was not associated with daytime blood pressure (Brondolo et al., 2008). The present study with a college sample of Black Canadian men and women, did not find any association between perceived racism and awake or asleep blood pressure, nor between perceived racism and percentage blood pressure dipped at night.

To the best of the author's knowledge, this is the first study to look at perceived racism and ambulatory blood pressure, and blood pressure dipping, in a Canadian Black population. Other studies, including those mentioned above, have all been conducted on American Black populations. It is possible that perceived racism differs in the extent of its presence, and/or the degree of its effects on psychological and physiological functioning linked to ambulatory blood pressure and the non-dipping phenomenon, amongst Canadian Blacks compared to American Blacks. As previously discussed, such differences could be linked to significant differences in the history of Blacks in Canada compared to the United States, and the differential influences/effects of such history on present day socio-cultural contexts for Blacks in each country. Though speculative, such factors could have contributed to the null findings for an association between perceived racism and blood pressure dipping in this study with Black Canadians. Replication of these results with larger and more diverse samples of Canadian Blacks is needed in order to assess the strength of such speculations.

In addition to potential differences between American and Canadian Blacks, various other sample characteristics stand out as possible contributors to the opposite findings for the relationship between perceived racism and non-dipping blood pressure in the Brondolo et al. (2008) study (discussed above) and the present research study. One

significant difference between the two studies is that the Brondolo et al. study used a community sample with individuals spanning a wide age range (24 to 65; mean age 40) compared to the present study that used a college sample with a smaller and generally younger age range (18 to 30; mean age 22). As well, the Brondolo et al. sample included both normotensive and unmedicated hypertensives, while the present study included only normotensive individuals. As such, it is reasonable to speculate that age-related or health-related variability in blood pressure regulation, potentially contributed to inconsistent findings between the two discussed studies. Moreover, the two studies varied widely in sample sizes: The Brondolo et al. study included 245 participants in the analyses for effects of perceived racism on nocturnal dipping, while only 41 participants were included in the relevant analyses in this study. The substantially greater power of the analyses in the Brondolo et al. study likely allowed for effects of perceived racism on non-dipping blood pressure to emerge, while the limited power of the present study was possibly insufficient for significant findings to be detected. It is possible that with a larger sample size and greater power, significant findings for effects of perceived racism on ambulatory sleep and wake blood pressure, and on percentage blood pressure dipped at night, would have emerged in the present study.

Given that perceived racism is considered a chronic stressor in the lives of Blacks, in making sense of the findings in this study it may be helpful to draw upon findings from related research with other types/measures of stressors associated with non-dipping blood pressure. One related study by Tomfohr et al. (2010) utilized the Everyday Discrimination Scale (Williams, Yu, Jackson, & Anderson, 1997) to measure discrimination in a mixed sample of Black and White Americans, and found an

association between higher report of everyday discrimination and less DBP and SBP dipping. Although the above study did not specifically look at racial discrimination, it is reasonable to infer that dealing with everyday discrimination (regardless of type) might be similar to dealing with the chronic stress of perceived racism, and a such, potentially has similar effects on non-dipping blood pressure. The following is a discussion of findings in the Tomfohr et al. study, which could speculatively help to explain the lack of findings for a significant effect of perceived racism on non-dipping blood pressure in the present study

An interesting observation in the study by Tomfohr et al. (2010) was that Blacks had a trend towards lower socio-economic-status (SES) and demonstrated less blood pressure dipping than Whites, suggesting that SES potentially moderated the effects between discrimination and non-dipping blood pressure in this study. Other studies have also found that SES is associated with non-dipping blood nocturnal blood pressure (Campbell, Key, Ireland, Bacon, & Ditto, 2008; Stepnowsky, Nelesen, DeJardin, & Dimsdale, 2004). Stepnowsky and colleagues found that SES explained a significant amount of variance in nocturnal blood pressure dipping after controlling for various life-style and demographic factors. Specifically, the lower the SES, the less blood pressure dipping observed at night. These authors suggest that being in a lower SES likely increases exposure to daily stressors associated with fewer economic and social resources. Stressors associated with lower SES may potentially contribute in additive or interactive ways to stress associated with perceived racism, thereby influencing effects on blood pressure dipping at night.

With regards to the present study, it is noteworthy that while all participants in the present study were university students, therefore of similar educational level (one measure of current SES), other unmeasured aspects of SES may have contributed to the findings in this study. In particular, research demonstrates that early/childhood SES is associated with adult blood pressure dipping (e.g. Campbell et al., 2008). Campbell and colleagues evaluated early SES in 174 healthy undergraduate university students by assessing the highest level of education completed by participants' parents. They found that after controlling for lifestyle factors, daytime blood pressure, BMI, alcohol use, smoking, or current SES, lower levels of childhood SES was associated with less SBP and DBP dipping at night. The authors conclude that "irrespective of adult achievement, childhood SES may have long lasting health implications" (p. 276). Childhood and other indices of SES (except for education level) were not assessed in the present study. Given potential additive or interactive relationships between SES and chronic stress of perceived racism and effects on non-dipping blood pressure, important associations may have been missed in the present study where a linear/direct relationship between perceived racism and non-dipping blood pressure was investigated and found not to be significant. It would be important for future studies to look at the moderating effects of SES, as measured with multiple indices, on the relationship between perceived racism and non-dipping blood pressure, in order to elucidate important associations that may contribute to the high risk for hypertension among Canadian Blacks.

Anger-in, Ambulatory Blood Pressure, and Non-Dipping. The thirteenth hypothesis, which stated that participants with high anger-in would have higher ambulatory sleep and wake blood pressure, and less percentage blood pressure dipped at

night, was not confirmed; however an anger-out x gender interaction (significant for MAP and SBP and marginally significant for DBP) was found for percentage blood pressure dipped at night. Post hoc analyses found that males compared to females, had significantly greater percentage blood pressure dipped at night for MAP, SBP, and DBP, in the high anger-out condition compared to the low anger-out condition. Potential explanations for these results are discussed below.

While most research have reported on the relationship between high anger-in and risk for hypertension, some researchers have shown a link between high anger-out and greater cardiovascular disease incidence (Mendes de Leon, 1992; Siegman, Dembroski, & Ringel, 1987), and greater risk for hypertension (Everson, Goldberg, Kaplan, Julkunen, Salonen, 1998; Harburg, Gleiberman, Russsell & Cooper, 1991). Given the strong predictive relationship between non-dipping blood pressure and cardiovascular morbidity and mortality (Fagard et al., 1997; Staessen et al., 1999), it can reasonably be speculated that high anger-out as a risk factor for hypertension would likely be associated with greater non-dipping blood pressure. However, the opposite was found for males in this study, i.e. high anger-out was found to be related to greater dipping in nocturnal blood pressure for males. It is possible that the latter association was a spurious finding based on small cell sizes for males in the relevant MANCOVA and ANCOVA analyses conducted in this study. Specifically, for males in the low anger-out condition, cell size was, n = 6, while for males in the high anger-out condition, cell size was, n = 8. Insufficient power associated with such a small sample size likely resulted in chance findings for a relationship between anger-out and males in this study. Future studies

would benefit from larger sample sizes to elucidate true associations between anger expression and nocturnal blood pressure dipping in the Black population.

Another possible factor contributing to the lack of findings for an effect of anger-in on ambulatory sleep and wake measures and non-dipping blood pressure in the present study, may involve methodological limitations of too narrowly classifying anger coping into anger-in and anger-out, as suggested in research by Linden and colleagues (2008). Linden and colleagues suggest that non-dipping blood pressure may be more influenced by anger coping styles or preferences, which are more diverse in nature. These researchers identify six anger coping preferences, as measured by the Behavioural Anger Response Questionnaire (BARQ), including Direct Anger-out, Avoidance, Diffusion, Support-seeking, Rumination, and Assertion (Linden et al., 2003). Linden and colleagues (2008) found that anger diffusion coping style was a positive predictor of blood pressure dipping, suggesting that the way one *processes* anger (as opposed to mere anger-in or anger-out) is significant for effects on blood pressure. The anger diffusion response style is characterized by acknowledgement of angry feelings and then a proactive way of dealing with/processing the feelings during the daytime. It is possible that anger-in/anger-out, as measured by the Spielberger Anger Expression (AX) Scale (Spielberger et al., 1985) used in this study, did not adequately capture anger coping responses that possibly mitigated levels of blood pressure dipping at night, which could have contributed to the lack of significant findings for a main effect of anger-in on percentage blood pressure dipped in this study.

In statistical comparisons of the BARQ with the AX scale, Linden et al. (2003) acknowledges strong overlap between the BARQ ‘Direct Anger-Out’ with the AX

'Anger-Out' construct, and some overlap between the BARQ 'Rumination' and the AX 'Anger-in' subscales. However, the results indicated that the other subscales, including the 'Diffusion' subscale of the BARQ (which, as discussed above, was found to be a positive predictor of blood pressure dipping in the Linden et al., 2008 study), are largely original. It is noteworthy that in addition to Linden and colleagues, other researcher groups have also proposed that the anger-in/anger-out dichotomy may be too narrow, and have supported exploring a broader range of anger scales to capture the dimensionality of anger expression styles (e.g. Riley & Trieber, 1989; Miller, Jenkins, Kaplan, & Solonen, 1995). Future studies would benefit from including measures of comprehensive anger coping styles, such as the BARQ, in order to detect important links between anger and ambulatory blood pressure/non-dipping blood pressure, and associated link with risk for development of future hypertension.

Limitations and Directions for Future Research

A major limitation of this study was the small sample size and related power issues, which may have significantly affected the results obtained. Fifty-three individuals participated in this study over a four-year period, each undergoing an extensive 21-day protocol including dietary manipulation, testing in several domains, and measurement of blood pressure in both laboratory and ambulatory/naturalistic settings. Not surprising for such an extensive research protocol, data collection for each participant was compromised by variable compliance issues, resulting in missing data and reduced sample sizes in different areas of data collection. First, 9 participants did not comply with the sodium diet. This resulted in a reduced sample size from 53 to 44 participants, whose data were used in analyses to determine effects of sodium on blood pressure responses to

laboratory stressors. Secondly, non-compliance in properly wearing the equipment during the 24-hour ambulatory blood pressure monitoring period, and/or the occasional malfunctioning of the equipment during that time, resulted in further reduction from 44 to 34 participants, whose data were used in analyses including sodium in the ambulatory protocol. These unanticipated reductions in sample size and related statistical power issues, potentially compromised the results of this study, and likely contributed to lack of confirmation for several of the study's hypotheses. Importantly however, these limitations were mitigated to some extent by the repeated measures design which allows for a more powerful test with fewer participants.

Repeated measures MANCOVA analyses were employed to investigate associations between multiple independent variables and multiple dependent variables, while controlling for covariates that may influence the outcome. MANCOVAs were determined to be the most advantageous method of analyses for this complex data set, which would allow for investigation of effects of sodium diet at two discrete time periods. However, there are some potential costs to this method linked to limitations in the recoding of continuous variables to create categorical variables necessary for the MANCOVA analyses. Specifically, continuous psychosocial measures of anger-in and perceived racism were dichotomized by splitting at the median to create discrete high and low anger-in and perceived racism groups. It is recognized that one disadvantage of creating such dichotomous variables is that there is a risk of loss of information about individual differences. On one hand, this loss of information may have contributed to non-confirmation of several hypotheses including anger-in or perceived racism in this study. On the other hand, it is notable that when exploratory regression analyses were run

(for each diet condition separately), lack of finding for a main effect for anger-in or perceived racism, was still the result. As such, we can reasonably speculate that loss of individual differences due to dichotomizing continuous variables, were likely not the reason for limited findings for effects of anger-in and perceived racism observed in this study.

Future studies with larger sample sizes are needed to investigate complex interactions between biopsychosocial factors including sodium-sensitivity, perceived racism, and anger-expression/coping, on cardiovascular reactivity and recovery, ambulatory sleep and wake blood pressure, and nocturnal blood pressure dipping, all of which have been associated with the high risk of developing hypertension in the Black population. Additionally, studies are needed to investigate the mediating/moderating effects of factors including socio-economic status (e.g. Campbell et al., 2008), racial identity status (e.g. Clark et al., 2006; Pieterse & Carter, 2010, and rumination (e.g. Key et al., 2008), among others, in order to elucidate pathways by which risk factors may influence the development of hypertension and cardiovascular diseases.

Unassesed ruminative cognitions, in particular, potentially contributed to findings, or lack of, for effects of predictor variables on cardiovascular recovery from laboratory stressors in the present study. Rumination as a mediator/moderator of cardiovascular recovery from stress has become an important focus in the research literature in recent years (e.g. Brosschot & Thayer, 2003; Gerin et al., 2006). It is thought that rumination contributes to hypertension risk by prolonging the psychological and physiological arousal that accompanies stress. Importantly, it has been shown that rumination is associated with higher sympathetic nervous system activity (Ottaviani & Shapiro, 2011),

which is linked to development of hypertension and cardiovascular diseases. More studies are needed in the future to investigate pathways by which rumination contributes to delayed cardiovascular recovery and risk for development of hypertension, which likely includes interaction with other factors. For example, gender differences in rumination have been found, with rumination about interpersonal events being more common in women (Mezulis, Abramson, & Hyde, 2002), and anger rumination likely being greater in men (Rusting & Nolen-Hoeksema, 1998). Future studies investigating gender differences in stress appraisal, coping, rumination, and cardiovascular recovery are needed to shed light on how these factors may contribute to differential risk/prevalence for hypertension and cardiovascular diseases in Black men and women.

One relevant but uncontrolled factor in the present study is sodium sensitivity. This is a limitation given that sodium sensitivity has been shown to influence cardiovascular reactivity to acute stressors (e.g. Deter, Buchholz, Schorr, Mathiak, & Sharma, 2001) as well as ambulatory blood pressure measures, particularly nocturnal non-dipping blood pressure (e.g. Damasceno et al., 2000). Sodium sensitivity, which is generally defined as a change in blood pressure in response to changes in salt and water homeostasis (Svetkey, McKeown, & Wilson, 1996), requires measurement of blood pressure following two separate manipulations of diet, i.e. a high sodium diet and a low sodium diet, each lasting at least a week in duration. Such a protocol is extensive and requires costly resources, which were unfortunately not available for the present study. However, given the importance of sodium in both the etiology as well as in treatment of hypertension, a methodological decision was made to include sodium as an independent variable, with conditions of a sodium-loaded diet (255 mmol/24 hr) versus the regular

diet, for each participant. In addition to sample size and statistical power issues, uncontrolled sodium-sensitivity may have contributed to the absence of replication of significant links previously found between predictor variables in this study and blood pressure responses.

To the best of the author's knowledge, no prior study has looked at effects of sodium-loading/sodium-sensitivity on cardiovascular recovery from laboratory stressors. This is surprising, given that both sodium-sensitivity and cardiovascular recovery are strongly linked to risk for development of hypertension. The research field, in the recent past, has experienced a surge in the number of studies investigating delayed cardiovascular recovery as a risk factor for hypertension. This focus on cardiovascular recovery is fast catching up to the prior research focus on cardiovascular reactivity. At the same time however, review of the literature suggests that there has been a decrease in focus in recent years, on studies investigating sodium as a risk factor for hypertension, particularly in interaction with other risk factors. Future studies are needed to address this gap in the research literature, to elucidate how sodium may interact with other risk factors to influence cardiovascular recovery from laboratory stressors, and how this may be linked to risk for development of hypertension.

Another possible limitation that may have contributed to the limited findings for association between anger expression and blood pressure, might relate to the conceptualization of anger expression as anger-in versus anger-out as measured by the Anger Expression Scale (Spielberger et al., 1985), used in this study. Linden et al. (2008) posits that the latter conceptualization may be too narrow, leaving the influence of diverse anger coping styles unaccounted for. They suggest that using a more

comprehensive measure of anger coping, such as the Behavioural Anger Response Questionnaire (BARQ) (Linden et al., 2003), might be advantageous in detecting important relationships moderating anger and blood pressure. Notably, the BARQ measures coping with anger by Direct Anger-out, Avoidance, Diffusion, Support-seeking, Rumination, and Assertion (Linden et al., 2003). Future studies are warranted to explore such diverse styles of anger coping that may mediate blood pressure responses and risk for development of hypertension, within the Black Canadian population.

With regards to research on non-dipping blood pressure as a risk factor for hypertension and cardiovascular diseases, it is increasingly being recognized that a significant amount of variance exists in dipping status from one night to the next (Parati & Staesssen, 2007). Such variability might impair attempts to elucidate important relationships between variables in studies that only measure blood pressure for one 24-hr ambulatory blood pressure session. Consistent with the latter, Tomfohr et al. (2010) showed that while data from a single 24-hr ambulatory monitoring session had limited power to detect a relationship between discrimination and nocturnal dipping, data collected on the second 24-hr session enhanced their ability to find a significant effect. Tomfohr and colleagues posit that variations on the first night of ambulatory blood pressure monitoring might be due to sleep disruptions, which may disrupt normal dipping pattern of individuals; on the other hand, potential habituation to the blood pressure monitor on the second night may result in less disrupted sleep and detection of more accurate blood pressure dipping patterns. As such, future studies investigating associations between psychosocial variables and non-dipping blood pressure may benefit from multiple consecutive sessions of 24-hr ambulatory blood pressure monitoring to

increase reliability of 24-hr ambulatory measures and determination of non-dipping status.

Notwithstanding the above discussed limitations, the present study, contributes to addressing the gap in research literature to date for factors which may contribute to the high risk for hypertension within the Canadian Black population, and findings of this study suggests important areas for future research to build on. A notable strength of this research study is its repeated measures/within subject design, which is known to decrease the effects of variation between participants and the results. Additionally, this study allowed for examination of individual differences in identifying factors that may put some Blacks at risk for hypertension compared to others in this high risk population, by limiting inclusion to young adult Black individuals who were born or raised in Canada from a young age. Being born or raised in Canada helps to control for socio-cultural-political climates that may influence psychosocial development of individuals, which helps to control for variability that may influence relationships between psychosocial factors and blood pressure responses as examined in this study. Another strength of this study is the inclusion of an ecologically valid stressor in the laboratory protocol, the Discrimination Recall task, which replicates a naturally occurring social stressor in the lives of Black individuals. Additionally, this study looked at multiple risk factors for hypertension, including sodium, cardiovascular reactivity, cardiovascular recovery, gender, anger-in, perceived racism, ambulatory sleep and wake blood pressure, and blood pressure dipping, combinations of which have been previously looked at in separate studies, but the total of which have not been previously investigated in a single study. Such an investigation increases the chances of identifying combinations of potential risk

factors which may act together in contributing to the complex multifactorial etiology underlying development of essential hypertension in the high risk Black population, and more specifically, in the Canadian Black population.

A primary importance of this study is that it is one of few that have investigated risk factors for hypertension within a Canadian Black population, and it is the first to investigate the relationship between perceived racism and cardiovascular responses in a Canadian context. Most previous studies have been conducted in the United States with American Black populations. It is reasonable to speculate that the significant differences in Black history of the US and Canada (previously discussed) and influences on present day socio-cultural environments of American and Canadian Blacks, possibly contribute to differences in effects of psychological and psychosocial factors on physiological functioning of individuals in these populations. It is possible that the absence of strong findings for effects of perceived racism and anger-in in the present study on Black Canadians, compared to studies done on Black Americans, is reflective of differences in the degree of chronic environmental stressors and/or differences in perception of, or coping with, such stressors. Further research exploring hypertension risk factors among Canadian Blacks, for age ranges across the lifespan, are needed to shed light on true associations between psychosocial stressors and risk for hypertension in this population.

Summary

The present study contributes to the sparse literature on risk factors for developing hypertension in the Canadian Black population. It is in many ways, a preliminary investigation of a unique combination of salient risk factors implicated in the development of hypertension, in this high risk population. Unexpectedly, no significant

findings for the effect of sodium on cardiovascular reactivity to, or recovery from, laboratory stress, were found in this study. However, marginally significant findings implicate a complex relationship between gender and sodium in cardiovascular recovery from stress. Additionally, sodium is indicated as an important contributor to non-dipping blood pressure amongst Black men and women. None-the-less, these findings for effects of sodium should be interpreted with caution, given that sodium-sensitivity was not controlled for in this study. Consistent with existing literature, this study found significant gender differences in cardiovascular responses, especially for cardiovascular reactivity to stress. Findings implicate a vascular pattern of reactivity for males, and a primarily myocardial pattern of reactivity for females. These reactivity differences may play an important role in differential risk for hypertension and cardiovascular diseases in Black men and women. Unexpectedly, gender differences in blood pressure levels were not found for ambulatory sleep or wake measures, contrary to what had been proposed. It is possible that confounding factors in the naturalistic environment contributed to these null findings. Albeit, strong confirmatory findings for higher heart rate in females compared to males was found, for both wake and sleep ambulatory periods.

Mixed findings emerged with the investigation of effects of anger-in and perceived racism, on cardiovascular reactivity, recovery, ambulatory sleep and wake measures, and blood pressure dipping. Unexpectedly, no effect for anger-in on any of the outcome measures was found; however, a relationship between anger-out, gender, and nocturnal blood pressure dipping, is suggested. The null findings for effect of anger-in may possibly be due to methodological limitations, including conceptualization of anger expression/coping styles, issues around measurement of anger expression (state versus

trait), and/or unmeasured mediating or moderating factors influencing anger coping, which may have prevented detection of important associations. No significant findings for the effect of perceived racism on nocturnal blood pressure dipping emerged; however perceived racism was found to interact with gender and stressor task in determining cardiovascular recovery from laboratory stressors. Specifically, it was found that males with high perceived racism, compared to similar females, have an increased propensity for delayed cardiovascular recovery from cognitive stressors. These are important findings because they suggest gender-specific pathways by which perceived racism may contribute to hypertension risk.

The overall findings of this study provide important directions for future research on risk factors for the development of hypertension in the high risk Black population. Future studies would benefit from larger samples sizes, with appropriate designs and methodologies to investigate complex processes by which biopsychosocial moderating/ mediating factors may complicate the relationship between predictor variables and cardiovascular responses. In particular, understanding how racism and discrimination interact with psychological and physiological aspects of the individual will help in the development and implementation of primary prevention and intervention strategies, in efforts to eliminate the troubling racial disparities in prevalence for hypertension and cardiovascular diseases, and decrease the high risk for these diseases in the North American Black population. Future studies are needed to clarify how these factors may contribute to high risk for hypertension amongst the Canadian Black population.

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Appendix A.

General Health Survey

Please answer the following questions to the best of your ability. All information will be kept confidential.

Date: _____

1. Name: _____ (Please print)

   Address: _____
   Street City Province Postal Code

   Telephone number (s): _____(home) _____(work)

   E-mail Address: _____

2. Date of Birth: _____ / _____ / _____ Age: _____
   Month Day Year

3. Sex: Male _____ Female _____

4. Marital Status: Single, never married _____ Living with partner _____ Married _____

5. Living Arrangements: Alone _____ Roomate (s) _____ Family _____

6. Race: White _____ Black _____ Asian _____ Hispanic _____

   Native Indian _____ Other _____ (specify)

7. Where were you born: City _____Province _____ Country _____

8. Nationality: Canadian _____ Other _____ (specify)

9. How many siblings do you have: Brothers _____ Sisters _____

   How many half siblings do you have: Brothers _____ Sisters _____

10. What is your birth order? Only child _____ Oldest _____ Middle _____ Youngest _____

12. Weight: _____ (lbs.) Height: _____ (inches)

13. Please describe any major illnesses, health problems or hospitalizations you have had during your adult life: _____

14. Has a doctor ever told you that your blood pressure was high? Yes ____ No ____

    If yes, please give the date it was found to be high: _____ / _____ / _____
    Month Day Year

15. Have you ever been prescribed medication to lower your blood pressure? Yes ___ No ___

    If yes, specify drug name: _____

    Are you currently taking such medications? Yes ____ No ____

16. When was the last time you were examined by a physician or other health care provider?

    Within the last month _____ Within the last 6 months _____

    Within the last year _____ More than a year ago _____

17. Have you ever been told that you have had: (Check yes or no)

	Yes	No
Chest Pains or Angina		
High Blood pressure		
Coronary artery disease		
Rheumatic heart disease		
Hardening of arteries (arteriosclerosis)		
Heart attack		
Stroke		
Any other heart or circulatory problems		
Kidney disease (other than stones)		
Diabetes (high blood sugar)		
Respiratory disorder		
Gastrointestinal disorder		
Ulcer		
Nervous or mental disorder		
Asthma		
Allergies		
History of high/marginally high cholesterol		
Reproductive system disease or disorder		
Cancer		
Arthritis or joint condition		

Certain health problems tend to run in families. Please answer these questions about your biological parents.

18. Is your father: Living _____ Deceased _____
Present age _____ Age at Death _____

19. Is your mother: Living _____ Deceased _____
Present age _____ Age at Death _____

20. Have your father and/or mother ever had a history of (Please check yes or no)

	Father		Mother	
	Yes	No	Yes	No
Chest Pains or Angina				
High Blood pressure				
Coronary artery disease				
Rheumatic heart disease				
Hardening of arteries (arteriosclerosis)				
Heart attack				
Stroke				
Any other heart or circulatory problems				
Kidney disease (other than stones)				
Diabetes (high blood sugar)				
Respiratory disorder				
Gastrointestinal disorder				
Ulcer				
Nervous or mental disorder				
Asthma				
Allergies				
History of high/marginally high cholesterol				
Reproductive system disease or disorder				
Cancer				
Arthritis or joint condition				

21. How many cups of caffeinated beverages (coffee, tea, cola) do you typically consume in a day? None ____ 1 cup ____ 2-3 cups ____ 4-5 cups ____ 6 or more cups ____

22. How much alcohol (beer, wine and/or hard liquor) do you consume in a typical day?

None ____ Occasional drink ____ 1-2 drinks ____ 3-4 drinks ____ 5 or more ____

23. Please read the following 7 statements that describe different patterns of alcohol use:

1. I have several drinks a day, every day.
2. I have several drinks on weekends but one or less than one on weekdays.
3. I have about one drink a day.
4. I drink about one drink a week.
5. I drink only on special occasions.
6. I never or almost never drink.
7. I used to drink but I quit.

Which of these 7 statements best describe your use of BEER? _____

Which of these 7 statements best describe your use of WINE? _____

Which of these 7 statements best describe your use of HARD LIQUOR? _____

24. Smoking Status: 1. Current Smoker _____

2. Non-smoker (never smoked) _____

3. Past smoker (smoked and quit) _____

25. <u>If you are a current smoker</u>, approximately how many cigarettes do you smoke in an average day? _____

a. What age did you start smoking cigarettes? _____

b. How many times have you tried to quit? _____

c. How many total years have your smoked, subtracting out years you may have quit in between? _____

26. <u>If you are a past smoker</u>, approximately how many cigarettes did you smoke in an average day? _____

a. What age did you start smoking?

b. What age did you quit smoking?

c. How many total years did you smoke, subtracting out the years you had quit in between? _____

27. Do you currently use any of the following drugs (see question 29) on a regular basis?

    Yes _____ No _____

    If yes, how often? _____ For how long? _____ At what age did you start? _____

28. Have you used drugs on a regular basis in the past? Yes _____ No _____

    If yes, how often? _____ At what age did you start? _____

    For how long? _____ At what age did you stop? _____

29. Please check off drug (s) used:

    Cannabis (Marijuana, Hashish, Hash oil) _____

    LSD (Lysergic acid) _____

    PCP (Phencyclidine) _____

    Mescaline or Peyote _____

    Stimulants (e.g. Cocaine, Crack, Amphetamines) _____

    Narcotic Analgesics (e.g. Opium, Codeine, Morphine, Heroin, Methadone, Demerol, Percodan)_____

    Barbiturates (e.g. Secobarbital) _____

    Sedatives Hypnotics (e.g. Valium, Serar, Ativan, Quaalude) _____

    Others _____

30. The following questions are about the foods you usually eat, your eating habits and other dietary practicies. Please circle the appropriate number.

		Never	Always
a.	When you eat meat (roast, steak), do you Eat the fat on the meat?	1…..2…..3…..4…..5…..6…..7	
b.	When you eat chicken, do you eat the skin?	1…..2…..3…..4…..5…..6…..7	
c.	Do you add pepper to food at the table?	1…..2…..3…..4…..5…..6…..7	
d.	Do you add salt to food at the table?	1…..2…..3…..4…..5…..6…..7	
e.	Do you add sugar to food at the table?	1…..2…..3…..4…..5…..6…..7	

31. How often do you eat each of the following foods?

	Regularly	Occasionally	Never or Almost Never
Diary Products			
Eggs			
Fish			
Red Meats (e.g. beef)			
Chicken			
Fruits			
Vegetables			
Salty Snacks (e.g. chips)			
Desserts			
Fast Food (e.g. McDonalds)			
Whole Grains (e.g. breads & cereals)			

32. When you eat dairy products (e.g. milk, cheese, yogurt, frozen yogurt) do you usually choose: Regular _____ Low Fat (Skim, 2%) _____ Non Fat _____ Do not eat them _____

33. Have you ever been on a diet? Yes _____ No _____ If yes, how many times? _____

34. When shopping for food, do you read labels? Yes _____ No _____

35. Are you conscientious of what you eat (e.g. try to eat healthy)? Yes _____ No _____

36. How would you rate your current health?

Excellent _____ Good _____ Fair _____ Poor _____

Appendix B.

INFORMED CONSENT FORM

Hypertension: Physical and Psychological Moderators in the Canadian Black Population

Research Study Conducted at Concordia University
Department of Psychology on Behalf of Dr. Sydney Miller

We would like you to participate in a study investigating moderators of risk factors for hypertension. In this study, changes in blood pressure and heart rate during normal daily activities will be recorded with an ambulatory (portable) blood pressure monitor, and during laboratory sessions with similar equipment. These procedures have been widely used in other research, and are completely safe and non-invasive. No needles or medication are involved and you will be asked to consume completely natural dietary sodium supplements. As is consistent with similar research, women who are pregnant will not be tested. Females will undergo a urine based pregnancy screen prior to beginning the test protocol.

Your participation in the study will require you to undergo a 30-minute general medical exam in which physical and mental health will be assessed, to come to the laboratory for 3 sessions lasting approximately 30 minutes, and 2 sessions lasting approximately 90 minutes. During the first 30-minute session you will be asked to read and sign this consent form, given instructions and provided a dietary protocol which may include natural sodium supplements. You will also be asked to take with you and complete a package of questionnaires. Included in these questionnaires is one examining your experiences with racism. At the subsequent two 30-minute sessions, you will be fitted with a blood pressure cuff similar to those used in a doctor's office. Two blood pressure readings will be taken and you will then be asked to relax for a 10-minute period. Following the rest period, three more blood pressure readings will be taken. During these 30-minute sessions you will be fitted with an ambulatory blood pressure monitor. This monitor will be programmed to take intermittent blood pressure readings for a 24-hour period while you go about your regular activities (except bathing, swimming, or contact sports). Although you may experience some minor temporary discomfort as blood pressure readings are taken, the procedure is completely harmless. During waking hours you will be required to complete and activity diary immediately after each blood pressure reading is taken. In addition, we ask that you complete a few short questionnaires before retiring for the night. During the first of these last two 30-minutes sessions you will be given further instructions and provided a dietary protocol, which may include natural sodium supplements. Prior to each of the two sessions during which you will be

fitted with the ambulatory blood pressure monitor, you may not consume coffee, cigarettes, alcohol, or recreational drugs beginning the evening (6 p.m.) prior to presenting at the laboratory, and must continue to abstain until the end of that session's laboratory testing (approximately).

As part of the dietary protocol you will follow a 10-day routine diet and a 10-day high sodium diet (detailed instructions will be provided). On the last day of both the routine and high sodium diet you will participate in the second 30-minute session (described above), be fitted with the ambulatory blood pressure unit and follow a 24-hour blood pressure monitoring (described above) and complete a 12-hour overnight urine collection. After this 24-hour period you will come back to the laboratory to return the blood pressure unit and participate in the first 90-minute session. In this 90-minute session, physiological changes such as increases in heart rate and blood pressure will occur. These changes will only be temporary, however, returning to normal immediately after the experiment and causing no ill effects. You will be equipped with physiological measurement devices that consist of transducers placed on the skin and are safe, painless, and non-invasive. The measures include blood pressure and heart rate as well as several other heart related measures. You will be asked to sit quietly for 15 minutes and physiological measurement will begin during the last 5 minutes. Following this you will be asked to participate in three tasks.

These will include:

(i) the placement of a cold gel pack chilled to a temperature of 4° Celsius on your forehead for two 2-minute periods separate by one minute. This procedure can be moderately painful but is harmless and the discomfort is no greater than what you would experience during a cold winter day.

(ii) a 3-minute interview where you will be asked to recall an event in your past where you felt you were discriminated against.

(iii) a 3-minute computerized math task.

After the 90-minute session you will begin the second diet period. On the last day of this diet period you will participate in the second 30-minute laboratory session (described above), and complete the final 24-hour ambulatory blood pressure monitoring and 24-hour urine collection. At the end of this 24-hour period you will return the blood pressure equipment to the laboratory and participate in the final 90-minute laboratory session (as described above).

Prior to leaving with the ambulatory blood pressure equipment and other materials, you will be asked to leave an identification card (e.g. social insurance card, student identification card) with the research assistant. Your identification card will be kept in a secure place at the

laboratory until it is returned to you the next morning with the return of the monitoring equipment and completed diary.

Moderators of blood pressure factors may include a family history of high blood pressure, high cholesterol level, stroke, and or other cardiovascular problems. In order to correct and verify this information we need to contact your parents for this information prior to your participation in this study. Please note that no information about your participation in the current study will be transmitted to your parents other than the fact that you have agreed to participate in the research project.

You are free to withdraw from the experiment at any time.

Participating in this study has no direct benefit for the participant or their family.

You will be paid $100.00 for your participation when complied with the dietary protocols, provided the urine samples, returned the completed questionnaires and diaries, monitoring equipment, and participated in the final 90-minute laboratory session. Failure to comply with the requirements of the study described above will result in a reduction in the amount of financial compensation you receive.

All information we obtain about you is completely confidential and will not be seen by anyone who is not a member of the research team. Ultimately, all data will be coded using participant numbers rather than names.

Once you have carefully studied and understood this form, you may sign it in indication of your free consent and agreement to participate in the study.

In summary, the steps you will have to complete if you participate in the research are as follows:

1. Health questionnaire for screening purposes.
2. Provide contact information and consent to contact your parent(s).
3. Participate in a medical examination.
4. Undergo a urine-based pregnancy screening test (females only).
5. Receive instructions and dietary materials and complete a questionnaire package (at home).
6. Follow a 10-day (high sodium diet) and a 10-day (regular diet) dietary protocol.
7. 30-minute laboratory sessions including resting blood pressure readings and instrumentation with a portable blood pressure unit for 24-hour monitoring.
8. 24-hour monitoring including activity log.

9. 12-hour overnight urine collection.
10. 90-minute laboratory stress testing session that includes the two 2-minute cold pressor tests, a 3-minute discrimination-recall interview & a 3-minute computerized math task. Complete several questionnaires.
11. Follow the second dietary protocol (regular or high sodium diet).
12. 30-minute laboratory session including resting blood pressure readings and instrumentation with a portable blood pressure unit for 24-hour monitoring.
13. 24-hour monitoring including activity log.
14. 12-hour overnight urine collection.
15. 90-minute laboratory stress testing session as described in step 10 with similar questionnaires.
16. Receive $100.00 remuneration as stipulated above.

Parent Information:

Mother's Name	_____	Father's Name	_____
Telephone #	_____	Telephone #	_____
Address	_____	Address	_____
	_____		_____
	_____		_____
E-mail	_____	E-mail	_____

Research Contacts:

Laboratory SP-252.00
848-2424 Ext. 2846

Regarding your rights as a subject: 848-2424 Ext. 4964

Concordia University Ombudsman

Researcher:

Sydney Miller, Ph.D.

NAME: (PLEASE PRINT) _____

SIGNATURE _____

DATE _____

INVESTIGATOR'S SIGNATURE _____

Appendix C.

CONSENT FOR RELEASE OF MEDICAL INFORMATION FORM

Releaser of Subject's Medical Information from Evaluating Physician to Researchers

RESEARCH PROJECT CONDUCTED AT

Department of Psychology, Concordia University
Sydney Miller, Ph.D., Department of Psychology, Concordia University

Hypertension: Physical and Psychological Moderators in the Canadian Black Population

I hereby authorize Dr. Lawrence Morris M.D. to release to the researchers, and/or their representatives, the results from the medical (physical) examination that I underwent as a preparatory step to my inclusion in this research project.

NAME (PLEASE PRINT) _____

PARTICIPANT'S SIGNATURE _____

DATE _____

PHYSICIAN'S SIGNATURE _____

Appendix D.

PHYSICAL EXAMINATION REPORT

For participation in research study:

Hypertension: Physical and Psychological Moderators in the Canadian Black Population.

Name: _____ **Sex:** F M

DOB: _____
Day **Month** **Year**

Height: **Weight:** **Blood Pressure:** **Pulse:** **Respiration:**

HEENT

_____
_____
_____

Thyroid: _____ **Lymph:**_____

Respiration: _____

Cardiovascular: _____
_____

GI: _____

GU: _____

Central Nervous System: _____

Skin: _____

Musculo Skeletal: _____

Pregnancy: Yes: _____ No: _____ **L.M.P.** _____
Day **Month** **Year**

HPI: _____
_____

Allergies:

_____

Previous Medical History:

_____

_____

_____

Family History:

Mother:

_____

_____

Father: _____

_____

Sibling(s): _____

_____

Functional Inquiry: (Positives Only)

_____

_____

_____

_____

I see not medical contraindication to his/her participation in your sodium and hypertension study.

Signature: _____ **Date:** _____

Day Month Year

Appendix E.

Visual Analog Mood Scale (VAMS)

Participant Code: _____

HOW ARE YOUR FEELING RIGHT NOW?

Indicate on each of the scales below by starting **a vertical stroke** through the line at the appropriate point.

Not at all Nervous _____ Very Nervous

Not at all Agreeable _____ Very Agreeable

Not at all Happy _____ Very Happy

Not at all Tense _____ Very Tense

Not at all Anxious _____ Very Anxious

Not at all Relaxed _____ Very Relaxed

Not at all Discouraged _____ Very Discouraged

Not at all Annoyed _____ Very Annoyed

Not at all Sad _____ Very Sad

Not at all Irritated _____ Very Irritated

Not at all Angry _____ Very Angry

Not at all Depressed _____ Very Depressed

Not at all Guilty _____ Very Guilty

Appendix F.

PERCEIVED RACISM SCALE

SECTION 1

INSTRUCTIONS – PLEASE READ

Please circle the number which corresponds to how often you experience each event. **Please circle only one number for question "a" and one number for question "b" for each item.** For example, if you felt, over the past year that you were assigned jobs no one else wanted, on average, "several times a month," you'd circle number "3" next to item 1a. If you felt, over your lifetime you were assigned jobs no one else wanted, on average "several times a year, " you would circle number "2" next to item 1b.

A. RACISM ON THE JOB:
(If you have never been employed, please skip this section and go to page 2, question 11, section B).

0=Not applicable, 1=Almost never, 2=Several times a year, 3=Several times a month, 4=Several times a week, 5= Several times a day

1. Because I am Black, I'm assigned the jobs no one else wants to do.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

2. At work, when different opinions would be helpful, my opinion is not asked for because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

3. I am treated with less dignity and respect than I would be if I were White.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

4. I am watched more closely than other workers because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

5. Racial jokes or harassment are directed towards me at work.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

0=Not applicable, 1=Almost never, 2=Several times a year, 3=Several times a month, 4=Several times a week, 5= Several times a day

6. Because I am Black, I feel as if I have to work twice as hard.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

7. Tasks that require intelligence are usually given to Whites, while Blacks get those that don't require much thought.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

8. I am often ignored or not taken seriously by my boss because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

9. Whites often assume I work in a lower status job than I do and treat me as such.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

10. A White co-worker with less experience and qualifications got promoted before me.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

A. RACIASM IN ACADEMIC SETTINGS:

11. I have been made to feel uncomfortable in a classroom of White students.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

12. Teachers and students assume I'm less intelligent because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

13. Whites assume I gained admission to school only because of Affirmative Action, not based on my abilities or intelligence.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

0=Not applicable, 1=Almost never, 2=Several times a year, 3=Several times a month, 4=Several times a week, 5= Several times a day

14. My graded assignments are judged more critically because I am Black.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

15. Although I am equally prepared and responsive, I am called on less than Whites in the class.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

16. When I excel academically, I am looked upon as an exception to my own race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

17. I find it difficult to trust White teachers and/or students.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

18. My academic advancement has suffered because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

19. Although I am equally intelligent, Whites often don't include me in study groups because I am Black.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

20. I have been taught in school that Europeans are civilized and Africans are primitive.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

0=Not applicable, 1=Almost never, 2=Several times a year, 3=Several times a month, 4=Several times a week, 5= Several times a day

B. RACISM IN THE PUBLIC REALM:

21. I have been called insulting names related to my race or skin color.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

22. When I go shopping, I am often followed by White security guards or watched by White clerks.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

23. I heard comments from Whites expressing surprise at my or other "minority" individuals' intelligence or industriousness.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

24. People "talk down" to me because I am Black.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

25. I have been refused rental housing, which was then later rented to Whites of similar standing (e.g. comparable family income).

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

26. I know of people who have gotten into trouble (gotten hurt, beaten up, shot) by Whites (individuals, gangs, police, White male groups).

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

27. I have difficulty getting a loan because I am Black.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

28. I am followed, stopped or arrested by White police more than others because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

0=Not applicable, 1=Almost never, 2=Several times a year, 3=Several times a month, 4=Several times a week, 5= Several times a day

29. I have tried to make my speech and posture appear passive when dealing with Whites.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

30. Waiters and waitresses have ignored me and served Whites first.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

31. White males talk about not desiring Black women for serious relationships versus those with White women.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

32. My house has been vandalized because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

33. I have had to allow Whites to obtain the best seats in public places.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

34. I have been denied hospitalization or medical care because of my race.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

35. I have known Black men who have suffered negative consequences for taking to White women (being hurt or killed).

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

36. I have encountered legal restrictions against Blacks. Please circle each one that applies: housing, marriage, jobs, use of public facilities.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

0=Not applicable, 1=Almost never, 2=Several times a year, 3=Several times a month, 4=Several times a week, 5= Several times a day

D. RESPONSES TO RACIST STATEMENTS:

37. "Over the past few years, Blacks have gotten more Economic and educational breaks than they deserve."

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

38. "Blacks should not push themselves into places where They are not wanted."

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

39. More Blacks are on welfare because they are too lazy to get a job.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

40. "If a Black family moved in next door to me, I would seriously think about moving."

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

41. "Black people are generally not as smart as Whites."

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

42. "Black men have an "animal-like" passion in bed."

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

43. Some Blacks are so touchy about their rights that it is difficult to get along with them.

a. How often has this happened in the past year?	0	1	2	3	4	5
b. How often has this happened during my life?	0	1	2	3	4	5

SECTION II: In answering the questions in this section, PLEASE CIRCLE A RESPONSE NEXT TO EACH EMOTION THAT BEST DESCRIBES HOW YOU FEEL IN THAT SETTING:

44. When I experience RACISM ON THE JOB,

		1=Not at all		3= Moderately		5= Extremely
I GENERALLY FEEL:	angry	1	2	3	4	5
	frustrated	1	2	3	4	5
	sad	1	2	3	4	5
	powerless	1	2	3	4	5
	hopeless	1	2	3	4	5
	ashamed	1	2	4	4	5
	strengthened	1	2	3	4	5

45. When I experience RACISM IN ACADEMIC SETTINGS,

		1=Not at all		3= Moderately		5= Extremely
I GENERALLY FEEL:	angry	1	2	3	4	5
	frustrated	1	2	3	4	5
	sad	1	2	3	4	5
	powerless	1	2	3	4	5
	hopeless	1	2	3	4	5
	ashamed	1	2	4	4	5
	strengthened	1	2	3	4	5

46. When I experience RACISM IN THE PUBLIC REALM, (e.g. a restaurant),

		1=Not at all		3= Moderately		5= Extremely
I GENERALLY FEEL:	angry	1	2	3	4	5
	frustrated	1	2	3	4	5
	sad	1	2	3	4	5
	powerless	1	2	3	4	5
	hopeless	1	2	3	4	5
	ashamed	1	2	4	4	5
	strengthened	1	2	3	4	5

47. When I HEAR RACIST STATEMENTS,

I GENERALLY FEEL:

	1=Not at all		3= Moderately		5= Extremely
angry	1	2	3	4	5
frustrated	1	2	3	4	5
sad	1	2	3	4	5
powerless	1	2	3	4	5
hopeless	1	2	3	4	5
ashamed	1	2	4	4	5
strengthened	1	2	3	4	5

SECTION III: In answering the questions in this section, PLEASE MARK THE BEHAVIOR OR BEHAVIORS THAT BEST DESCRIBE HOW YOU DEAL WITH RACISM IN THAT SETTING:

48. When I experience RACISM ON THE JOB, I generally deal with it by:

A. speaking up ____ B. accepting it ____ C. ignoring it ____ D. trying to change things ____

E. keeping it to myself ____ F. working harder to prove them wrong ____ G. praying ____

H. avoiding it ____ I. getting violent ____ J. forgetting it ____ K. OTHER (please list) _____

49. When I experience RACISM IN ACADEMIC SETTINGS, I generally deal with it by:

A. speaking up ____ B. accepting it ____ C. ignoring it ____ D. trying to change things ____

E. keeping it to myself ____ F. working harder to prove them wrong ____ G. praying ____

H. avoiding it ____ I. getting violent ____ J. forgetting it ____ K. OTHER (please list) _____

50. When I experience RACISM IN THE PUBLIC REALM (e.g. a restaurant), I generally deal with it by:

A. speaking up ____ B. accepting it ____ C. ignoring it ____ D. trying to change things ____

E. keeping it to myself ____ F. working harder to prove them wrong ____ G. praying ____

H. avoiding it ____ I. getting violent ____ J. forgetting it ____ K. OTHER (please list) _____

51. When I HEAR RACIST STATEMENTS, I generally deal with it by:

A. speaking up ____ B. accepting it ____ C. ignoring it ____ D. trying to change things ____

E. keeping it to myself ____ F. working harder to prove them wrong ____ G. praying ____

H. avoiding it ____ I. getting violent ____ J. forgetting it ____ K. OTHER (please list) _____

SECTION IV: In answering the questions in this section, PLEASE RANK THE ITEMS BELOW EACH SETTING (e.g. job, school…) IN THE ORDER OF IMPORTANCE THAT THEY HAVE FOR YOU; FROM 1 to 4, WITH 1 BEING THE MOST IMPORTANT ITEM AND 4 BEING THE LEAST IMPORTANT.

52. When I experience RACISM ON THE JOB, I generally:
(1 = most important to 4 = least important)

_____ think Whites have a problem.

_____ think that the person being racist has a problem.

_____ feel bad about being Black.

_____ feel bad about myself.

53. When I experience RACISM AT SCHOOL, I generally:
(1 = most important to 4 = least important)

_____ think Whites have a problem.

_____ think that the person being racist has a problem.

_____ feel bad about being Black.

_____ feel bad about myself.

54. When I experience RACISM IN PUBLIC SETTINGS, I generally:
(1 = most important to 4 = least important)

_____ think Whites have a problem.

_____ think that the person being racist has a problem.

_____ feel bad about being Black.

_____ feel bad about myself.

55. When I HEAR RACIST STATEMENTS, I generally:
(1 = most important to 4 = least important)

_____ think Whites have a problem.

_____ think that the person being racist has a problem.

_____ feel bad about being Black.

_____ feel bad about myself.

Appendix G.

Spielberger Anger Expression Scale

A number of statements which people have used to describe themselves when they feel angry or furious are given below. Read each statement and then circle the appropriate answer to indicate how often you feel or act in the manner described when you feel angry or furious. There are no right or wrong answers. Do not spend too much time on any one statement. Please answer all questions.

	When angry or furious:	Almost Never	Sometimes	Often	Almost Always
1.	I control my temper …….	1	2	3	4
2.	I express my anger ……..	1	2	3	4
3.	I keep things in ……	1	2	3	4
4.	I make threats I don't really mean to carry out ..	1	2	3	4
5.	I pout or sulk ……...	1	2	3	4
6.	I withdraw from people ……	1	2	3	4
7.	I make sarcastic remarks to others……..	1	2	3	4
8.	I keep cool ……..	1	2	3	4
9.	I do things like slam doors ……...	1	2	3	4
10.	I boil inside but I don't show it …….	1	2	3	4
11.	I argue with others ……	1	2	3	4
12.	I tend to harbour grudges that I don't tell anyone about ……..	1	2	3	4
13.	I strike out at whatever infuriates me…	1	2	3	4
14.	I am secretly quite critical of others …..	1	2	3	4
15.	I am angrier than I am willing to admit….	1	2	3	4
16.	I calm down faster than most people ……	1	2	3	4
17.	I say nasty things …….	1	2	3	4
18.	I am irritated a great deal more than people are aware of ……	1	2	3	4
19.	I calm down and think about whatever angered me before I settle the problem …..	1	2	3	4
20.	I remain patient with others I'm working with even when provoked ……..	1	2	3	4
21.	I don't let other things irritate me further….	1	2	3	4

	When angry or furious:	Almost Never	Sometimes	Often	Almost Always
22.	I cover up my angry feelings so that I can continue my work …….	1	2	3	4
23.	I will brood about it and feel resentful ……	1	2	3	4
24.	I control my temper so that I can handle the problem …….	1	2	3	4
25.	I am afraid to express my anger to someone who is angry at me …….	1	2	3	4
26.	I can relax while I think about whatever made me angry …….	1	2	3	4
27.	I don't brood or feel resentful because it only makes the problem worse …….	1	2	3	4
28.	I keep my cool so that I can handle the problem that bothered me ……..	1	2	3	4
29.	I feel anxious about expressing or showing my anger ……..	1	2	3	4
30.	I have trouble keeping my cool when I'm criticized ……..	1	2	3	4
31.	I instantly try to figure out what got me angry...	1	2	3	4
32.	I lose my temper ……	1	2	3	4
33.	I am apt to tell others how I feel ……..	1	2	3	4
34.	I try to stay calm even though I think I was treated unfairly……..	1	2	3	4
35.	I think about what made me angry……..	1	2	3	4
36.	I try to calmly handle the problem that made me angry ……	1	2	3	4
37.	I will lash out at whatever angered me ……	1	2	3	4
38.	I try to calmly talk with the person I'm angry with at a later time …….	1	2	3	4
39.	I feel hurt and stay silent …….	1	2	3	4
40.	I am fearful about expressing my feelings ……	1	2	3	4
41.	I'm irritable a great deal more than people are aware of …….	1	2	3	4

Appendix H.

Script for Math Task

- A series of mathematical subtraction equations will be presented on the computer monitor.
- You must respond by pressing one of the buttons on the mouse.
- Press the right button, the one marked “C”, if you think that the answer on the screen is correct.
- Press the left button, the one marked “I”, if you think that the answer on the screen is incorrect.
- If you are accurate in your response, the computer will emit a high-pitched tone.
- If you are inaccurate in your response, the computer will emit a low-pitched tone.
- You will be presented with 60 equations in total.
- You will have 3 seconds to respond to each equation.
- At the end of 2 seconds the colour of the equation on the monitor will change.
- This indicates that you have 1 second left to respond.
- At this time we suggest that you make a guess and respond by pressing either “C” or “I”, even if you do not know what the correct response is.
- You will score an automatic 0 if no response is made, and no tone will be emitted.
- It is better to guess rather than not respond since it has been shown that most of the time, a person will guess correctly as the brain has already processed most of the information presented.

Appendix I.

Script for Cold Pressor Task

- I will place a cold pack on your forehead and hold it in place with a Velcro band for 2 minutes.
- Please keep your arms and legs relaxed at all times.
- After a period of 2 minutes, I will take the ice pack off.
- After a break of 1 minute, I will again put the ice pack on your forehead for another 2-minute period.
- I will remove the ice pack at the end of these two minutes.
- If at any time you feel that you cannot continue with this task and want to stop the task, please tell me this and I will remove the ice pack at your request.

Appendix J.

Script for Discrimination Recall Task

- I want you to think about an event in your life when you felt discriminated against.
- I would like you to imagine that you are back in that situation.
- Try to re-experience the situation, especially, how you were feeling at that time.
- I would like you to tell me about that event, describing your feelings to me as best as you can.
- Talk to me about your experience and your feelings for 3 minutes.
- I will tell you when to stop.
- Begin now.

Appendix K.

Sodium Diet Instructions

During the 10 designated special diet days please use the following procedures.

- Day 1
  - Consume regular diet **plus** one package of Lipton Noodle soup (provided) and the packet of table salt (provided).
  - You may consume the soup over the course of the day or in one sitting. Please note that the soup requires preparation as per the package instructions and be sure to entirely dissolve the soup base.
  - The salt may be added to one or more portions of your normal dietary intake (e.g. oatmeal cereal, rice, sprinkled on moist vegetables such as sliced tomato, or added to any compatible beverages such as juice or plain water)

- Days 2-8
  - Please follow the same dietary procedures as on Day 1.

- Day 9
  - Please follow the same dietary procedures as on Days 1-8.
  - You may not consume coffee, cigarettes, alcohol, or recreational drugs from 6:00 p.m. until after the laboratory procedures on day 11 (see below).

- Day 10
  - Please follow the same dietary procedures as on Days 1-9.
  - Please continue to refrain from consuming coffee, cigarettes, alcohol, or recreational drugs.
  - Please present yourself at the laboratory (as instructed) to receive your ambulatory blood pressure unit and 12-hour overnight urine collection kit (kit contains necessary instructions).

- Day 11
  - Please follow the same dietary procedures as on Days 1-10.
  - Please continue to refrain from consuming coffee, cigarettes, alcohol, or recreational drugs.
  - Please present yourself at the laboratory (as instructed) to return your ambulatory blood pressure unit and urine specimen, and to participate in the laboratory segment of the study.

- General Information
  - It is extremely important that you follow the diet consumption instructions, as we will be analyzing the urine specimen.
  - We will be contacting you on a daily basis in order to ensure protocol compliance.

If you have any questions during any phase of the study, please contact the researchers at:

848-2424 Ext. 2846

We will be telephoning you periodically during these 10 days to maintain contact.

Appendix L.

Urine Collection Instructions

Kit Contents:

Large plastic collection container (brown)
Urine specimen vial (clear plastic tube with blue cap)
Pair of latex gloves

- As this is an overnight 12-hour urine collection it is important that you begin collecting urine output by 8:00 p.m. in order to ensure a full 12-hour collection.

- It is important to remember that you may not consume coffee, cigarettes, alcohol, or recreational drugs during this period.

- Please indicate in the space provided below the time of the first urine collection (e.g. 8:25 p.m.). Collect the urine in the large brown plastic container.

- If you urinate again before retiring for the night or if you wake during the night and need to urinate, please collect the entire output in the same container. There is no need to indicate these collection times.

- When you wake up please collect the entire first morning urine output in the same large brown collection container. If this is the last collection of the 12-hr period, please indicate the time in the space provided below (e.g. 7: 53 a.m.). Please note that you will be presenting yourself at the laboratory at 9:00 a.m.

- After the morning collection, place the container on a flat level surface and verify the quantity of urine in the container. Write down the quantity in the space provided below.

- After you have written the total quantity below, please pour a sufficient quantity of the urine from the brown collection container into the urine specimen vial (please fill to approximately 1 cm below the top of the tube) and place the blue cap firmly on the vial.

- Please insert the filled vial into the plastic bag (provided) and bring the vial to the scheduled laboratory appointment (same morning).

First (evening) collection time: _____ Last (morning) collection time: _____

Urinary output quantity: _____ ml

Participant # _____
Date: _____

If you have any questions during any phase of the study, please contact the researchers at

848-2424 Ext. 2846