Evaluating Psychosocial Variables and their Link to Hypertension Using Mindfulness-Based Stress Reduction

by

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Institute of Medical Science
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Abstract

Previous research has reported positive associations between anger, perceived stress and blood pressure. These associations have largely been based on cross-sectional data and a small number of longitudinal works. Using a prospective longitudinal cohort design, this study more directly tested the relationships between anger, perceived stress and blood pressure by using a psychological therapeutic intervention (mindfulness-based stress reduction) to manipulate anger and perceived stress. Anger and perceived stress were in turn evaluated for association with blood pressure. Despite improvements in psychosocial functioning and reductions in blood pressure, findings from this study failed to demonstrate an association between change in anger or perceived stress with change in daytime or 24-hour ambulatory blood pressure. A model where these variables interact indirectly via stress coping mechanisms or health behaviours may be the linking mechanism in this study. Results from this thesis have contributed evidence to a divided field dominated by cross-sectional research.

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Chapter 1 Introduction

1 Hypertension: A Global Pandemic

In 1961, the Framingham Heart Study identified elevated blood pressure as a risk factor for heart disease.³ Today, hypertension is recognized as one of the major risk factors for cardiovascular disease and death.^{4,5} This view has been supported by the World Health Organization which has stated that preventing and controlling hypertension is “one of the single most cost effective strategies for reducing the global burden of premature cardiovascular disease and death”.^{1,6} Hypertension affects approximately 25% of Canadian adults aged 35-64 years⁷ and over 50% of people aged 65 years and older.^{8,9} Based on globally collected data, the number of adults with hypertension is estimated to increase by 60 percent by 2025.¹⁰ Taken together, this information outlines the significant contribution made by hypertension to worldwide death and chronic disease and its growth into a global pandemic.

1.1 Risk Factors for Hypertension

The origin of primary hypertension remains unknown. To date, the development of hypertension is best understood as the by-product of an accumulation of environmental and genetic risk factors.¹¹ These traditional risk factors include diabetes, age, sex, excessive sodium consumption, lack of exercise and genetic background.¹² Together, these factors are understood to increase the risk for blood pressure elevation and hypertension development.¹²

Psychosocial variables have also been linked to blood pressure as suspected risk factors implicated in the development of hypertension.¹³⁻¹⁶ The recognition of a relationship between psychosocial factors and blood pressure is reflected in the Canadian blood pressure guidelines. In 2007, the Canadian Hypertension Education Program (CHEP) – an organization focused on delivering evidence-based blood pressure guidelines for clinicians across Canada – adopted stress management as a lifestyle recommendation for hypertensive patients in whom stress appears to be an issue.^{17,18} The assimilation of stress management into national blood pressure guidelines indicates a growing acceptance of psychosocial factors as possible contributors to hypertension development.

The consideration of psychosocial variables has also been recognized in the cardiovascular disease literature. The 2004 INTERHEART case-control study, carried out in 52 countries in over 29,000 patients, revealed that psychosocial stress was associated with an increased risk for myocardial infarction among all sexes, ages and regions of the world.¹⁹ The following year Rozanski et al. published a “State-of-the-Art Paper” in the *Journal of the American College of Cardiology* where the focus of the manuscript was to persuade clinicians to consider psychosocial risk factors in cardiac practice.²⁰ Currently, the Heart and Stroke Foundation cites stress as a modifiable risk factor for heart disease²¹ and the Canadian Psychological Association cites psychosocial risk factors for heart disease.²² Taken together, this evidence outlines the growing acceptance of psychosocial variables as modifiable risk factors not only for hypertension but also cardiovascular disease.

1.2 Pathophysiological Evidence & Hypothesized Casual Pathways – Linking Psychosocial Variables & Blood Pressure

Several lines of evidence outline possible pathways by which psychosocial risk factors could affect blood pressure. Mechanisms underlying those relationships have been divided into 2 categories: pathophysiological mechanisms such as the over-stimulation of neuroendocrine pathways²³ and behavioural mechanisms whereby psychosocial variables increase the risk of engaging in poor health choices.²⁴

One hypothesized pathway linking emotional arousal and blood pressure includes the over-activation of the sympathetic nervous system (SNS) and hypothalamic-pituitary-adrenal (HPA) axis. More specifically, that repeated stimulation of the cardiovascular system from SNS/HPA neurohormones due to psychosocial stress contributes to the progression and development of hypertension. Elevations in neurohormones have been demonstrated in response to acute^{25, 26} and chronic stress,²⁷⁻²⁹ and evidence from the animal literature has demonstrated that long-term neurohormone infusion can produce sustained hypertensive states.³⁰ Taken together, this information outlines how chronic emotional arousal could be linked to the development of hypertension via the over-stimulation of neuroendocrine pathways.

Research on cardiovascular reactivity has also provided a possible pathway connecting psychosocial stress and blood pressure. Cardiovascular reactivity is most frequently used as a

laboratory tool to measure physiological stress responses to psychological stressors. Blood pressure is a measureable component of cardiovascular reactivity. Evidence has linked specific psychosocial variables such as anger to greater cardiovascular reactivity.³¹⁻³⁵ Increased cardiovascular reactivity has in turn been associated with long-term increases in blood pressure³⁶ and increased cardiovascular risk status.³⁷ Together this information outlines an additional pathophysiological pathway by which chronic exposure to psychosocial stressors could influence the development of hypertension by altering cardiovascular reactivity profiles.

Psychosocial variables have also been linked to blood pressure via baroreflex pathways. Various types of psychosocial stress have been associated with reduced baroreflex sensitivity, including anxiety³⁸ and post-traumatic stress disorder (PTSD).³⁹ Reduced baroreflex sensitivity has been associated with reduced parasympathetic and increased sympathetic activity.^{38, 40} This altered baroreflex profile has been associated with increased blood pressure, even upon follow-up five years later.⁴¹ Anxiety and PTSD are very different illnesses; however they both share the commonality of a chronically stressed psychological state. Thus, taken together, this evidence outlines another possible mechanistic pathway by which chronic psychosocial stressors may be linked to elevated blood pressure through altering baroreflex sensitivity.

More recently, the immune system and inflammatory molecules have been implicated in hypertension development.⁴²⁻⁴⁵ Specific immunological features have been identified, such as “serum immunoglobulin [and] alterations in both humoral and cellular immune functions”⁴² that distinguish hypertensive patients from their normotensive counterparts. Interestingly, chronic psychosocial stress has been reported to have negative effects on the immune system,^{46, 47} and recent research has also documented psychosocial stressors such as perceived stress to co-vary with specific immunological markers like proinflammatory chemokine interleukin 8.⁴⁸ The linkages between these variables outline another possible mechanism whereby psychosocial stressors could contribute to chronic diseases of inflammation via the immune system.⁴⁹ Although evidence from this field is largely preliminary, it nonetheless outlines another pathway by which psychosocial variables may exert an effect on blood pressure.

Several pathways have been hypothesized that link psychosocial variables to elevated blood pressure and the development of hypertension. They include: the over-stimulation of neuroendocrine pathways, increased cardiovascular reactivity, reduced baroreflex sensitivity and

altered immunological profiles. Evidence from this field of research has helped to strengthen the legitimacy of psychosocial variables as possible risk factors for hypertension development.

1.3 Anger & Perceived Stress as Psychosocial Risk Factors

Anger^{24, 50-77} and perceived stress⁷⁸⁻⁹⁰ have been associated with higher blood pressure and poor health outcomes. Most evidence has been limited to observational research. A better understanding of anger and perceived stress as suspected risk factors for hypertension development is necessary to manage the burden of hypertensive disease. Prospective longitudinal cohort and experimental studies, specifically those that are designed to manipulate anger and perceived stress, would enable a more rigorous evaluation of these relationships. Current evidence on the relationship between anger, perceived stress and blood pressure, as well as the use of therapeutic psychological interventions to design a prospective longitudinal cohort study that manipulates anger and perceived stress will be discussed.

2 Anger & Blood Pressure

2.1 Historical Context & Definitions

Anger is recognized as both an emotional state and a personality type. Most commonly, anger is thought of as “feelings that vary in intensity from mild irritation or annoyance to intense fury and rage”.² As a personality type, anger refers to the propensity for some individuals to experience angry feelings more frequently than others. Anger is often mistakenly used interchangeably with other behaviours or personalities such as hostility and aggression. Much of this misuse is due to the contribution of anger to the characterization of these other behaviours and personalities. For instance, hostility involves the “frequent experience of angry feelings, but also has the connotation of a complex set of attitudes that includes meanness and viciousness, as well as aggressive and often vindictive behaviour”.² For this reason, anger is “generally considered to be a more fundamental concept than hostility or aggression [as] anger refers to feelings [while the latter]... describe [a collection of] negative attitudes and destructive or punitive behaviour”.² Thus, anger is both an emotion and a personality type that maintains its own construct and definition separate from other related psychosocial concepts.

A relationship between anger and blood pressure was first proposed in 1939 when Franz Alexander published the concept of the “hydraulic model”.⁹¹ Suppressed anger was hypothesized

to have a maladaptive effect on blood pressure while expressed anger was thought to have the opposite. This distinction between suppressed and expressed anger was further developed by Funkenstein et al. in 1954.⁹² Despite these early beginnings, research on the effects of anger on the cardiovascular system did not begin in earnest until the 1980s when the role of anger as the central component of Type A behaviour syndrome was identified.⁹³ Two teams run by Ernest Harburg and Charles D. Spielberger led the research on studying anger's contribution to the etiology and progression of medical disorders. In a long-term hypertension research project, Harburg and colleagues investigated the relationships between anger expression and blood pressure using responses to hypothetically anger-proving questionnaires.^{52, 53, 94-96} Preliminary results linked anger expression to blood pressure. These findings were combined with cardiovascular reactivity literature on blood pressure and peripheral resistance,⁹⁷ together developing the idea of a "hypertensive personality".^{98, 99} These combined efforts built the framework necessary to push research on the psychobiological effects of anger to a new level.

Attempts to explain these new relationships between anger and blood pressure emerged. Early psychoanalytic theories from Alexander's time proposed anger expression inward to cause feelings of guilt and depression which somehow contributed to negative physiological consequences.^{2, 100} Later theories attempted to explain findings on a more basic pathophysiological level. These included linking anger expression to over-activation of the SNS and HPA axis where repeated increases in heart rate, blood pressure, vascular resistance and secretion of cortisol and catecholamines were hypothesized to initiate a hypertensive cascade.^{101, 102} The growth and expansion of pathophysiological research demonstrated the increasing consideration of anger as a possible contributor to the development of hypertension.

As research progressed, calls began to emerge for more conceptual distinctions of anger to more accurately assess anger's contribution to different diseases. Spielberger was the most notable contributor to this movement, ultimately developing the validated State-Trait Anger Expression Inventory (STAXI).^{103, 104} The questionnaire was novel for 2 reasons: 1) it further differentiated anger from hostility and aggression; and 2) it identified and measured additional and distinct anger-related constructs, separate from anger expression. Anger expression was also further expanded upon by the new questionnaire. This collective work from both Harburg and Spielberger developed anger into two separate but related concepts: **anger experience** and **anger expression**.

Anger experience addressed the physical experience of anger, asking: how often and with what intensity did individuals experience angry feelings? In order to measure this, anger experience was differentiated into “state” and “trait” anger. State anger was defined as a “psychobiological emotional state or condition marked by subjective feelings that var[ied] in intensity from mild irritation or annoyance to intense fury and rage”² accompanied by physiological responses from both neuroendocrine and autonomic nervous systems. State anger was further divided into subscales: feeling angry, feeling like expressing anger verbally and feeling like expressing anger physically. As opposed to angry feelings at a particular moment in time, trait anger measured “individual differences in the disposition to perceive a wide range of situations as annoying or frustrating and by the tendency to respond to such situations with elevations in state anger”.² It was expected that “individuals with high trait anger scores [would] experience state anger more often and with greater intensity than individuals who [were] low in trait anger”.² Trait anger was also divided into subscales: anger temperament (which measured the disposition to experience anger without specific provocation) and anger reaction (which measured the frequency that angry feelings were experienced in situations that involved frustration and/or negative evaluations). Thus, according to the developments by Spielberger, state anger measured the level of angry feelings at a specific moment of testing whereas trait anger measured how often angry feelings were experienced over time.

Anger expression as defined by Spielberger was differentiated into 2 different subtypes: 1) anger expression outward, which involved the expression of anger toward other persons or objects in the environment; and 2) anger expression inward, which represented anger directed inside such as holding in or suppressing angry feelings.² Spielberger also included the concept of “anger control” in the category of anger expression, viewing the concept of anger expression on a continuum with the control of anger as one endpoint and the expression of anger as the other. Anger control was similarly categorized: anger control outward measured the control of angry feelings by preventing the expression of anger toward other persons or objects in the environment, and anger control inward measured the control of suppressed angry feelings by calming down or cooling off.² As part of addressing the existence of anger expression on a continuum, Spielberger also developed an overall omnibus anger expression measure titled the “anger expression index”. The anger expression index provided a total measure of anger

expression, based on how anger was expressed (inward or outward) and how anger was controlled (inward or outward). Combined, these scales defined anger expression.

Evidence supporting the validity of this framework for understanding anger was demonstrated by the widespread uptake of anger as a valid measureable construct.^{105, 106} The STAXI itself was translated into different languages,^{107, 108} further reinforcing the validity of its theoretical framework. No other validated anger questionnaire has contributed as substantially to our understanding of anger and its psychobiological effects. The combination of groundwork laid by Harburg, improved conceptualization of anger by Spielberger and development and widespread uptake of the STAXI questionnaire together represent some of the most important contributions to the field of anger research.

Thus, to summarize, anger is both an emotional state and a personality type that is distinct from other anger-like constructs such as hostility and aggression. Research studying the relationship between anger, blood pressure and cardiovascular disease began in 1939 with Franz Alexander and has evolved significantly since that time. Evidence from this research on the psychobiological relationship between anger, blood pressure and cardiovascular disease has significantly shaped the conceptualization of anger from both a psychological and physiological perspective. Teams led by Harburg and Spielberger have contributed significantly to our understanding of anger. Most notable has been the development of the validated STAXI questionnaire which has enabled a better understanding of anger and its relationship to different diseases.

2.2 Evidence to Date on Anger & Blood Pressure

The contribution of anger to the development of hypertension has been studied extensively. Since the proposition of Alexander's original hydraulic model, where anger expressed inward was associated with increased blood pressure and anger expressed outwards was associated with decreased blood pressure, to date approximately half of the accumulated literature has reported some association between these 2 variables.¹⁰⁹

However, interpreting this literature has been complicated by several factors including different outcome measures between studies (i.e., examining associations between anger and blood pressure as continuous variables vs. dichotomous or categorical variables), a lack of prospective,

rigorously controlled longitudinal cohort or experimental research that measures these variables at more than one point in time, a lack of consideration for sex/gender or race-ethnicity and a lack of consistent pathophysiological mechanistic explanations. Evidence both supporting and refuting a relationship between anger and blood pressure will be presented below.

The majority of anger and blood pressure research has used the STAXI questionnaire. As the STAXI measures distinct anger constructs (*state anger, trait anger, anger expression inward, anger expression outward, anger control inward and anger control outward*) many of these constructs have individually been investigated for association with blood pressure. Therefore, the following review of the literature will address each anger construct and its relationship to blood pressure individually.

2.2.1 State Anger

State anger (the experience of angry feelings and associated physiological response at the moment of testing) is, at best, weakly associated with blood pressure. In total, 5 cross-sectional studies have examined this relationship. Four of these cross-sectional studies examined state anger by comparing the following groups: hypertensives and normotensives;⁶⁶ mild hypertensives and normotensives;⁹⁹ medicated hypertensives, unmedicated hypertensives and normotensives;¹¹⁰ and hypertensives and white coat hypertensives.⁵⁸ One cross-sectional study examined associations between state anger and blood pressure as continuous variables.⁵⁷ From all 5 studies, only 1 reported a significant difference in state anger between hypertensives and normotensives.⁶⁶ No state anger subscales have been investigated for association with blood pressure. See Table 1 for a summary.

The state anger studies were roughly evenly distributed in terms of male and female subject representation, with a slight preference for male subjects. All studies administered the STAXI and all but 1 reported the use of a reliable method of blood pressure measurement⁶⁶ (where reliable blood pressure measurement refers to either automated office, automated home, ambulatory or standardized manual blood pressure measurement). Interestingly, the only study that used unreliable blood pressure measurement techniques was the only study to report significant findings.⁶⁶

Based on the abovementioned evidence there appears to be, at best, a weak signal between state anger and blood pressure. As mentioned, only 1 study reported a significant finding. This study also implemented the least rigorous blood pressure measurement techniques therefore making their findings the least robust. In addition to weak methodology, a second concern was the inclusion of war veterans as the hypertensive sample under study. The inclusion of such participants raises questions about the impact of war experiences on anger and thus the generalizability and applicability of the results. Thus, while a relationship between state anger and blood pressure may exist, based on the appraisal of the available evidence this appears unlikely.

Part of the struggle in identifying a relationship between state anger and blood pressure may lie in the fundamental construct of state anger itself. State anger addresses how angry an individual feels during that particular moment of testing. Thus, an ideal study would test for associations between state anger and blood pressure when individuals are in an angry state. Only 1 study attempted this approach and reported negative results.⁵⁷ The paucity of such research exists for several reasons; most obvious are the logistical challenges of designing such a study. One approach could involve a case-control design where levels of state anger and 24-hour ambulatory blood pressure are compared between subjects suffering from anger-related issues and non-angry controls. Based on the current status of the state anger literature, confirming any associations between state anger and blood pressure will likely be a slow process.

The above-mentioned results are fairly consistent. State anger appears, at best, weakly associated with blood pressure. If future research is contemplated it should use more rigorous measurement tools and strive to examine associations between state anger and blood pressure when subjects are in an angry state.

2.2.2 Trait Anger

Trait anger measures how often angry feelings are experienced over time. Compared to state anger, trait anger innately assumes a more constant role in individuals' lives. This difference has been reflected in the literature by the increased number of studies examining trait anger and blood pressure. Reports from the literature suggest an association between trait anger and blood pressure; however, this relationship is potentially moderated in part by sex/gender and race/ethnicity.

To date, 9 cross-sectional, 2 longitudinal and 1 interventional study have examined trait anger and blood pressure.^{57, 58, 66-69, 72-75, 99, 110} See Table 2 for a summary. All studies used the STAXI and all but 3 reported a reliable method of blood pressure measurement.^{66, 67, 74} Thus, it can be concluded that most studies were methodologically sound with respect to measurement techniques. Trait anger subscales and their relationship to blood pressure have only been evaluated in 1 study that examined war veterans.⁶⁶ Due to the possible impact of war experiences on anger and the greater amount of trait anger research this study will not be considered in this section.

Trait Anger: Cross-Sectional Evidence

Beginning with cross-sectional evidence reporting no association between trait anger and blood pressure, 4 out of 9 cross-sectional studies fall in this category. Three of these studies examined trait anger by comparing the following groups: mild hypertensive and normotensive adults (all male and majority Caucasian);⁹⁹ medicated hypertensive, unmedicated hypertensive and normotensive adults (all female and African American);¹¹⁰ and hypertensive and white-coat hypertensive university students (majority male and Caucasian).⁵⁸ The remaining cross-sectional study reporting null results examined associations between trait anger and blood pressure as continuous variables among African American and Caucasian male and female high school-students.⁵⁷ The 5 remaining cross-sectional studies that did report significant findings similarly examined differences in trait anger or blood pressure between specific groups or associations between trait anger and blood pressure as continuous variables. All 5 studies reporting positive results included normotensive subjects along with either white-coat hypertensives,⁷² mild hypertensives^{68, 73} or hypertensives.^{66, 67} Together, this information outlines the diversity and significant number of studies that have examined relations between trait anger and blood pressure.

Comparing the positive and negative studies revealed an interesting pattern. All 5 positive studies included at least 40% female participants or more (with the exception of the excluded male war veteran study).⁶⁶ This contrasted with the 4 negative studies which were all male,^{58, 99} a combination of males and females⁵⁷ or only females.¹¹⁰ Closer inspections of the negative studies that did include female subjects revealed specific factors that may have explained their null results. A critical appraisal of these factors will be discussed in the following section. In the

interim, it seems possible that trait anger is moderated in part by sex – or, more specifically, that a signal may only be observed among females. Most studies did not re-analyze their data by sex; therefore, it is not possible to verify this observation. However, this pattern does outline the possible existence of a sex effect and an explanation for the trend observed in the literature.

As stated above, an appraisal of the negative studies that included female subjects revealed specific factors that could explain the null findings. The first factor was age. One of the 2 negative studies recruited male and female high school students (aged 15–17 years).⁵⁷ Hypertension is more prevalent among older individuals.¹² Therefore, the generalizability of data from high school students is questionable. The second factor was choice of outcome measure; more specifically, the potential to obtain different results when studying differences between groups rather than associations between variables. The second negative study reported no difference in trait anger between medicated hypertensive, unmedicated hypertensive and normotensive middle-aged females.¹¹⁰ While the authors reported no difference between groups, they did not demonstrate that trait anger was not associated with blood pressure. By comparison, a positive cross sectional study by Durel et al. did report significant findings among females when trait anger was analyzed for association with blood pressure.⁷³ Thus, it is possible that results from the second neutral study (despite its inclusion of female subjects) could be accounted for by choice of outcome measure. Together, this information highlights specific study features that may explain why the 2 negative studies that included female subjects contrast with the overall observed sex-effect pattern.

In summary, more cross-sectional research has reported positive associations between trait anger and blood pressure (5 positive studies vs. 4 negative studies). This relationship may be moderated in part by sex/gender whereby the effect of trait anger on blood pressure is restricted to females.

Trait Anger: Longitudinal Evidence

The cross-sectional research studying the linkage between blood pressure and trait anger has been followed up by longitudinal research. In 1991, Markovitz et al. conducted a prospective 3-year longitudinal follow-up of normotensive middle-aged females (7% identifying as non-Caucasian).⁶⁹ The authors found that increases in trait anger were independently associated with increases in systolic and diastolic blood pressure over three years of follow-up. In 2007, as part

of secondary analyses from the Atherosclerosis Risk in Communities (ARIC) study, Player et al. explored the influence of trait anger on the progression of prehypertension to hypertension.⁷⁴ The authors followed a cohort of middle-aged males and females for 4-8 years (20% identifying as African American, 80% not identifying as African American). High levels of trait anger were associated with a progression to hypertension. However, after stratifying by sex, trait anger was predictive for males only.

Again, while at first contradictory, discrepant findings between these 2 studies may be attributed to different outcome measures and sample populations under study. Markovitz et al. studied blood pressure change over time among normotensive females who almost all self-identified as Caucasian. No comparator group existed for male or African American subjects in this setting. Player et al. studied males and females but specifically changes in hypertensive status from prehypertension to hypertension rather than a change in blood pressure. One explanation for these discrepant findings could include that trait anger contributes to blood pressure change over time, but that this change in blood pressure does not have the same effect in changing hypertensive status between males and females. It is well established that sex has an important impact on blood pressure. Until menopause¹¹¹ females demonstrate lower blood pressure than males.¹¹²⁻¹¹⁴ Findings from Player et al. may be a reflection of prevalence trends, where a greater proportion of males become hypertensive at middle age compared to age matched females.

In summary, the longitudinal evidence suggests that trait anger has some impact on blood pressure over time. This evidence also reinforces sex/gender and race/ethnicity as possible mediators of that relationship.

Trait Anger: Experimental/Interventional Evidence

One experimental study has added more clarity to the literature.⁷⁵ In a simulated call center study, female subjects were instructed to react to a complaining customer “either authentically and show their true emotions” or “serve with a smile and hide negative emotions”. Race/ethnic background was not specified. Using the STAXI and automated blood pressure measurement, the authors found that trait anger moderated effects on diastolic blood pressure in the “serve with a smile” scenario. Higher trait anger was also associated with higher diastolic blood pressure. This experimental evidence further supports the possible existence of a relationship between trait

anger and blood pressure and reinforces the cross-sectional and longitudinal evidence reporting a relationship between these variables among females.

Trait Anger: Summary

Based on the cross-sectional, longitudinal and experimental research, trait anger appears to have some effect on blood pressure, possibly affecting females more than males. However, some of the mixed-sex cross-sectional research and longitudinal evidence would suggest that trait anger affects males as well. Racial/ethnic contributions are still unclear and could still play significant mediating roles.

2.2.3 Anger Control

Anger control has been less studied, perhaps due in part to its more complicated theoretical construct. Two research teams that examined the relationship between anger control and blood pressure produced conflicting results.^{24, 115} Igna et al. studied 705 middle-aged males in a trial focused on the prevention of metabolic syndrome, type-2 diabetes and cardiovascular disease. They found that anger control was significantly positively associated with blood pressure. By contrast, Ohira et al. who studied 790 male Japanese nuclear power plant workers determined that anger control was not associated with blood pressure. Both studies used the STAXI as well as standardized manual blood pressure measurement. See Table 3 for a summary.

Based on the limited number of conflicting studies it is not possible to make meaningful conclusions. Future research remains to be completed on the relationship between anger control and hypertension. If contemplated, it should investigate the existence of sex differences and follow participants longitudinally to clarify these authors' findings.

2.2.4 Anger Expression

Anger expression is the most frequently researched anger construct with respect to effects on blood pressure. To review, anger expression has been separated into 2 types: anger expressed in (which refers to anger directed inward such as holding in or suppressing angry feelings) and anger expressed out (which refers to the expression of anger toward other persons or objects in the environment).² For the purposes of this study, suppressed anger will always be referred to as anger expressed in and expressed anger will always be referred to as anger expressed out. The

term “anger expression” does not specify which type of expression is being discussed and thus will refer to the broad category encompassing both anger expressed in and anger expressed out.

In general, the literature has reported either a positive association or no association between anger expressed in and blood pressure, and more mixed associations between anger expressed out and blood pressure (i.e., positive, negative and no association). Unfortunately, summarizing the anger expression literature has been complicated by methodological inconsistencies between studies such as the use of differing anger assessment tools and outcome measures. Differing anger assessment tools are due in part to the concept of anger expression emerging in 1939 and the STAXI questionnaire emerging much later (1980s). During this time, anger was assessed using various instruments. The use of these different instruments has made comparisons between studies more difficult. Differing study outcome measures has also added complexity. As highlighted in the trait anger research, one study may evaluate associations between variables and another may dichotomize variables and study differences between groups. Although these approaches are asking the same fundamental question, they are giving different answers. Combining these methodological inconsistencies with the large number of studies has made synthesizing the anger expression literature much more complex (Table 4).

Explaining this state of the literature is necessary to outline the path taken in this review. Studies have first been separated by anger expression type (expressed in or expressed out). Following this, each type has been subdivided into studies reporting a negative relationship, positive relationship or no relationship. The type of blood pressure measurement tool, self-report anger questionnaire, details of the study subjects and outcome measure have been included where appropriate. A summary of this anger expression literature can be found in Table 5.

2.2.4.1 Anger Expressed In

To review, anger expressed inward addresses how often angry feelings are experienced but not expressed.

Anger Expressed In: Negative Relationship

To the best of our knowledge, no study has reported a negative relationship between anger expression inwards and blood pressure.

Anger Expressed In: Positive Relationship

In 1973, Harburg et al. were among the first to report that anger expressed in, as measured by the Harburg questionnaires, was positively associated with standardized manual diastolic blood pressure.⁵³ The average magnitude of difference between subjects who expressed anger inward versus expressed anger outward was ± 3.93 mmHg. Additionally, men who expressed anger inwards reported a greater prevalence of hypertension (19%) compared to those who expressed anger outwards (12.25%). Approximately 10 years later in a follow-up study, the same research team examined the effects of race, sex and anger expression style on blood pressure.⁵² On average, subjects reporting high anger expression inward had significantly higher diastolic blood pressure levels compared to subjects reporting medium or high levels of anger expression outward. Anger expressed in was also independently associated with an increased risk for being classified as hypertensive versus normotensive.*

* Note of clarification: It is stated in the Harburg 1973 publication that “*the use of Funkenstein, et al.'s terms of 'Anger In' and 'Anger Out' [was] solely for ease of labelling; we did not measure the biological responses of anger.*” However, 10 years later in Gentry et al.'s 1982 publication, the authors stated the following: “*Elsewhere, Harburg et al. [authors are referring to Harburg et al. 1973 publication] have used the concept of 'suppressed hostility' to describe an individual's tendency to 1) inhibit aggressive attitudes or actions and 2) feel guilty if such behavior should be expressed. In this paper, we have chosen to use the term 'suppressed anger,' which (based on the [same] actual questionnaire items) describes the individual's tendency not to get annoyed, mad, or angry and which essentially identifies the emotional component of the frustration-aggression sequence*”. Based on this information, it has been understood that the authors were measuring anger expression in 1973. However, at that time it appears that it was categorized as an emotion under the broader construct of hostility. Thus, for the purposes of this literature review, Harburg et al. did look at anger expression in 1973 as it is understood at present day.

Since the publication of the Harburg et al.'s findings, 7 cross-sectional studies have supported their reports.^{50, 51, 57, 58, 67, 70, 71} These consist of 3 studies where the primary analysis evaluated associations between anger and blood pressure as continuous variables^{51, 57, 71} and 4 studies where the primary analysis examined blood pressure or anger levels between groups.^{50, 58, 67, 70} One longitudinal cohort study has also provided support.⁶² Using prospectively collected epidemiological data, the longitudinal study found that every 1-point increase in anger expressed in on the STAXI corresponded to a 12% increased risk for hypertension after 4 years of follow-up among male subjects. The analysis used in this study was specified a priori thus reinforcing these authors' findings. Taken together, this cross-sectional and longitudinal evidence outlines the current support for a positive association between anger expressed inward and blood pressure.

The studies presented above are strictly those that have reported a positive relationship between anger expression inward and blood pressure. On an individual study basis, there are occasional differences with respect to sex/gender and race/ethnicity. However, the distribution of studies reporting significant results for female subjects^{70, 71} is balanced by the number of studies reporting significant results for all male studies⁵⁰ or majority male studies (i.e., 91% male subjects).⁵⁸ Thus, underlying sex/gender and race/ethnic effects remain unclear.

Anger Expressed In: No Relationship

To date, 6 cross-sectional studies have reported no association between anger expressed inward and blood pressure.^{24, 72, 77, 99, 110, 115} Three of these studies examined associations between blood pressure and levels of anger expression inward^{24, 77, 115} while 3 studied differences of anger expression inward or blood pressure between groups.^{72, 99, 110} All studies used the STAXI. Together these studies outline the cross-sectional evidence reporting no association between anger expressed inward and blood pressure.

There are also 2 longitudinal cohort studies that have reported no association between anger expressed in and blood pressure.^{61, 69} Markovitz et al. conducted the prospective longitudinal follow-up of normotensive middle-aged women using the STAXI questionnaire and standardized manual blood pressure measurement. They reported that anger expression inward was not associated with blood pressure over the 3-year follow-up period.⁶⁹ The second longitudinal study recruited male and female subjects, used the Harburg questionnaire and manual blood pressure

measurement.⁶¹ These authors also found no association between anger expression inward and blood pressure over the 7-year follow-up period. Together, these 2 works outline the longitudinal evidence against an association between anger expressed in and blood pressure.

Although the above-mentioned longitudinal evidence offers more robust evidence against an association between anger expressed inward and blood pressure, it should be known that one author's (Julius et al.) decision to analyze psychosocial functioning and blood pressure was included as post-hoc analysis.⁶¹ Based on this detail, Julius et al.'s findings can only be interpreted as hypothesis generating. By contrast, Markovitz et al. specified their longitudinal analysis a priori. Thus, their findings can be considered valid evidence of no association between anger expressed inwards and blood pressure among female subjects.⁶⁹ It is necessary to know this information in order to accurately evaluate the contribution of these studies to the literature.

Anger Expressed In: Summary

To summarize, 10 studies reported a positive relationship between anger expressed in and blood pressure while 8 studies reported no association. From the 10 studies that supported a positive relationship, 9 were cross-sectional and 1 was longitudinal. Among the 8 studies reporting no association, 6 studies were cross-sectional and 2 were longitudinal. Based on an appraisal of the analysis used in one of the no association longitudinal studies, it was determined that only 1 longitudinal study provided acceptable evidence against an association between anger expressed inward and blood pressure.⁶⁹ Thus, there are only 7 valid studies reporting no relationship between anger expressed inward and blood pressure. Overall, there are more studies supporting a relationship between anger expressed inwards and blood pressure rather than refuting it.

Taking a collective approach to the cross-sectional research reveals that study details were relatively homogenous between those reporting positive associations versus no associations. Overall, the methodological quality, which addressed blood pressure measurement techniques and self-report questionnaires as well as appropriateness and generalizability of the sample population under study, was consistent. The proportion of male and female subjects was generally even, as were the number of studies recruiting Caucasian and non-Caucasian subjects. Finally, types of outcome measures were also evenly distributed (i.e., number of studies that evaluated associations between continuous variables vs. studies that examined anger or blood

pressure levels between groups). In summary, methodological differences between studies reporting positive associations versus no associations were minimal.

An evaluation of the longitudinal evidence does not reveal the same consistency. Significant differences between the 3 longitudinal studies include the sex of sample populations under study and outcome measures.^{61, 62, 69} The low number of studies has likely amplified any underlying effects from confounding or biases; therefore, results must be interpreted accordingly. In summary, the longitudinal evidence has also produced conflicting findings. However, the degree to which these findings can be explained by the relationship between anger and blood pressure is constrained by methodological inconsistencies between studies.

To summarize, the literature on anger expression inward and blood pressure is largely divided between studies reporting a positive association and studies reporting no association. However, based on raw number of studies, there may be a stronger evidence for a positive relationship. This has been outlined by both cross-sectional and, to a lesser degree, longitudinal research.

2.2.4.2 Anger Expressed Out

To review, anger expression outward addresses how often angry feelings are expressed using verbally or physically aggressive behaviours. Guided under Alexander's original 1939 hypothesis – where anger expression outward was an adaptive behaviour that decreased blood pressure – anger expressed outward has traditionally been investigated for a negative association with blood pressure. However, as will be discussed, compared to anger expression inward, the evidence emerging from this field has been more varied.

Anger Expressed Out: Negative Relationship

Six cross-sectional studies support Alexander's original hypothesis.^{24, 50, 52, 53, 76, 115} These 6 studies are methodologically fairly uniform. In their primary analyses, three studies evaluated associations between anger expression outward and blood pressure as continuous variables^{24, 76, 115} while 3 dichotomized anger and examined association with blood pressure levels.^{50, 52, 53} All used reliable blood pressure measurement techniques. However, 4 studies did not use the STAXI, instead opting for either the Harburg Anger-In/Anger-Out Scale^{50, 52, 53} or the Behavioural Anger Response Questionnaire.⁷⁶ Thus, their findings should be interpreted

accordingly. In terms of racial distribution, there was a slight favour for Caucasian subjects. With respect to sex/gender, approximately half of the studies included only male subjects with the other half including at least 50% males. Therefore, an underlying sex bias may exist. But overall these 6 cross-sectional studies are relatively similar.

The studies presented here are strictly those that have supported a negative association between anger expression outward and blood pressure. Besides the use of different anger assessment instruments, these studies are relatively homogenous. However, generalizability of results to females is questionable due to the low number of female participants studied.

Anger Expressed Out: Positive Relationship

Four studies have reported a positive association between anger expression outwards and blood pressure. This consists of 3 cross-sectional^{57, 72, 77} and 1 longitudinal study.⁶² All used the STAXI and reliable blood pressure measurement techniques. Outcomes included associations between anger and blood pressure^{57, 77} as well as anger differences between hypertensive classification groups.⁷² The longitudinal study has provided the most robust evidence for a positive association; every 1-point increase in anger expressed outward corresponded to a 12% increased risk for hypertension after 4 years of follow-up.⁶² While racial/ethnic distributions were relatively even between studies, sex/gender was not. The 2 largest studies (one of which was the longitudinal cohort) included only middle-aged males;^{62, 77} therefore, it is unclear if this relationship would be observed among females. This potential male bias is reinforced by one of the mixed-sex cross-sectional studies which only reported an association among Caucasian males.⁵⁷ Taken together, these 4 studies suggest that anger expression outward is positively associated with blood pressure, possibly interacting with sex/gender and affecting males more than females. The lack of female participants limits the generalizability of results. Results should be interpreted accordingly.

Anger Expressed Out: No Relationship

Slightly more research has reported no relationship between anger expressed outward and blood pressure. This evidence consists of 7 studies: 2 longitudinal^{61, 69} and 5 cross-sectional.^{58, 67, 70, 71,}

From the cross-sectional research, 1 study examined blood pressure and anger scores as continuous variables⁷¹ and 4 dichotomized anger expression outward or blood pressure and examined levels between groups.^{58, 67, 70, 99} All but 1 study used the STAXI,⁷¹ all but 2 reported the use of reliable blood pressure measurement techniques^{67, 71} and all but 2 included both sexes/genders⁹⁹ (30/33 male⁵⁸). In terms of race/ethnicity, approximately three quarters of the participants studied were Caucasian. Given the generally consistent use of reliable blood pressure measurement techniques, validated anger questionnaires and inclusion of male and female study participants these studies outline some of the valid cross-sectional evidence reporting no association between anger expressed outward and blood pressure, at least among Caucasian subjects.

Once more, the evidence from the longitudinal research is less robust.^{61, 69} This is attributed in part to the hypothesis generating findings from Julius et al. who examined anger and blood pressure through post-hoc analyses.⁶¹ Due to this arrangement, their findings cannot be considered in a conclusive context. The remaining longitudinal study (which did arrange its analysis a priori) reported no association between anger expressed outward and blood pressure among female participants (97% Caucasian) after 3 years of follow-up.⁶⁹ Although these results are more robust compared to cross sectional evidence, 1 longitudinal study on Caucasian females cannot determine the direction for the field.

Anger Expressed Out: Summary

To summarize, 6 studies reported a negative association between anger expressed out and blood pressure, 4 reported a positive association and 7 reported no association. The 6 studies reporting a negative association were all cross-sectional. Among the 4 studies reporting a positive association 3 were cross-sectional and 1 was longitudinal. Finally, among the 7 studies reporting no association, 5 were cross-sectional and 2 were longitudinal. Based on an appraisal of the analysis approach used in one of the no association longitudinal studies, it was determined that only 1 longitudinal study provided acceptable evidence against an association between anger expressed outward and blood pressure.⁶⁹ Overall, the methodological quality, which refers to type of blood pressure measurement technique and self-report anger questionnaire as well as appropriateness and generalizability of sample population under study, was relatively consistent between groups (where ‘groups’ refers to studies reporting a negative association vs. a positive

association vs. no association). Exceptions to this were the negative association and positive association studies that studied more males than females. Only among the 7 no association studies was there more equal representation of males and females. Taken together, this trend may outline an interaction with sex/gender whereby a relationship between anger expression outward and blood pressure (be it positive or negative) is only found among males. Conflicting findings between the positive relationship and negative relationship studies remain to be clarified. Thus, at this time, no clear direction for the field can be discerned.

2.2.5 Synthesizing the Evidence

Overall, anger (as measured by its respective subcomponents) may be associated with blood pressure. However, this is not consistent across each subcomponent. For some subcomponents, the evidence is inconclusive. A summary of each anger construct and its relationship to blood pressure is presented below.

State Anger

State anger appears to be, at best, weakly positively associated with blood pressure. Research examining associations between state anger and blood pressure when subjects are in an angry state is necessary to determine whether an association exists with blood pressure. From a longitudinal perspective, it would also be of interest to determine whether repeated exposure to (or greater) state anger is associated with a significant increase in blood pressure over time.

Trait Anger

Trait anger appears to be positively associated with blood pressure. However, this relationship may be moderated by other covariates such as sex/gender and race/ethnicity. Associations between these 2 variables cannot be confirmed until prospective longitudinal cohort and experimental studies, particularly those that manipulate anger, are conducted on both sexes.

Anger Control

Two studies examining the association between anger control and blood pressure reported conflicting results. Both studied male-only sample populations. Much more research is required to clarify these findings.

Anger Expression (inward and outward)

The literature is almost evenly divided with respect to either a positive association or no association between anger expressed inward and blood pressure. The literature on anger expressed outward is also divided but between all relationship types: negative, positive or no association. While there are a greater number of no association studies in the anger expressed outward research (7 studies vs. 6 and 4 studies in the other categories), the uneven distribution of male and female subjects between the categories outline a potential sex-bias. Therefore, the available anger expression evidence, both inward and outward, is inconclusive with respect to either supporting or refuting a significant association with blood pressure.

Literature Gaps

Several literature gaps emerge when considering the collective effects of anger on blood pressure.

First is the limited or inconsistent consideration of race/ethnicity and gender/sex as covariates in many research models. It is understood that elevated blood pressure is more common among males than females until females reach menopause¹¹¹ and that elevated blood pressure more is common among African American populations.¹¹⁶ There is also evidence suggesting that African Americans and Caucasians do not score the same on validated anger questionnaires; a finding that interacts with sex/gender on some occasions.^{54-57, 117} Based on this information, the consideration of sex/gender and race/ethnicity is necessary to better understand the relationship between anger and blood pressure. This view has been shared by many others.^{73, 99, 118-123}

Second is the use of non-validated anger questionnaires and reliable blood pressure measurement techniques. The use of unreliable tools raises the question of reliability of results. Moreover, poor or differing methodology makes comparison of results between studies very difficult. With respect to blood pressure, it has been reported that compared to standardized blood pressure assessments, non-standardized manual blood pressure measurement techniques can change the hypertension–normotension classification of blood pressure in over 50% of persons.^{124, 125} This evidence underscores the critical need for reliable, standardized measurement techniques, both for blood pressure (i.e., office blood pressure assessed with an automated device such as the BpTru) and psychosocial functioning (i.e., validated questionnaires).

The third literature gap is the lack of prospective longitudinal cohort and experimental research; in particular, research that manipulates the independent psychosocial variables of interest. Currently, the majority of the literature has been based on cross-sectional research where measures are obtained at one point in time and with no manipulation of independent anger predictor variables of interest. These study designs do not provide the strongest evidence of causality. Prospective longitudinal cohort and experimental studies that manipulate anger and obtain measures at more than one time point would enable a more rigorous evaluation of these relationships. This lack of more robust evidence remains as a barrier to determining the existence and validity of a relationship between anger and blood pressure.

To summarize, literature gaps to the field of anger and blood pressure research include:

- A lack of consideration for potential racial/ethnic and sex/gender effects
- A lack of consistent use of validated and reliable self-report anger questionnaires
- A lack of consistent use of reliable blood pressure measurement techniques
- A lack of research based on prospective longitudinal cohort or experimental design; in particular, research that manipulates independent psychosocial variables of interest

These limitations must be addressed in future research in order to determine the existence and validity of a relationship between anger and blood pressure.

3 Perceived Stress & Blood Pressure

3.1 Historical Context & Definitions

The internalization of psychological stress has been argued as the most relevant for studying the psychobiological effects of stress on the body.¹²⁶⁻¹²⁹ More specifically, that “how an individual appraises stress, rather than the objective occurrence of stressful events, is what determines one's response to stressors”.^{126, 127, 129} While external measures of stress are objective and easier to measure (i.e., a stressful event either occurred or did not occur) they cannot address the issue that stressful events elicit varying responses from individuals. For example, personality factors such as a strong commitment to self, an attitude of vigorousness toward the environment, a sense of meaningfulness and an internal locus of control (collectively referred to as “hardiness”) have been identified as factors that predict illness onset due to stressful life events.¹³⁰ This evidence

outlines the existence of innate factors that trigger different internal stress responses and, moreover, that objective measures of stress could not have predicted the outcomes. For these reasons, perceived stress is an appropriate type of stress for investigating the contribution of stress to disease processes.

Richard Lazarus was the first to address appraised stress in 1966 using a conceptual psychological framework called the ‘transactional model of stress.’¹²⁶ Over time, through combined and sometimes competing efforts, Richard Lazarus and Sheldon Cohen contributed most significantly to our current understanding of appraised stress.^{127-129, 131} Both teams developed tools to measure appraised stress including the Ways of Coping Checklist published in 1980 by the Lazarus team,¹³² the Hassles and Uplifts Scales published in 1981 by the Lazarus team¹³³ and the Perceived Stress Scale (PSS) published in 1983 by the Cohen team.¹²⁹ The Ways of Coping Checklist was a 68-item questionnaire, the Hassles Scale was a 117-item ‘hassles’ questionnaire, the Uplifts Scale was a 135-item ‘uplifts’ questionnaire and the PSS was a 4, 10 or 14-item perceived stress questionnaire (depending on the version). Together, the development of these questionnaires enabled researchers to begin measuring and understanding the contribution of appraised stress to different disease processes.

While all questionnaires measured some type of appraised stress and contributed to our current understanding of the concept, the PSS was unique in its brevity. The PSS was a 4, 10 or 14-item self-report questionnaire designed to measure the “degree to which situations in one’s life appear[s] as stressful”.¹²⁹ More specifically, it was designed to measure how unpredictable, uncontrollable, and overloaded individuals appraised their lives to be. The PSS was sensitive to a variety of stressors, including “chronic stress deriving from ongoing life circumstances...stress from expectations concerning future events...stress from events not listed on a particular life-events scale, and to reactions to the specific events included on any scale”.¹²⁹ The questionnaire was brief, asked general questions and only required a junior high school education to be completed.

As the PSS reliably measured appraised stress using a brief, easily administrable questionnaire the PSS gave researchers the opportunity to more easily study the contribution of “nonspecific appraised stress [to] the etiology of disease and behavioural disorders”¹²⁹ including hypertension and cardiovascular disease. Since its publication, the PSS has been used successfully in this

regard, both for studying hypertension^{83, 84, 134, 135} and cardiovascular disease.^{86, 88} As a result of this work, perceived stress has been identified as an independent predictor of symptomatic coronary heart disease⁸⁷ and has demonstrated association with blood pressure when studied alone^{83, 84, 134, 135} and in conjunction with other measures of race-based discrimination.⁸⁹ Thus, in addition to reliably measuring the “degree to which situations in one’s life appear[ed] as stressful”,¹²⁹ the PSS enabled researchers to begin accurately studying the psychobiological effects of perceived stress on blood pressure.

Despite the brevity of the PSS questionnaire, the relationship between perceived stress and blood pressure has also been evaluated using non-validated perceived stress questionnaires. The decision to use non-validated perceived stress measurement tools over the validated PSS is rarely explained by authors. Non-validated perceived stress questionnaires are typically designed by researchers themselves, often consisting of a single question accompanied by a visual analog scale. Researchers may be inclined to use non-validated questionnaires due to easier administration and scoring. Support for these single-item stress questionnaires exists as several studies have cited positive associations between perceived stress and blood pressure;^{78-82, 85} The most famous stress study that has used this approach is the 2004 INTERHEART study.¹⁹ However, the contribution of these non-validated questionnaires has been both a benefit and a hindrance to the literature. The significant uptake and use of non-validated perceived stress questionnaires has increased research productivity in the field. However, the lack of theoretical background behind these non-validated questionnaires creates difficulty when attempting to assess what psychological construct these questionnaires are measuring. In summary, while the non-validated perceived stress literature supports an association between perceived stress and blood pressure, deriving clinical and scientific meaning from this literature is questionable.

Thus, to summarize, perceived stress addresses how an individual appraises the stress in his or her own environment. This type of psychological stress measurement is more appropriate than objective measures of stress as it considers the impact of the environment on an individual from the perspective of that individual. Research on appraised stress originated in 1966 with Richard Lazarus’s ‘transactional model of stress’.¹²⁶ Together, Lazarus and Cohen developed the concept of appraised stress as well as several questionnaires to measure the construct. Among these was Cohen’s Perceived Stress Scale. The combination of foundational research laid down by Lazarus and Cohen and the brevity/ease of administration of Cohen’s Perceived Stress Scale equipped

researchers with the necessary tools to effectively study the relationship between appraised stress and blood pressure. Both the PSS as well as non-validated perceived stress questionnaires have been used to evaluate the psychobiological effects of perceived stress on blood pressure. With half of the validated perceived stress literature and a majority of the non-validated perceived stress literature reporting a positive association with blood pressure, both questionnaire types have contributed to our understanding of the psychobiological effects of stress on the body and blood pressure.

3.2 Evidence to Date on Perceived Stress & Blood Pressure

Overall, the available evidence has reported a positive association between perceived stress and blood pressure. Limitations to the perceived stress literature are similar to those reported in the anger literature. This includes mixed results, varying outcome measures, lack of evidence based on prospective longitudinal cohort or experimental studies that manipulate perceived stress and lack of pathophysiological mechanistic explanations. However, of most significant concern to the perceived stress field is the overwhelming use of non-validated psychometric questionnaires. In order to address this issue, the following literature review has been divided into studies that have used the validated perceived stress measures and those that have not. A summary of all perceived stress findings can be found in Table 6.

3.2.1 Blood Pressure & Validated Measures of Perceived Stress

Since the development of Cohen's Perceived Stress Scale, 4 studies have examined the relationship between perceived stress and blood pressure. This has consisted of 3 cross-sectional studies^{83, 134, 135} and 1 longitudinal cohort.⁸⁴

Validated Perceived Stress Measures: Positive Association

Two of the 4 studies have reported a positive association between perceived stress as measured by the validated PSS and blood pressure. In 2011 Huang et al. evaluated PSS scores and nighttime blood pressure dipping pattern.⁸³ Non-dipping is known as the absence of a normal nighttime decrease in blood pressure. Absence of this pattern has been associated with hypertensive target organ damage among both hypertensives and normotensives.¹³⁶⁻¹³⁹ Stratifying 307 Chinese essential hypertensives (52% male) as either dippers or non-dippers, it was found that non-dippers were more likely to report a higher stress status. Similarly, decline in

nighttime systolic or diastolic blood pressure was inversely related to PSS score. The second study reporting a positive association between PSS and blood pressure was the longitudinal Spokane Heart Study.⁸⁴ One hundred and seventy male subjects asymptomatic for cardiovascular disease were evaluated at 2-year intervals for at least 6 years. The authors found that higher PSS scores were associated with higher levels of diastolic blood pressure at follow-up as measured by standardized manual blood pressure measurement. These 2 studies outline the current evidence in support of a positive association between perceived stress and blood pressure.

Validated Perceived Stress Measures: No Association

Two studies have found no association between PSS scores and blood pressure. In a recent cross-sectional study of 102 Korean Americans (40% male); PSS scores were not correlated with systolic or diastolic blood pressure when measured by an automated device.¹³⁴ A second cross-sectional study on 100 university students (38% male), who participated in a mental arithmetic task, also reported no relationship between PSS scores and change in systolic or diastolic blood pressure.¹³⁵

Validated Perceived Stress Measures: Summary

Among the studies that used Cohen's validated PSS questionnaire, 2 studies reported a positive association and 2 studies reported no association. However, closer inspection of these studies suggests an overall positive association between perceived stress (as measured by the validated PSS) and blood pressure. This conclusion is based on larger sample sizes and use of a longitudinal cohort design in 1 of the 2 positive studies. A lack of generalizability from 1 of the negative studies on American university students reinforces the above-mentioned conclusion.¹³⁵ However, due to the low number of studies, much more research remains to be completed in order to determine the existence and validity of a relationship between perceived stress as measured by the PSS and blood pressure. Narrow racial/ethnic diversities also cannot rule out possible underlying racial/ethnic effects. Future efforts should focus on the inclusion of subjects from more diverse racial/ethnic backgrounds, as well as continued recruitment of male and female study participants.

3.2.2 Blood Pressure & Non-Validated Measures of Perceived Stress

Ten studies have investigated associations between perceived stress as measured by a non-validated perceived stress questionnaire and blood pressure. This includes 1 longitudinal⁸⁵ and 9 cross-sectional studies.^{78-82, 140-143}

Non-Validated Perceived Stress Measures: Positive Association

Six studies have reported a positive association between perceived stress and blood pressure. This evidence consists of 1 longitudinal⁸⁵ and 5 cross-sectional studies.⁷⁸⁻⁸²

Examining the 5 cross-sectional studies in more detail, it can be seen that 2 evaluated blood pressure as a continuous variable^{78, 82} and 3 made perceived stress a categorical variable and studied blood pressure levels between 2 groups for their primary analyses.⁷⁹⁻⁸¹ All studies assessed perceived stress using a 1 item questionnaire (i.e., 0 = low stress to 10 = high stress) and recruited large sample sizes across a wide range of subject demographics. All reported the use of reliable blood pressure measurement methods (4 studies used ambulatory and 1 used home blood pressure monitoring). One study assessed blood pressure and perceived stress simultaneously at 30-minute intervals using daytime ambulatory blood pressure monitoring.⁸¹ Based on this information, these 5 cross-sectional studies provide convincing evidence in support of a positive association between perceived stress (as measured by non-validated questionnaires) and blood pressure. At minimum, this evidence sets the stage for further investigation with the use of validated perceived stress questionnaires.

One longitudinal study also reported a positive association. Perceived stress and risk of hypertension development were studied using prospectively collected cohort data from the Copenhagen Heart Study.⁸⁵ Following 7066 male and female subjects for approximately 10 years, the authors found that high perceived stress was associated with a greater risk of hypertension diagnosis at follow-up compared to low perceived stress. The authors measured perceived stress in terms of stress intensity and stress frequency using a 2-item questionnaire. Stress scores were then categorized into low, medium and high perceived stress. The large sample size and substantial follow-up time reinforce these results and suggest that non-validated

perceived stress could play some role in increasing the long-term risk for hypertension development.

Non-Validated Perceived Stress Measures: No Association

Three cross-sectional studies have reported no association between perceived stress and blood pressure.^{140, 141, 143} Two studies assessed perceived stress based on a single question^{140, 141} and 1 study developed more elaborate questionnaires.¹⁴³ All studies except 1 used a reliable method of blood pressure measurement.¹⁴³ The sample populations under study are more heterogeneous compared to the positive association studies. Nonetheless, these 3 studies outline that not all of the non-validated perceived stress literature supports a positive association between perceived stress and blood pressure.

Non-Validated Perceived Stress Measures: Negative Association

One cross-sectional study reported a negative association between perceived stress and blood pressure.¹⁴² The authors measured perceived stress among 1601 Caucasian males using a 1-item questionnaire. While drawing conclusions based on 1 study is difficult, this evidence does outline the existence of different types of relationships between perceived stress as measured by non-validated questionnaires and blood pressure.

Non-Validated Perceived Stress Measures: Summary

Overall, more studies using non-validated measures of perceived stress reported positive associations between perceived stress and blood pressure (6 out of 10 studies, including 1 longitudinal study). The methodological quality of positive studies was generally higher than the negative or no association studies. However, the lack of a validated perceived stress questionnaire makes generalizing and comparing results very challenging. At a minimum, these studies have set the stage for further investigation with validated perceived stress questionnaires. Given the narrow racial/ethnic diversity of studies in this section (due in part to lack of reporting), it is possible there are underlying racial/ethnic effects mediating these relationships. Future efforts should focus on the inclusion and reporting of subjects from more diverse racial/ethnic backgrounds and continue recruitment of male and female study participants.

3.2.3 Synthesizing the Evidence

Based on evidence from both the validated and non-validated perceived stress literature, it appears that perceived stress is positively associated with blood pressure in some capacity. This conclusion aligns with what is observed in the cardiovascular literature.⁸⁶⁻⁹⁰ However, this conclusion has primarily been based on cross-sectional research where the independent variable (perceived stress) was not manipulated and variables were measured only once. Thus, these results must be interpreted accordingly. Prospective longitudinal cohort and experimental studies that employ more robust methodology and manipulate the independent psychosocial variables of interest are necessary to clarify this literature. The existence of underlying racial/ethnic effects is still unclear. Therefore, future efforts should also focus on the inclusion of subjects from diverse racial/ethnic backgrounds, as well as continued recruitment of male and female study participants.

Literature Gaps

Based on the evidence presented above, the major literature gaps facing the perceived stress and blood pressure field are:

- A lack of research based on validated perceived stress questionnaires
- A lack of research based on prospective longitudinal cohort or experimental design; in particular, research that manipulates independent psychosocial variables of interest
- A lack of reporting/enrolment of subjects from diverse racial/ethnic backgrounds

These limitations must be addressed in future research in order to confirm the existence and validity of a relationship between perceived stress and blood pressure

4 Moving Forward: Prospective Longitudinal Cohort Designs

The preceding sections have reviewed the literature on the relationships between anger, perceived stress and blood pressure. Almost all of this literature has been based on cross-sectional data that has not manipulated independent psychosocial variables of interest or measured variables at more than one point in time. The major obstacle faced by the current literature is the inability to establish whether change in psychosocial functioning is indeed

associated with change in blood pressure. More specifically, the lack of strong study designs and manipulation of psychosocial predictor variables make it difficult to provide strong evidence of causality. Thus, in addition to the overwhelming division reported in the literature, the restriction to cross-sectional observational studies further prevents the formulation of robust conclusions.

However, study designs such as prospective longitudinal cohort designs that manipulate independent psychosocial variables of interest, could be used to test these relationships more directly. More specifically, by manipulating anger or perceived stress it would be possible to evaluate whether changes in psychosocial variables are associated with changes in blood pressure. Therapeutic psychological interventions are one tool by which anger or stress could be manipulated in a prospective longitudinal cohort setting. Preliminary evidence from the literature has outlined the potential use of therapeutic psychological interventions for this purpose.

4.1 Therapeutic Interventions

Therapeutic Interventions: Anger

Preliminary evidence exists in support of using therapeutic psychological interventions to test the relationship between anger and blood pressure. Five studies that used anger management, relaxation or stress reduction therapies demonstrated that improvements in anger were associated with improvements in blood pressure.¹⁴⁴⁻¹⁴⁸

Among the anger management research, it was found that male and female subjects with mild essential hypertension who completed a 6-week anger management program demonstrated significantly lower casual diastolic blood pressure compared to no-treatment controls.¹⁴⁷ Two other studies demonstrated that concurrent decreases in verbal and physical aggressive behaviours and increases in constructive anger verbal behaviour successfully reduced resting ambulatory blood pressure.^{144, 146} Of note, one of these 2 studies was also directly interested in clarifying the relationship between psychosocial risk factors and blood pressure.¹⁴⁴ Finally, through the use of stress and type-A management interventions, unmedicated mildly hypertensive male subjects demonstrated reductions in systolic blood pressure reactivity that were associated with reductions in measures of anger expression inward and expression outward.¹⁴⁸ Together, this evidence outlines the utility of therapeutic psychological interventions such as anger management to study the relationship between anger and blood pressure.

Relaxation therapy has also been used effectively to manipulate anger to evaluate its association with blood pressure.¹⁴⁵ One study enrolled male subjects and allocated them to either relaxation or no-treatment control. Reductions in anger-related hostility were significantly associated with reductions in standing systolic blood pressure. Interestingly, these results were most significant for subjects high in norepinephrine, suggesting a possible pathophysiological link between relaxation, anger and blood pressure. This was also one of the few studies directly interested in clarifying the relationship between psychosocial risk factors and blood pressure. This relaxation intervention study provides further support for the use of therapeutic psychological interventions in a prospective longitudinal cohort setting to study the relationship between anger and blood pressure.

Therapeutic Interventions: Perceived Stress

One study has provided evidence supporting the use of therapeutic psychological interventions to study the relationship between perceived stress and blood pressure. In 2003, McCraty et al. evaluated the effect of a workplace stress-reduction program on emotional health and blood pressure among 38 hypertensive employees.¹⁴⁹ The authors reported that decreases in stress symptoms were significantly correlated with reductions in systolic blood pressure as measured by an automated device. Although limited in evidence, this preliminary study provides support for using a psychological therapeutic intervention to test the relationship between perceived stress and blood pressure.

Therapeutic Interventions: Summary

Preliminary evidence has outlined the appropriateness of using a therapeutic psychological intervention as a tool to manipulate stress and anger in order to more directly test their association with blood pressure. Limitations to the current evidence include its preliminary nature, lack of consistent use of validated self-report questionnaires, lack of reliable blood pressure measurement techniques and lack of standardized therapies. Nonetheless, this information provides the necessary framework for further testing with more rigorous methodology.

4.2 Mindfulness-Based Stress Reduction

Mindfulness-Based Stress Reduction (MBSR) is a standardized group stress reduction therapy that could be used as a tool to test the association between anger, stress and blood pressure.

In 1985, Jon Kabat-Zinn introduced MBSR to treat chronic pain.¹⁵⁰ Today, MBSR is used to also treat anxiety,¹⁵¹⁻¹⁵⁵ depression,^{151, 154, 156-159} fibromyalgia,¹⁵⁹⁻¹⁶¹ binge-eating¹⁶² and psoriasis.¹⁶³ Currently, MBSR can be found across Canada at major hospitals and local community health centers for the treatment of the abovementioned conditions. MBSR teaches individuals mindfulness “as an approach to adapt to stress, pain and illness”.¹ Improved mindfulness enables individuals to “more clearly see the full context of a situation, have access to a broader range of emotional responses and therefore cope with stressful situations more effectively”.^{1, 153} In addition to the above-mentioned conditions, MBSR has also demonstrated efficacy in improving components of anger¹⁶⁴⁻¹⁶⁶ and perceived stress¹⁶⁷⁻¹⁷¹ when measured using validated questionnaires. The psychological construct known as “mindfulness” has been proposed as a mediating mechanism for improving both perceived stress^{170, 172} and anger.¹⁷³⁻¹⁷⁵

MBSR can be summarized as an established, standardized therapy with evidence documenting its efficacy for improving anger and perceived stress. Combining this information with previously discussed evidence citing the success of other therapeutic psychological interventions illustrates that MBSR has potential to be used in a prospective longitudinal cohort setting to more directly test associations between anger, perceived stress and blood pressure.

4.3 Summary

To summarize, preliminary evidence exists in support of using therapeutic psychological interventions to test the relationship between anger, perceived stress and blood pressure. Mindfulness-based stress reduction is a potential tool that could be used to manipulate anger and perceived stress in a prospective longitudinal cohort setting. Using MBSR might clarify the existence and validity of a relationship between anger, perceived stress and blood pressure.

5 Rationale & Hypothesis

5.1 Rationale

Evidence exists suggesting an association between anger, perceived stress and blood pressure. However, much of this evidence is divided and has been based on cross-sectional research measured at one point in time and with no manipulation of independent psychosocial variables of interest. Prospective longitudinal cohort research that includes an intervention designed to impact anger and perceived stress would add more robust evidence to the literature and may help to clarify currently divided findings.

Therapeutic psychological interventions are one tool by which the relationship between anger, perceived stress and blood pressure can be tested more directly in a prospective longitudinal cohort setting. Preliminary evidence has outlined the appropriateness of this approach. MBSR is one intervention that could be used in a prospective longitudinal cohort study of this kind. MBSR was previously tested prospectively for its efficacy in blood pressure lowering therefore providing an opportunity to test the relationship between anger, perceived stress and blood pressure.

This study used MBSR as a tool to manipulate anger and perceived stress to evaluate their association with blood pressure. More specifically, the objective of this study was to determine the existence of an association between change in anger (measured by the State-Trait Anger Expression Inventory) and change in perceived stress (measured by Cohen's Perceived Stress Scale) with change in ambulatory blood pressure among unmedicated stage-1 hypertensive individuals who underwent a standard 8-week MBSR program.

Findings from this prospective longitudinal cohort study will contribute to the current understandings of anger and perceived stress as suspected risk factors for hypertension development.

5.2 Hypothesis & Objectives

The objective of this study was to establish the existence of an association between change in anger and perceived stress with change in ambulatory blood pressure among untreated stage-1 hypertensive subjects who underwent a standard 8-week MBSR protocol.

It was expected that associations between psychosocial stressors and blood pressure would occur while subjects were awake, undergoing daily activities and managing psychosocial stressors. Therefore, daytime and 24-hour ambulatory blood pressures were selected as primary outcome measures in this study. The primary hypothesis tested was that change in psychosocial variables thought to be linked to blood pressure (trait anger, trait anger temperament, trait anger reaction, anger expressed in, anger expressed out and perceived stress) would demonstrate an association with change in daytime and 24-hour ambulatory blood pressure among untreated stage-1 hypertensive subjects who underwent a standard 8-week MBSR protocol.

Based on the possibility that blood pressure could be affected by anger or stress while subjects slept and subconsciously replayed the events of their day through their mind, the secondary hypothesis tested whether change in the above-mentioned psychosocial variables would demonstrate an association with change in nighttime blood pressure among untreated stage-1 hypertensive subjects who underwent a standard 8-week MBSR protocol.

Three anger constructs (anger control in, anger control out and the anger expression index) received less support from the literature. Thus, they were investigated for exploratory purposes. The specific exploratory hypotheses tested for the existence of an association between change in exploratory anger measures (anger control in, anger control out and the anger expression index) and change in daytime, 24-hour and nighttime blood pressure among untreated stage-1 hypertensive subjects who underwent a standard 8-week MBSR protocol.

Additional exploratory hypothesis tested for the existence of sex differences by re-testing all of the abovementioned hypotheses after stratifying by sex.

State anger was not investigated in this study due to the specific situational nature of the construct and the lack of support from the literature.

Test of the hypotheses are outlined in the Methods section in the next chapter.

Chapter 2 Methods

6 Study Design

Subjects included in this thesis study were from the HARMONY trial (**H**ypertension **A**nalysis of stress **R**eduction using **M**indfulness meditati**ON** and **Y**oga). The original HARMONY study was a prospective wait-list randomized controlled trial designed to test the use of mindfulness based stress reduction (MBSR) for blood pressure lowering among stage-1 unmedicated hypertensive subjects. The specific objective of HARMONY was to examine the effect of a standard 8-week MBSR therapy program on ambulatory blood pressure among stage-1 unmedicated hypertensives, as has been described.¹

Using a prospective longitudinal cohort within-group design with two time points (baseline and after MBSR), this thesis study used data from the HARMONY study to test the above-mentioned hypotheses. The specific objective of this study was to examine the existence of an association between change in anger and perceived stress with change ambulatory blood pressure among untreated stage-1 hypertensive subjects who underwent a standard 8-week MBSR protocol.

Based on the understanding that behavioural interventions often result in small to moderate effect sizes, and that all participants in the HARMONY study underwent all measurements and participated in the mindfulness intervention, the within-group design was selected to increase the sample size and thus the possibility of detecting an association. Thus while HARMONY was conducted as a wait-list randomized controlled trial, this thesis evaluated all subjects before and after MBSR.

7 Participants

Participants for the HARMONY study were recruited from referring physicians, advertisements in local newspapers and posters at local hospitals. Eligible subjects were between the ages of 20 – 75 years of age. Main inclusion criteria included a diagnosis of stage-1 hypertension as confirmed by ambulatory blood pressure monitoring. Specific ambulatory blood pressure eligibility criteria included daytime ambulatory blood pressure $\geq 135/85$ mmHg or 24-hour

ambulatory blood pressure $\geq 130/80$ mmHg. Subjects could qualify based on either their systolic or diastolic blood pressure.

In addition to a stage-1 diagnosis, subjects were required to be untreated (no antihypertensive medication) for at least 6 months prior to the baseline screening visit. Additional exclusion criteria included a diagnosis of diabetes, secondary hypertension or target organ damage. Labs and echocardiograms were collected to rule out target organ damage and diabetes. Study requirements also included a willingness and ability to participate in the MBSR program and attend all necessary study visits and safety blood pressure evaluations. More detailed study inclusion and exclusion criteria can be found in Table 7.

8 Screening & Blood Pressure Measurement

All study visits took place at Sunnybrook Health Sciences Centre, Toronto, Ontario. All MBSR classes were conducted at Toronto General Hospital, Toronto, Ontario. Study subjects were not given incentive for participation in the study; however, they were reimbursed for parking expenses. All experimental protocols and informed consent processes were approved by Research Ethics Boards at both institutions and conducted according to the principles of Good Clinical Practice and the Declaration of Helsinki.

Office blood pressure measurements were performed by trained research staff using an automated blood pressure measurement device (BpTru Limited., Coquitlam, BC, Canada). Six blood pressure readings were taken at 2-minute intervals; the first reading was discarded with the average of the last 5 representing the average seated office automated blood pressure. Office automated blood pressure was used to screen for stage-1 hypertension. All other office blood pressure checks were for blood pressure safety monitoring purposes.

Ambulatory blood pressure monitoring determined study eligibility via either baseline daytime or 24-hour blood pressure results (Model 90207, Spacelabs Medical Inc., Redmond, WA). Subjects wore an ambulatory blood pressure monitor for 24 hours. Blood pressure readings were taken at 15-minute intervals during the day (07:00 – 11:00) and at 30-minute intervals at night (11:00 – 07:00). Daytime and nighttime ambulatory blood pressure readings were adjusted according to participant diaries. Research staff were trained on all proper blood pressure

measurement protocols and regularly supervised. Only cuff sizes appropriate for subjects' arm circumferences were used.

9 Study Flow

At study entry all participants underwent anthropomorphic measurements and were administered Cohen's Perceived Stress Scale (PSS) and the State-Trait Anger Expression Inventory-2 (STAXI-2). All subjects were scheduled to begin MBSR within 4 – 12 weeks of the baseline visit. Participants received no mindfulness training during the wait-list period. All subjects returned after MBSR and underwent ambulatory blood pressure monitoring, office blood pressure measurements, anthropomorphic measurements and completed the PSS and STAXI-2. Following this, participants began a follow-up period and returned 12 weeks after MBSR for a final close out ambulatory blood pressure monitoring.

10 Intervention

The MBSR program implemented in the HARMONY study has been described elsewhere but is explained below.¹ MBSR is a “multi-component group therapy that provides systematic training in mindfulness meditation as a self-regulation approach to stress reduction and emotion management. [It] provides training in formal meditation approaches with the primary goal of cultivating psychological resilience and resistance to stress. This is achieved through fostering the quality of “mindfulness”, defined as the capacity to intentionally be in the present moment without judgment”.^{1, 176} The therapy “teaches attendees to approach stressful situations “mindfully”; allowing them to identify what is occurring in their bodies and minds and to step back from thoughts and feelings during stressful situations. This affords participants the opportunity to make wise choices with respect to managing stressful situations, such as choosing to avoid engaging in anxious worry that may otherwise escalate into a cycle of stress reactivity and contribute to more emotional distress”.^{1, 150, 176-178}

In the study, MBSR was delivered over a 9 week period, consisting of one introductory session and 8 weekly 2.5 hour sessions. All MBSR therapy was administered at Toronto General Hospital. There was also a 6 hour silent retreat held on a Saturday or Sunday between the 6th and 7th week of the program. The MBSR program “incorporate[d] four major therapeutic elements: formal meditation, informal mindfulness practice (such as bringing mindfulness to daily

activities), psycho-educational activities, and self-monitoring/reflection exercises”.¹ Each week participants learned new techniques to assist with cultivating the state of mindfulness. Techniques included mindfulness meditation, mindful walking, mindful eating, loving-kindness meditation, yoga etc. In addition to physical activities, each class included group discussion. Progress through the MBSR program was captured via weekly homework logs which were faxed weekly from Toronto General Hospital to Sunnybrook Health Sciences Centre.

11 Outcomes

11.1 Primary

The primary outcome measure of this thesis study was post intervention 24-hour and daytime systolic and diastolic blood pressure as measured by 24-hour ambulatory blood pressure monitoring. Additional measures included post-intervention perceived stress and anger as measured by the PSS and STAXI-2. The primary objective of this study was to establish whether changes in primary predictor variables (post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) were related to changes in daytime or 24-hour blood pressure after participants completed an 8-week MBSR program.

11.2 Secondary

The secondary outcome measure was post-intervention nighttime blood pressure as measured by 24-hour ambulatory blood pressure monitoring. Secondary objectives included establishing whether changes in primary predictor variables (post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) were related to changes in nighttime blood pressure after participants completed an 8-week MBSR program.

11.3 Exploratory

Post-intervention daytime, 24-hour and nighttime blood pressure were also used as outcome measures for the exploratory analyses. The exploratory objectives included establishing whether change in exploratory predictor variables (post intervention anger control inwards, anger control

outwards and the anger expression index) were related to change daytime, 24-hour and nighttime blood pressure.

Additional exploratory objectives included establishing the existence of sex any differences.

12 Measures

12.1 State Trait Anger Expression Inventory (STAXI)

The STAXI is a validated self-report questionnaire that measures anger as outlined by Spielberger's theory: anger experience (state and trait anger) and anger expression (expression and control of anger). The STAXI is designed to provide "easily administered and objectively scored measures of the experience, expression and control of anger for adolescents and adults".² Completion of the STAXI requires 12 – 15 minutes and a 6th grade reading level. The second version of the STAXI (the STAXI-2) was used in this study.

The STAXI-2 consists of 57 questions that use a 4-point Likert response format. The Likert response format ranges from *Not at all* (1) to *Very much so* (4) for the State Anger questions, and *Almost never* (1) to *Almost always* (4) for the Trait Anger, Anger Expression and Anger Control questions. Higher ratings reflect a greater intensity of angry feelings or higher frequencies to experience, express, suppress or control anger. In total, the questionnaire consists of 6 scales and 5 subscales (subscales are included in square brackets): State anger (S-Ang) [feeling angry (S-Ang/F), feel like expressing anger verbally (S-Ang/V), feel like expressing anger physically (S-Ang/P)], trait anger (T-Ang) [trait anger temperament (T-Ang/T), trait anger reaction (T-Ang/R)], anger expression in (AXI), anger expression out (AXO), anger control in (ACI), anger control out (ACO) and the anger expression index (AXX). A summary can be found in Table 8.

The 15-item State Anger scale measures the intensity of angry feelings and the extent to which a person feels like expressing anger at a particular time. As the state-anger subscale was not investigated in this study it will not be described in further detail. The Trait Anger scale includes two subscales, Angry Temperament and Angry Reaction. Trait Anger scale consists of 10 questions that measure how often angry feelings are experienced over time. The Angry Temperament subscale consists of 4 questions that measure the disposition to experience angry feelings without provocation. The four-item Angry Reaction subscale measures the frequency

that angry feelings are experienced in situations that involve frustration and/or negative evaluations. The STAXI also includes 8-question sections on anger expression in which measures how often angry feelings are experienced but not expressed, anger expression out which measures how often angry feelings are expressed using verbally or physically aggressive behaviour, anger control in which measures how often a person attempts to control angry feelings by calming down or cooling off, and anger control out which measures how often a person controls the outwards expression of angry feelings. Finally, the Anger Expression Index combines the anger expressed in, anger expressed out, anger control in and anger control out scales to yield an omnibus measure of how one reacts or behaves when angry.

The STAXI-2 and its subscales have been extensively studied on normative and psychiatric samples and have demonstrated good internal consistency and convergent validity. Test-retest reliability of the STAXI-2 scales have also received support.^{179, 180} As reported by Spielberger,¹⁰⁴ alpha coefficients (measures of internal consistency) for state and trait anger were high across all scales and subscales (0.84 or higher) except for trait anger reaction which was 0.76 and 0.73 for normal females and males, respectively. In terms of convergent validity, Trait Anger was found to be highly correlated with the Buss-Durkee Hostility inventory total score¹⁸¹ and the Hostility¹⁸² and Overt Hostility¹⁸³ scales of the Minnesota Multiphasic Personality Inventory.¹⁸⁴ Both state anger and trait anger correlated with neuroticism and psychoticism scales from the Eysenck Personality Questionnaire¹⁸⁵ and the state and trait anxiety and curiosity subscales of the State Trait Personality Inventory.¹⁸⁶ Alpha coefficients for anger expression in, anger expression out, anger control in, anger control out and the anger expression index have also been reported to be adequate and uniform across all scales and subscales (0.73 or higher).² In terms of convergent validity, the anger expression scales have demonstrated correlations with other anger measures (such the Harburg Anger-In/Anger-Out Scale)⁹⁴ as well as other personality measures (such as the state and trait anxiety and curiosity subscales of the State Trait Personality Inventory).¹⁰⁴ In terms of construct validity, Spielberger also demonstrated an association between anger expression scores and written responses to hypothetical anger scenarios.²

12.2 Cohen's Perceived Stress Scale

Perceived stress was measured using the Perceived Stress Scale (PSS) developed by Cohen, Kamarck, and Mermelstein.¹²⁹ The PSS is the only empirically established index of general

stress appraisal. In this study, the 14-item version was administered to assess the degree to which participants appraised their current situations as unpredictable, uncontrollable or overwhelming. The PSS includes easy-to-understand questions about participants' stressful thoughts or feelings related to situations in their lives within the last month. Each item is rated on a 5-point Likert scale ranging from *Never* (0) to *Very Often* (4). A total PSS score is calculated by reverse-scoring the seven positive items and then summing scores for all 14 items. Questions 4, 5, 6, 7, 9, 10, and 13 are the positively stated items. PSS scores can range from 0 to 56. A junior high school education is required to take the PSS. As the PSS is not a diagnostic instrument there are no cut-offs;¹³¹ there can only be comparisons with other samples.^{129, 187}

The PSS has effectively assessed perceived stress among healthy^{129, 188} and psychiatric populations¹⁸⁹ and has been used in collaboration with external measures of stress such as cortisol.^{190, 191} With internal consistency scores (alpha coefficients) ranging from 0.84 to 0.86, the PSS has demonstrated high levels of reliability and validity.^{129, 187} This finding has been supported by other studies.¹⁹¹⁻¹⁹⁴ The PSS has demonstrated high convergent validity by correlating with specific life-event impact scores.¹²⁹ High construct validity has also been demonstrated with studies on smoking-cessation. More specifically, PSS scores have been positively associated with health, health service utilization, health behaviours, life satisfaction and help seeking.¹⁸⁷

12.3 Ambulatory Blood Pressure Monitoring

Twenty-four hour ambulatory blood pressure monitoring (ABPM) was used for the primary, secondary and exploratory outcome measures in this study. ABPM is referred to as the gold standard in blood pressure measurement for several reasons. First, is the reduction in measurement error due to the entirely automated measurement process. Second, is the elimination of observer bias as blood pressure is measured while subjects undergo their regular daily activities with no health care providers present. This type of blood pressure measurement reduces blood pressure variability and associated observer effects such as white coat and masked hypertension. Finally, by taking approximately 80 blood pressure measurements over 24 hours, ABPM reduces variability and distills a blood pressure average closer to an individual's 'true' blood pressure. Compared to other blood pressure measurement methods, ABPM results correlate much better with long term target organ damage and are better at predicting

cardiovascular risk.¹⁹⁵ ABPM results have also demonstrated association with various types of psychosocial functioning.^{109, 196}

13 Statistical Methods

13.1 Baseline Correlations

Comparisons of baseline data between male and female study subjects were evaluated using unpaired t-tests or chi-squared tests. Baseline correlations between psychosocial variables and daytime, 24-hour and nighttime blood pressure were evaluated using Pearson product moment zero order correlation matrices. Spearman ranked correlation matrices were used to evaluate any correlations with trait anger temperament due to right skewed data. The same analysis approach was used for all primary, secondary and exploratory analyses. All analyses were repeated after stratifying by sex.

13.2 Pre-Post Changes

Paired t-tests were used to examine changes from pre to post intervention for blood pressure and the psychometric variables of interest. 'Pre' and 'post' referred to baseline measures and measures taken after the MBSR program, respectively. All analyses were repeated after stratifying by sex. A sign test was used to evaluate pre-post differences of trait anger temperament due to its non-normal baseline distribution.

Anger scores were also dichotomized according to those who scored <75th percentile and those who scored $\geq$ the 75th percentile. According to work completed by Spielberger, "individuals with anger scores above the 75th percentile experience and/or express angry feelings to a degree that may interfere with optimal functioning"¹⁹⁷... The anger of these individuals may contribute to difficulties in interpersonal relationships or dispose them to develop psychological or physical disorders".² Furthermore, "high anger expression inwards scores, especially when associated with low anger expression outwards and high levels of anxiety, have been shown to be associated with elevated blood pressure and hypertension"^{57, 66, 198}... Very high anger scores (i.e., $\geq$ 90th percentile) on both the [anger expressed out] and [anger expressed in] scores have been associated with vulnerability to coronary artery disease and heart attacks".² Based on this information, anger scores were dichotomized to examine changes from pre to post intervention according to the 75th percentile threshold. McNemar's test for paired proportions was used to test

whether the proportion of those scoring $<$ or $\geq 75^{\text{th}}$ percentile on the anger measures changed from pre to post intervention. Cut-offs for the 75^{th} percentile or greater were taken from the STAXI-2 professional manual and applied to each category of analysis respectively (i.e., males and females, females only and males only). Exact values from the test have been reported. All analyses were repeated stratifying by sex.

13.3 Psychometric Properties

Cronbach's alpha was calculated to assess internal consistency.¹⁹⁹ Standardized values have been reported.

13.4 Change Correlations

Change correlations were evaluated by incorporating pre to post change scores into Pearson product moment zero order correlation matrices. This consisted of all psychosocial variables with daytime, 24-hour and nighttime blood pressure. Spearman ranked correlation matrices were used to evaluate any change correlations with trait anger temperament due to right skewed change score data. The same analysis approach was used for all primary and secondary analyses. All analyses were repeated after stratifying by sex.

13.5 Multivariate Linear Regressions

Primary multivariate linear regression analyses were used to evaluate whether psychosocial functioning (post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) was associated with ambulatory blood pressure (24-hour and daytime blood pressure), after adjusting for baseline blood pressure and baseline psychosocial values and other covariates.

Based on previous work from our lab the following variables were selected as covariates and included in all models²⁰⁰⁻²⁰²: age, sex (female), baseline body mass index (BMI), race (Caucasian=yes), family history of premature cardiovascular disease (yes), baseline drinking of ≥ 10 drinks/wk. (yes), education level $>$ post-secondary (yes), regular practice of relaxation at baseline (yes) and regular exercise at baseline (yes). Dependent variables in the primary analysis included daytime and 24-hour systolic and diastolic blood pressure. Dependent variables in the secondary analysis included nighttime systolic and diastolic blood pressure. Dependent variables

in the exploratory analysis included daytime, nighttime and 24-hour systolic and diastolic blood pressure. Baseline anger, perceived stress and blood pressure were included as covariates in all models to account for the Law of Initial Values.²⁰³

It should be noted that post intervention trait anger temperament scores were slightly skewed and not amenable to transformation. Therefore, a modified full main effects model was evaluated for exploratory purposes. In this modified model, post intervention trait anger temperament scores were stratified into quartiles and used as predictor variables in the model. This was done for exploratory purposes to compare findings with the main model.

Secondary multivariate linear regression analyses evaluated whether changes in primary psychosocial variables were associated with changes in nighttime blood pressure. Additional anger measures (specifically anger control inwards, anger control outwards and the anger expression index) were evaluated in the exploratory multivariate linear regression analyses to establish the existence of any associations with daytime, 24-hour or nighttime blood pressure. All analyses (primary, secondary and exploratory) were repeated after stratifying by sex. The same covariates were used in all analyses.

13.6 Effect Sizes

Standardized regression coefficients were generated to evaluate standardized effects of the primary predictor variables (trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) on the primary outcome variables (24-hour and daytime systolic and diastolic blood pressure). Standardized regression coefficients were also generated for any anger or stress predictor variables that emerged as statistically significant in the secondary or exploratory analyses.

13.7 Post Hoc-Analyses

Post-hoc analyses after study completion included evaluating changes in anthropomorphic measurements, drinking habits and exercise levels from pre to post intervention. This was accomplished using paired t-tests and McNemar's test for paired proportions.

13.8 Software

Statistical analyses were performed using SAS version 9.3 (SAS Institute Inc., Cary, NC).

Chapter 3 Results

14 Study Details

14.1 Participant Flow

In total 101 subjects were included in the study. Four hundred and eighty-three individuals were screened for study eligibility and 254 were excluded by telephone. The most common reason for telephone exclusion was current or recent antihypertensive therapy. From the remainder, 51 declined to participate and 77 were not stage-1 hypertensive upon ambulatory blood pressure monitoring.

Of the 101 subjects enrolled in the study 99 completed a pre intervention ambulatory blood pressure monitoring, 96 completed a pre intervention STAXI questionnaire and 90 completed a pre intervention PSS. Eight-five subjects completed a post intervention ambulatory blood pressure monitoring, 76 subjects completed a post intervention STAXI questionnaire (Note: only 75 subjects completed the trait-anger reaction subscale due to missing information in one questionnaire) and 86 subjects completed a post intervention PSS questionnaire. All subjects who had complete pre-post datasets were included in this study.

14.2 Recruitment

Age-eligible participants were recruited from May 2008 to December 2010. After the baseline screening visit, participants returned for study visits every 12 weeks for 6 – 10 months.

14.3 Antihypertensive Treatment

Nine subjects were advised to start antihypertensive therapy during the study due to the development of stage-2 hypertension. Subjects prescribed therapy continued in the study. Blood pressure data from subjects initiated on antihypertensive therapy was censored from the point of antihypertensive initiation onward.

15 Baseline Data

All subjects with available pre-post data were included in the analysis. All subjects were stage-1 hypertensive and untreated for at least 6 months prior to study entry. The mean 24-hour ambulatory blood pressure for all participants at baseline was 135/81 mmHg (SD 7.5/5.6). Mean daytime ambulatory blood pressure was 141/86 mmHg (SD 7.5/6.0) and mean nighttime ambulatory blood pressure was 122/71 mmHg (SD 10.31/7.0) at baseline. The sample was approximately two thirds female with 82% of subjects self-identifying as Caucasian. A summary of this information can be found in Table 9. Overall, the cohort was fairly healthy and highly educated. Baseline psychosocial data can be found in Table 10. Percentile ranking of STAXI-2 anger scores demonstrated that the cohort was average in terms of anger levels. Perceived stress levels were also comparable to PSS scores from the normative samples used to develop the PSS questionnaire.¹²⁹

Details regarding MBSR class attendance and homework completion were also collected. Out of 8 MBSR classes (excluding an introductory session and silent retreat) subjects attended an average of 6 classes (SD 1.5) and completed 32 minutes (SD 15.2) of additional MBSR homework per day (Table 11).

15.1 Baseline Correlations

There were no significant correlations between baseline primary psychosocial variables (trait anger, trait anger temperament, trait anger reaction, anger expressed inwards, anger expressed outwards or perceived stress) and baseline daytime or 24-hour or systolic or diastolic blood pressure (See columns labelled 'All Subjects' in Table 12 and Table 13). There were also no significant correlations between baseline primary psychosocial variables and baseline nighttime blood pressure (See column labelled 'All Subjects' in Table 14).

Among the baseline exploratory psychosocial measures (anger control inwards, anger control outwards and the anger expression index) only baseline anger control inwards was positively correlated with baseline daytime systolic blood pressure ($p=0.0429$). A negative correlation between baseline anger expression index and baseline daytime systolic blood pressure approached statistical significance ($p<0.10$) (see column labelled 'All Subjects' in Table 15, Table 16 and Table 17).

16 Anger & Stress

16.1 Pre-Post Changes

Trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress demonstrated statistically significant decreases from pre to post intervention (See rows labelled 'All Subjects' in Table 18). Change in the proportion of subjects scoring in the 75th percentile or greater for respective anger subscales was also evaluated. All anger measures, except for anger expression outwards, demonstrated statistically significant decreases from pre to post intervention. Anger expression outwards decreased by a non-significant amount ($p\text{-value}=0.0654$) (See rows labelled 'All Subjects' in Table 19). Overall, participants improved their psychosocial functioning for the primary psychosocial variables from pre to post intervention.

Among the exploratory psychosocial measures, anger control inwards and anger control outwards demonstrated statistically significant increases from pre to post intervention while the anger expression index demonstrated a statistically significant decrease (See rows labelled 'All Subjects' in Table 18). Changes in the proportion of subjects scoring in the 75th percentile or greater were also evaluated. There was a significant increase in the proportion of subjects scoring in the 75th percentile or greater for anger control inwards and a significant decrease for the anger expression index. The proportion of subjects scoring in the 75th percentile or greater for anger control outwards also increased, but by a non-significant amount ($p\text{-value}=0.1185$) (See rows labelled 'All Subjects' in Table 19). Overall, participants improved their psychosocial functioning for the exploratory psychosocial variables from pre to post intervention.

16.2 Psychometric Properties

Evaluations of baseline and post intervention PSS and STAXI-2 scores revealed high internal consistency for trait anger, trait anger temperament, trait anger reaction, anger expression inwards, perceived stress, anger control inwards and anger control outwards, except for anger expression outward which demonstrated slightly lower post intervention internal consistency ($\alpha = 0.62$) (Table 20). Sufficient internal consistency was determined using Nunnally & Bernstein's suggested value of 0.70.²⁰⁴

17 Blood Pressure

17.1 Pre-Post Changes

Twenty four hour and daytime systolic blood pressure demonstrated small but statistically significant decreases from pre to post intervention (-1.89 mmHg (SD 6.83), p -value=0.0124 and -2.12 mmHg (SD 7.09), p -value=0.0072, respectively). Twenty four hour diastolic, daytime diastolic and nighttime systolic and diastolic blood pressure did not demonstrate statistically significant changes from pre to post intervention (See rows labelled 'All Subjects' in Table 21).

17.2 Change Correlations with Psychosocial Measures

There were no significant correlations between change in primary psychosocial variables (trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards or perceived stress) and change in daytime or 24-hour blood pressure from pre to post intervention (See column labelled 'All Subjects' in Table 22 and Table 23). There were also no significant correlations with change in nighttime blood pressure (See column labelled 'All Subjects' in Table 24). Finally, there were no significant correlations between change in exploratory psychosocial variables (anger control inwards, anger control outwards and the anger expression index) and change in 24-hour, daytime and nighttime blood pressure from pre to post intervention (See column labelled 'All Subjects' in Table 25, Table 26 and Table 27).

17.3 Multivariate Linear Regressions

Results in this section have been organized in order of primary, secondary and exploratory hypotheses. The primary hypotheses testing the existence of an association between change in primary psychosocial variables (trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) and change in daytime and 24-hour blood pressure from pre to post intervention have been summarized in Tables 28 to Table 41. The secondary hypotheses testing the existence of an association between change in primary psychosocial variables and change in nighttime blood pressure from pre to post intervention have been summarized in Tables 42 to Table 48. The exploratory hypotheses testing the existence of an association between change in exploratory psychosocial variables (anger control inwards, anger control outwards and the anger expression index) and change in daytime,

24-hour and nighttime blood pressure from pre to post intervention have been summarized in Tables 49 to Table 57.

17.3.1 Primary Hypotheses: Primary Psychosocial Measures & 24-hour and Daytime Blood Pressure

After controlling for baseline blood pressure and baseline psychometric score the full main effects multivariate regression models revealed that post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inward, anger expression outward and perceived stress were not significantly associated with post intervention 24-hour or daytime systolic or diastolic blood pressure, respectively (For trait anger, see column labelled ‘All Subjects’ in Table 28 and Table 29. For trait anger temperament, see column labelled ‘All Subjects’ in Table 30, Table 31, Table 32 and Table 33. For trait anger reaction see column labelled ‘All Subjects’ in Table 34 and Table 35. For anger expression inward see column labelled ‘All Subjects’ in Table 36 and Table 37. For anger expression outward see column labelled ‘All Subjects’ in Table 38 and Table 39. For perceived stress see column labelled ‘All Subjects’ in Table 40 and Table 41).

17.3.2 Secondary Hypotheses: Primary Psychosocial Measures & Nighttime Blood Pressure

After controlling for baseline nighttime blood pressure and baseline psychometric score the full main effects multivariate regression models revealed that post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inward, anger expression outward and perceived stress were not significantly associated with post intervention nighttime systolic or diastolic blood pressure, respectively (For trait anger see column labelled ‘All Subjects’ in Table 42. For trait anger temperament see column labelled ‘All Subjects’ in Table 43 and Table 44. For trait anger reaction see column labelled ‘All Subjects’ in Table 45. For anger expression inward see column labelled ‘All Subjects’ in Table 46. For anger expression outward see column labelled ‘All Subjects’ in Table 47. For perceived stress see column labelled ‘All Subjects’ in Table 48).

17.3.3 Exploratory Hypotheses: Exploratory Psychosocial Measures & 24-hour, Daytime and Nighttime Blood Pressure

After controlling for baseline blood pressure and baseline psychometric score, the full main effects multivariate regression models revealed that post intervention anger expression inwards, anger expression outwards and the anger expression index were not significantly associated with post intervention 24-hour, daytime or nighttime systolic or diastolic blood pressure, respectively (For anger control inwards see column labelled ‘All Subjects’ in Table 49, Table 50 and Table 51. For anger control outwards see column labelled ‘All Subjects’ in Table 52, Table 53 and Table 54. For anger expression index see column labelled ‘All Subjects’ in Table 55, Table 56 and Table 57).

17.4 Effect Sizes

Beta coefficients of primary anger and stress predictor variables (trait anger, trait anger temperament, trait anger reaction, anger expressed in, anger expressed out and perceived stress) from the multivariate linear regression analyses were standardized to further examine their effect on 24-hour and daytime systolic and diastolic blood pressure. Cohen’s guidelines the interpretation of standardized effects are >0.50 = large, $0.50 - 0.30$ = moderate, $0.30 - 0.10$ = small and <0.10 = insignificant.²⁰⁵ Based on these guidelines, standardized effects from the primary psychosocial predictor variables ranged from insignificant to small (Table 58 and Table 59).

17.5 Post-Hoc Analyses

Post-hoc analyses included evaluating changes in anthropomorphic measurements, drinking habits and exercise levels from pre to post intervention. This included evaluating whether changes in drinking habits or exercise levels had taken place since baseline (yes or no) as well as changes in total number of drinks consumed per week and total number of times exercised per week.

Thirteen percent of respondents reported a change in drinking habits and 41% reported a change in exercise habits since baseline. Paired t-tests revealed that the total average number of drinks consumed per week decreased and almost reached statistical significance (p -value=0.0528).

Drinking habits did not demonstrate statistically significant changes when treated as a categorical variable (i.e., drink > 10 drinks per week, yes or no). Vigorous and moderate exercise levels (only available as categorical variables) did not demonstrate statistically significant changes from pre to post intervention. One anthropomorphic measurement, waist size measured at the iliac crest, demonstrated a statistically significant decrease from pre to post intervention (p -value = 0.0441). BMI also decreased but by a non-significant amount.

18 Exploratory Sex-Based Analyses

18.1 Baseline Data

Mean baseline 24-hour ambulatory blood pressure for males was 134/82 mmHg (SD 7.3/4.7) and for females was 135/82 (SD 8.3/6.3). Mean daytime ambulatory blood pressure for males was 140/87 mmHg (SD 7.6/5.4) and for females was 141/87 (SD 7.9/6.8). Mean baseline nighttime ambulatory blood pressure for males was 119/70 mmHg (SD 9.6/7.2) and for females was 123/72 (SD 11.6/7.6). A summary of this information can be found in Table 60. There were no significant differences for any baseline variables between males and females except for waist circumference ($p=0.0011$), nighttime systolic blood pressure ($p=0.0400$), proportion of males vs. females who drank >10 drinks per week ($p=0.0076$) and proportion of males vs. females who used relaxation techniques regularly ($p=0.0040$).

Percentile ranks for anger scores indicate that the cohort was average in terms of anger levels. Perceived stress scores were also comparable to those observed with normative samples used to generate the PSS questionnaire.¹²⁹ A summary of this information can be found in Table 61. Male and female study subjects demonstrated similar psychometric scores except for anger expression inwards (men = 18 (SD 4.5) and women = 16 (SD 4.3), p -value = 0.0231), anger control inwards (men = 20 (SD 5.4) and women = 23 (SD 5.6), p -value = 0.0186) and the anger expression index (men = 38 (SD 13.0) and women = 31 (SD 14.6), p -value = 0.0136).

18.2 Baseline Correlations

There were no significant correlations between baseline primary psychosocial variables (trait anger, trait anger temperament, trait anger reaction, anger expressed inwards, anger expressed outwards or perceived stress) and baseline 24-hour and daytime blood pressure among female subjects (See rows labelled 'Female' in Table 12 and Table 13). Among male subjects, one

significant negative correlation emerged between baseline daytime systolic blood pressure and baseline anger expression inwards (p -value = 0.0428) (See rows labelled ‘Male’ in Table 12 and Table 13). There were no significant correlations between baseline primary psychosocial variables and baseline nighttime blood pressure for either sex (See columns labelled ‘Female’ and ‘Male’ respectively in Table 14). Only baseline perceived stress approached statistical significance for a positive correlation with baseline nighttime systolic blood pressure among females ($p < 0.10$).

There were no significant correlations between baseline exploratory psychosocial variables (anger control inwards, anger control outwards and the anger expression index) and baseline daytime, 24-hour and nighttime blood pressure among female subjects (See columns labelled ‘Female’ in Table 15, Table 16 and Table 17). Among male subjects, one significant negative correlation emerged between baseline anger expression index and baseline daytime systolic blood pressure ($p = 0.0492$). One positive correlation approached statistical significance between baseline anger control inwards and baseline daytime systolic blood pressure ($p < 0.10$) (See columns labelled ‘Male’ in Table 15, Table 16 and Table 17).

19 Anger & Stress

19.1 Pre-Post Changes

All primary psychosocial variables (trait anger, trait anger temperament, anger expressed inwards, anger expressed outwards and perceived stress) except for trait anger reaction demonstrated statistically significant decreases from pre to post intervention among female subjects (See rows labelled ‘Female’ in Table 18). Among males, all primary psychosocial variables except for trait anger temperament and anger expression outwards demonstrated statistically significant decreases from pre to post intervention (See rows labelled ‘Male’ in Table 18). Changes in the proportion of subjects scoring in the 75th percentile or greater from pre to post intervention were also evaluated. While decreases were observed for all primary psychosocial variables for both sexes, only females demonstrated statistically significant decreases in the proportion of subjects scoring in the 75th percentile or greater for trait anger temperament and anger expression inwards (p -values = 0.0078 and 0.0013, respectively) (See rows labelled ‘Female’ and ‘Male’ Table 19). Overall, females appeared to improve on more primary psychosocial variables from pre to post intervention than males in this study.

Exploratory psychosocial measures (anger control inwards, anger control outwards and the anger expression index) demonstrated similar patterns of improvement in psychosocial functioning from pre to post intervention. For both males and females, anger control inwards and anger control outwards increased by a statistically significant amount from pre to post intervention and the anger expression index decreased by a statistically significant amount from pre to post intervention. The only exception was anger control outwards among male subjects which increased by a non-significant amount (p -value=0.1507) (See rows labelled 'Female' and 'Male' in Table 18). Again, changes in the proportion of subjects scoring in the 75th percentile or greater from pre to post intervention were also evaluated. Among female subjects, a statistically significant increase was observed for anger control inwards (p =0.0225) while a statistically significant decrease was observed for the anger expression index (p =0.0010). Anger control outwards also increased but by a non-significant amount. The same trends were observed for male subjects but the changes did not reach statistical significance (See rows labelled 'Female' and 'Male' in Table 19). Overall, females appeared to improve on more exploratory psychosocial variables than males.

20 Blood Pressure

20.1 Pre-Post Changes

Among females, 24-hour systolic and daytime systolic and diastolic blood pressure all decreased significantly from pre to post intervention (-3.09 mmHg (SD 6.26), p -value=0.0006, -3.47 mmHg (SD 6.16), p -value=0.0001, -1.31 mmHg (SD 4.70), p -value=0.0436, respectively). Twenty four hour diastolic and nighttime systolic and diastolic blood pressure demonstrated non-significant decreases (-1.11 mmHg (SD 4.52), p -value=0.0741, -2.49 (SD 9.81), p -value=0.0652 and -0.42 (SD 5.65), p -value=0.5851 respectively) (See rows labelled 'Female' in Table 21). No change in any ambulatory blood pressure parameter was observed for male subjects (See rows labelled 'Male' in Table 21).

20.2 Change Correlations with Psychosocial Measures

Despite improvements in psychosocial functioning and modest reductions in blood pressure from pre to post intervention, there were no significant correlations between change in primary psychosocial variables (trait anger, trait anger temperament, trait anger reaction, anger

expression inwards, anger expression outwards or perceived stress) and change in daytime or 24-hour blood pressure from pre to post intervention for male or female subjects (See columns labelled 'Female' and 'Male' in Table 22 and Table 23). Among females, change in anger expression inwards approached statistical significance for positive correlations with daytime and 24-hour systolic blood pressure ($p < 0.10$). Similar non-significance was reported for correlations between change in primary psychosocial variables and change in nighttime blood pressure (See columns labelled 'Female' and 'Male' in Table 24).

Statistically significant correlations emerged after stratifying by sex between changes in exploratory psychosocial measures (anger control inwards, anger control outwards and the anger expression index) and changes in 24-hour, daytime and nighttime blood pressure from pre to post intervention. Among female subjects, there was a significant positive correlation between change in the anger expression index and change in daytime systolic blood pressure ($p = 0.0319$) (See column labelled 'Female' in Table 26). Three additional correlations approached statistical significance ($p < 0.10$): a positive correlation between change in anger expression index and change in 24-hour systolic blood pressure (See column labelled 'Female' in Table 25), a negative correlation between change in anger control inwards and change in daytime systolic blood pressure (See column labelled 'Female' in Table 26) and a negative correlation between change in anger control outwards and change in daytime systolic blood pressure (See column labelled 'Female' in Table 26). Among male subjects, a significant positive correlation was found between change in anger control inwards and change in daytime systolic and diastolic blood pressure ($p = 0.0351$ and 0.0330 , respectively) (See column labelled 'Male' in Table 26).

20.3 Multivariate Linear Regressions

Results from this sex-based analysis are entirely exploratory. However, for ease of reporting they have been organized in the same order as the main multivariate linear regression results. More specifically they have been organized in the same order as the main results (primary, secondary and exploratory hypotheses). To review, the primary hypotheses testing the existence of an association between change in primary psychosocial variables (trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) and change in daytime and 24-hour blood pressure from pre to post intervention have been summarized in Tables 28 to Table 41. The secondary hypotheses testing the existence

of an association between change in primary psychosocial variables and change in nighttime blood pressure from pre to post intervention have been summarized in Tables 42 to Table 48. The exploratory hypotheses testing the existence of an association between change in exploratory psychosocial variables (anger control inwards, anger control outwards and the anger expression index) and change in daytime, 24-hour and nighttime blood pressure from pre to post intervention have been summarized in Tables 49 to Table 57.

20.3.1 Primary Hypotheses: Primary Psychosocial Measures & 24-hour and Daytime Blood Pressure

After controlling for baseline blood pressure and baseline psychometric score, the full main effects multivariate regression models revealed that post intervention trait anger, trait anger reaction, anger expression inward and anger expression outward were not significantly associated with post intervention 24-hour or daytime systolic or diastolic blood pressure, respectively for either male and female subjects (For trait anger see columns labelled 'Female' and 'Male' respectively in Table 28 and Table 29. For trait anger reaction see columns labelled 'Female' and 'Male' respectively in Table 34 and Table 35. For anger expression inward see columns labelled 'Female' and 'Male' respectively in Table 36 and Table 37. For anger expression outward see columns labelled 'Female' and 'Male' respectively in Table 38 and Table 39)

However, post intervention trait anger temperament and perceived stress were found to be significantly associated with blood pressure after stratification by sex.

Trait Anger Temperament

Post intervention trait anger temperament was significantly positively associated with post intervention 24-hour systolic blood pressure for female subjects ($p=0.01$) (See column labelled 'Female' Table 30). More specifically, every one point increase in trait anger temperament scale was associated with a 2.71 mmHg increase in 24-hour systolic blood pressure. A positive association with daytime systolic blood pressure approached statistical significance ($p=0.07$, see column labelled 'Female' in Table 31). When trait anger temperament was reevaluated for exploratory purposes as a multilevel categorical predictor variable, after adjustment for baseline blood pressure and baseline trait anger temperament, post intervention trait anger temperament

was still significantly associated with 24-hour systolic blood pressure (See column labelled 'Female' in Table 32). More specifically, being in the lowest quartile compared to the upper quartile of trait anger temperament scores after the MBSR program was associated with lower blood pressure ($p=0.02$), as was being in the second quartile compared to the upper quartile ($p=0.03$). Scoring in the third quartile compared to the upper quartile for post intervention trait anger temperament scores almost reached statistical significance ($p=0.07$). The same positive association approached statistical significance for trait anger temperament and daytime blood pressure (See column labelled 'Female' in Table 33). No association between trait anger temperament and 24-hour or daytime blood pressure was reported for male subjects (See column labelled 'Male' in Table 30, Table 31, Table 32 and Table 33).

Perceived Stress

After controlling for baseline blood pressure and baseline perceived stress score, the full main effects model revealed that post intervention perceived stress was significantly negatively associated with post intervention 24-hour systolic blood pressure among females ($p=0.03$) (See column labelled 'Female' in Table 40). More specifically, every one point increase in perceived stress was associated with a 0.35 mmHg decrease in 24-hour blood pressure. However, among male subjects, post intervention perceived stress score was significantly positively associated with post intervention 24-hour and daytime systolic blood pressure ($p=0.02$ and $p=0.02$, respectively) (See columns labelled 'Male' in Table 40 and Table 41). Every one point increase in perceived stress was associated with a 0.83 mmHg or 0.91 mmHg increase in 24-hour or daytime systolic blood pressure, respectively.

20.3.2 Secondary Hypotheses: Primary Psychosocial Measures & Nighttime Blood Pressure

After controlling for baseline nighttime blood pressure and baseline psychometric score the full main effects multivariate regression models revealed that post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inward and anger expression outward were not significantly associated with post intervention nighttime blood pressure in either sex (For trait anger see column labelled 'Female' and 'Male' in Table 42. For trait anger temperament see column labelled 'Female' and 'Male' in Table 43 and Table 44. For trait anger reaction see column labelled 'Female' and 'Male' in Table 45. For anger expression inward see column

labelled 'Female' and 'Male' in Table 46. For anger expression outward see column labelled 'Female' and 'Male' in Table 47).

However, post intervention perceived stress was found to be significantly associated with post intervention nighttime systolic blood pressure among both males and females.

Perceived Stress

After controlling for baseline blood pressure and baseline perceived stress score, the full main effects model containing female subjects revealed that post intervention perceived stress was significantly negatively associated with post intervention nighttime systolic blood pressure ($p=0.02$) (See column labelled 'Female' in Table 48). More specifically, every one point increase in perceived stress was associated with a 0.52 mmHg decrease in nighttime systolic blood pressure. Among male subjects, post intervention perceived stress was significantly positively associated with post intervention nighttime systolic blood pressure ($p=0.04$) (See column labelled 'Male' in Table 48). Every one point increase in perceived stress was associated with a 0.61 mmHg increase in nighttime systolic blood pressure.

20.3.3 Exploratory Hypotheses: Exploratory Psychosocial Measures & 24-hour, Daytime and Nighttime Blood Pressure

After controlling for baseline blood pressure and baseline psychometric score, the full main effects multivariate regression models revealed that post intervention exploratory variables (anger expression inwards, anger expression outwards and the anger expression index) were not significantly associated with post intervention 24-hour, daytime or nighttime blood pressure among males or females (For anger control inwards see column labelled 'Female' and 'Male' in Table 49, Table 50 and Table 51. For anger control outwards see column labelled 'Female' and 'Male' in Table 52, Table 53 and Table 54. For the anger expression index see column labelled 'Female' and 'Male' in Table 55, Table 56 and Table 57).

20.4 Effect Sizes

Beta coefficients for anger or stress predictor variables that emerged as statistically significant in the exploratory sex-based multivariate linear regression analyses were standardized to further

examine effects on blood pressure. To review, Cohen's guidelines the interpretation of standardized effects are >0.50 = large, $0.50 - 0.30$ = moderate, $0.30 - 0.10$ = small and <0.10 = insignificant.²⁰⁵ Based on these guidelines, trait anger temperament was observed to have a large effect on 24-hour systolic blood pressure among females (standardized $\beta = 0.62$ (SE 0.22), Table 68). Perceived stress had moderate effects on 24-hour and nighttime systolic blood pressure among females (standardized $\beta = -0.30$ (SE 0.13) and -0.36 (SE 0.14), respectively, Table 69). Among males, perceived stress had moderate effects on 24-hour and nighttime systolic blood pressure (standardized $\beta = 0.49$ (SE 0.19) and 0.33 (SE 0.15), respectively, Table 70) and a large effect on daytime systolic blood pressure (standardized $\beta = 0.52$ (SE 0.21), Table 70).

21 All Results Summarized

All measures of anger and perceived stress demonstrated statistically significant improvements from pre to post intervention when all subjects were examined together (Table 18 and Table 19). Blood pressure also demonstrated modest but statistically significant decreases from pre to post intervention for 24-hour and daytime systolic blood pressure when subjects were examined together (See column labelled 'All Subjects' in Table 21).

Despite the significant above-mentioned changes from pre to post intervention, baseline correlations and change correlations revealed no significant associations between both primary and exploratory psychometric measures and any ambulatory blood pressure parameter. The only exception to this was an exploratory anger measure (baseline anger control inwards) which demonstrated a positive correlation with baseline daytime systolic blood pressure (See column labelled 'All Subjects' in Table 16). After adjusting for baseline blood pressures, baseline psychometric values and other covariates, the multivariate linear regression analyses revealed that no primary psychosocial measure (post intervention trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards and perceived stress) nor exploratory psychosocial measures (post intervention anger control inwards, anger control outwards and the anger expression index) were associated with post intervention 24-hour, daytime or nighttime systolic or diastolic blood pressure (See column labelled 'All Subjects' in Tables 62 - 67). Effect sizes as measured by standardized regression coefficients ranged from insignificant to small.

The exploratory sex-based analysis revealed interesting points of significance. First, female participants improved on more psychometric measures than males in the study. Second, only female participants decreased blood pressure from pre to post intervention in the study, reducing their 24-hour and daytime systolic blood pressure by -3.09 mmHg and -3.47 mmHg, respectively. Male participants either did not change or marginally raised their blood pressure from pre to post intervention in the study.

Exploratory baseline and change correlation analyses revealed some correlations of significance; however, most significant were findings from the follow-up multivariate linear regression analyses. After adjusting for baseline blood pressures and baseline psychometric values, among female subjects post intervention trait anger temperament was significantly positively associated with post intervention 24-hour systolic blood pressure among females while post intervention perceived stress was significantly negatively associated with post intervention 24-hour and nighttime systolic blood pressure. Among males, perceived stress was significantly positively associated with 24-hour, daytime and nighttime systolic blood pressure (See column labelled 'Female' and 'Male' in Table 62, Table 63, Table 64). Effect sizes as measured by standardized regression coefficients ranged from moderate to large.

Chapter 4 Discussion

22 General Overview

Previous research has suggested an association between anger, perceived stress and blood pressure. These assertions have largely been based on cross-sectional studies that collected data at one time point that did not manipulate independent psychosocial variables of interest. More rigorous prospective longitudinal cohort research was proposed to test these relationships more directly thus add more robust evidence to the literature.

This thesis study was interested in using a prospective longitudinal cohort design to test the relationship between anger, perceived stress and blood pressure. This design was implemented by using a psychological therapeutic intervention to manipulate anger and perceived stress. Changes in anger and perceived stress were in turn evaluated for association with changes in ambulatory blood pressure. In the present study, mindfulness-based stress reduction (MBSR) was used as the tool to manipulate anger and perceived stress.

Findings from this study failed to demonstrate an association between change in anger or perceived stress with change in daytime, 24-hour or nighttime blood pressure after participants completed a standard 8-week MBSR program. Corresponding standardized regression coefficients indicated small to insignificant effects on blood pressure. Thus, these results failed to support the hypothesis of an association between change in anger and perceived stress with change in blood pressure after participants completed a standard 8-week MBSR program. Exploratory analyses revealed significant associations after stratification by sex.

This study has helped to clarify the anger, perceived stress and blood pressure literature by producing results that have been based on a stronger study design. More specifically, this study has contributed results that have been based on a prospective longitudinal cohort design where an intervention was used to manipulate the independent psychosocial variables of interest. By comparison, the majority of anger, perceived stress and blood pressure literature has been based on single time point cross-sectional observational studies that did not manipulate independent psychosocial predictor variables of interest. Based on a stronger study design, the results of this

thesis have helped to clarify the literature and our understanding of anger and perceived stress as suspected risk factors for hypertension development.

23 Findings, Explanations and Implications

23.1 Primary Findings

To review, the primary objective of this study was to establish the existence of an association between change in trait anger, trait anger temperament, trait anger reaction, anger expression inward, anger expression outward, perceived stress and change in 24-hour and daytime blood pressure among unmedicated stage-1 hypertensive subjects who completed a standard 8-week MBSR protocol. Secondary objectives included establishing associations between changes in these primary psychosocial variables and changes in nighttime blood pressure. Exploratory analyses included evaluating the existence of an association between change in anger control inwards, anger control outwards and the anger expression index with change in 24-hour, daytime and nighttime blood pressure. Additional exploratory analyses included evaluating the existence of any sex differences.

Participants enrolled in the study were physically healthy with the exception of elevated blood pressure. Baseline psychosocial data indicated that the group was average in terms of their anger profiles based on percentile rank information from the STAXI-2 professional manual.

Comparing mean baseline PSS scores from this study to the normative samples used during PSS validation in 1983, the mean score in this study was almost identical to scores obtained for the normative samples (normative sample 1 = 23 (SD 7.3), normative sample 2 = 24 (7.8), PSS average in this study = 24 (8.2)).¹²⁹ Compared to a separate probability sample collected in 1983 in the United States, the mean PSS score from this study was only slightly above average (probability sample mean = 20 (SD 7.5)).¹⁸⁷ Based on this information it can be concluded that participants in this study were overall physically and mentally healthy.

Taking a collective and comprehensive approach to reviewing the results reveals that subjects benefitted psychologically (improved anger and stress) and physiologically (reduced blood pressure) from enrolling in MBSR. All measures of anger and perceived stress demonstrated statistically significant improvements from pre to post intervention. For anger, this was maintained when anger constructs were evaluated both as raw scores and clinically relevant

binary variables dividing scores between the top 25% percentile and bottom 75% percentile (Table 18 and Table 19). The only exceptions were anger expression outwards and anger control outwards when evaluated as binary variables. However, both variables still demonstrated changes in the direction of therapeutic benefit that approached statistical significance ($p=0.0654$ and $p=0.1185$, respectively). In addition to psychosocial functioning, 24-hour and daytime systolic blood pressure demonstrated statistically significant decreases from pre to post intervention. Taken together, this information outlines improved psychosocial and physiological profiles from pre to post intervention for the subjects enrolled in this study.

But despite these significant pre-post changes, baseline correlation analyses and pre-post change correlation analyses revealed no significant correlations between anger, perceived stress and any ambulatory blood pressure parameter. The only exception was a significant positive baseline correlation between an exploratory variable (anger control inwards) and daytime systolic blood pressure. However, after adjusting for baseline blood pressures, baseline psychometric values as well as other covariates, the multivariate linear regression analyses revealed that post intervention anger and perceived stress were not associated with post intervention 24-hour, daytime or nighttime blood pressure. In summary, pre-post data illustrated statistically significant improvements in psychosocial functioning and blood pressure. However, these variables failed to demonstrate association with one another in follow-up correlation and multivariate linear regression analyses.

Together these results highlight a significant feature: subjects improved anger and stress, as well as modestly improved their blood pressure. However, these changes were unassociated with one another in this study.

Excluded Explanations

A number of possible explanations for these findings can be considered unlikely and thus excluded. First and foremost is the possibility that predictor variables did not change sufficiently from pre to post intervention (i.e., that MBSR did not manipulate anger or stress by a sufficient amount to detect an association with blood pressure). Raw pre-post data suggests this is not the case. Statistically significant reductions for trait anger, trait anger temperament, anger expression inward, anger expression outward, perceived stress and the anger expression index were observed from pre to post intervention for all subjects. Correspondingly, there were statistically

significant increases in anger control inward and anger control outward. According to Spielberger “the development of internal controls over the experience and expression of anger is generally seen in a positive light” particularly if they are accompanied by an improvement in anger expression. Thus, these reciprocal changes in anger expression (which was reduced) and anger control (which was improved) align with a therapeutic improvement in subjects’ anger profile.

Further evidence of improved psychosocial functioning included statistically significant improvements in the proportion of subjects scoring in the 75th percentile or greater on all of the anger subscales except for anger control outwards and anger expression outwards. Although anger control outwards and anger expression outwards did not demonstrate statistically significant changes, these two variables still demonstrated improvement trending in the direction of therapeutic benefit. According to Spielberger, anger scores in the 75th percentile greater “have been shown to be associated with elevated blood pressure and hypertension”^{2, 57, 66, 198}. Considering this information, the improvement observed in the proportion of subjects scoring in the 75th percentile or greater is clinically significant and reinforces raw score data outlining that participants became less angry from pre to post intervention in this study. Taken together this information excludes the possible explanation that a lack of association in this study was due to lack of psychosocial improvement.

Measurement error and methodological inconsistencies are also unlikely explanations for this study’s findings. This study was prospective and was rigorously executed, using only validated psychometric questionnaires and the gold standard in blood pressure measurement. While measurement error can never be entirely eliminated, poor methodology is an unlikely explanation for the non-significance reported in this study.

Lack of participant engagement is also an unlikely explanation for this study’s findings. The HARMONY trial was advertised as a non-pharmacological treatment intervention for blood pressure lowering for individuals with untreated stage-1 hypertension. Anecdotally, many participants in the study had been informed of their elevated blood pressure by a health care provider and that pharmacological treatment may be required in the future. Many subjects enrolled in the HARMONY study were thus highly motivated to find non-drug alternatives for blood pressure lowering.

Evidence of this motivation can be demonstrated by adherence to the MBSR program. This was quantified by the number of minutes spent per day on homework meditation practice. The average amount of homework completed was 32 minutes (± 15 minutes) out of the required 45 minutes per day; approximately 71%. Adherence or ‘acceptable compliance’ has been defined by pharmacological anti-hypertensive literature as taking $\geq 80\%$ of prescribed antihypertensive medications.^{206, 207} Using this guideline, acceptable compliance to the MBSR program would be 45 minutes $\times 80\% = 36$ minutes. Considering that average adherence to pharmacological therapy is estimated to be 50% - 70%²⁰⁸ adherence in the MBSR program was on par, if not slightly higher than the average adherence to pharmacological therapy. Moreover, some items on the homework logs did not measure number of minutes of homework practiced but still represented some form of mindfulness meditation. Therefore, this MBSR adherence estimate is likely a conservative estimate, with more participants engaging in the daily homework meditation practices than captured by homework logs. Taken together, it can be concluded that lack of participant engagement in the MBSR program is an unlikely explanation for the non-significant findings reported between stress, anger and blood pressure in this study.

To summarize, there are a number of explanations for these findings that are unlikely and can therefore be excluded: lack statistically significant improvement in the predictor variables (psychosocial functioning), poor measurement methodology and lack of participant engagement/low treatment adherence. The exclusion of these variables has been supported by evidence from the study.

Potential Explanations

In this study perceived stress, anger and blood pressure decreased by statistically significant amounts from pre to post intervention. However, these decreases failed to demonstrate an association with one another. One hypothesis to explain these findings may be that the relationship between anger, perceived stress and blood pressure is mediated by a third variable.

The existence of a third variable mediating the relationship between psychosocial variables and blood pressure could exist. Evidence from one study provides strong support for this hypothesis.²⁰⁹ In this study, information was collected on job stress, blood pressure, as well as adaptive and maladaptive stress coping behaviours. The authors found no association between work stress and blood pressure. However, the authors did find that “the ways [in which]

individuals reported coping with stress were significantly related to blood pressure, with blood pressure elevation effects appearing to be mediated largely by dietary and drinking habits and physical inactivity”.²⁰⁹ In another separate study, improved stress management was individually associated with improved hypertensive risk factors, such as weight.²¹⁰ Taken together, this evidence outlines a possible model by which improved psychosocial functioning and blood pressure could be indirectly associated via health behaviours or stress coping mechanisms.

Post-hoc analyses of changes in anthropomorphic measurements, drinking habits and exercise levels from pre to post intervention revealed some support for this model. Indications of lifestyle and health behaviours known to be linked to blood pressure, specifically waist size²¹¹ and alcohol consumption,²¹²⁻²¹⁴ improved from pre to post intervention in this study. Based on the evidence that individuals respond to stress with different stress coping mechanisms that are linked to blood pressure (such as drinking habits and exercise²⁰⁹) and that stress management has been individually associated with improved hypertensive risk factors²¹⁰ suggests that improved health behaviours/stress coping mechanisms may be the mechanism linking the association between psychosocial variables and blood pressure in this study. It should be mentioned that the post-hoc analysis does not claim anything other than indications of improved lifestyle. However, results from this analysis do align with a possible model whereby improved psychosocial functioning and hypertension are indirectly associated with one another via health behaviours or stress coping mechanisms. This model offers one possible explanation for the overall findings of this study.

Given that this was a neutral study other possible explanations for these findings exist. First is the possibility that baseline anger and perceived stress were too low to demonstrate an association with blood pressure. As stated earlier, high anger scores particularly those $\geq 75^{\text{th}}$ percentile “have been shown to be associated with elevated blood pressure and hypertension”.^{2, 57, 66, 198} This raises the possibility that associations between psychosocial variables and blood pressure may exist among those who suffer from severe anger or stress issues. If true, this could explain the lack of association observed in this thesis study. Future directions should include evaluating associations between stress, anger and blood pressure among those who suffer from severe stress or anger issues.

Another possible explanation for these results includes the narrow racial/ethnic profile of participants in this study. Eighty two percent of participants in this study self-identified as Caucasian. As reviewed in the introduction, the existence of underlying racial/ethnic effects is still unclear; thus it is possible that different findings could emerge when studying participants from different racial/ethnic backgrounds.

Thus to summarize, a number of explanations exist that could explain the study's findings. First is the existence of a mediating variable such as a health related behaviour or stress coping mechanism that indirectly links psychosocial variables and blood pressure. Post-hoc analyses support this possible hypothesis. Other explanations include that associations between anger, perceived stress and blood pressure may only exist among those suffering from severe anger or stress issues. A final possible explanation includes that the relationship between psychosocial variables and blood pressure among participants of different racial/ethnic backgrounds. This list of possible explanations is by no means exhaustive. If contemplated, any future research should focus on these unexplored areas to clarify these possible explanations.

Implications

To the best of our knowledge, this is the first study to use a prospective longitudinal cohort design to test the relationships between perceived stress, anger and blood pressure by using validated questionnaires, a gold standard blood pressure measurement technique and a tool to manipulate the psychosocial predictor variables of interest. The results from this study have added clarity to the field by contributing findings that are rooted in a stronger study design than has been previously reported by the majority of the literature.

As covered in the first chapter, the literature was equivocal (but not entirely) in terms of an association between anger and blood pressure. The literature was more positive with respect to an association between perceived stress and blood pressure. Reasons for a divide in the anger literature are unclear, but proposed explanations include differences in race/ethnicity, sex/gender and varying blood pressure measurement techniques. Differing selection of covariates between studies has also been proposed as an explanation for varying findings.²⁴ With respect to perceived stress, the majority of the literature reporting a positive association was based on non-validated measures of perceived stress. These divides in the literature, poor study designs and lack of robust measurement tools was the impetus behind this study.

Results from this study failed to demonstrate an association between change in anger and perceived stress and change in ambulatory blood pressure among stage-1 untreated hypertensive subjects who underwent an 8-week mindfulness based stress reduction program. The results from this study conform to approximately half of the anger and blood pressure literature and less than half of the perceived stress literature suggesting no association between these variables and blood pressure.

The conclusions of this thesis have a number of implications on the literature. Most important is the failure to support the hypothesis of an association between anger, perceived stress and blood pressure in a field of research that is highly divided on the issue. The implications of this rejection are particularly significant due to the robustness of the evidence provided by this study, which was achieved through the use of a stronger study design than has been previously reported by the majority of the literature

However, what has also emerged from this work is the possibility that a different model may link these variables. Psychosocial functioning and blood pressure improved from pre to post intervention but failed to demonstrate association with one another in the correlation and multivariate regression analyses. Based on this trend, it is possible that these variables are linked indirectly by another variable such as stress coping mechanisms or health behaviours. Evidence from the literature combined with results from the post-hoc analyses preformed in this study support this proposed model. This potential explanation has its own implications on the literature, particularly with regards to changing our current understanding of the relationship between psychosocial variables and blood pressure.

In summary, the implications of this study on the literature include clarifying our understanding of anger and perceived stress as suspected risk factors for hypertension, as well as a call for new research that focus on evaluating indirect associations between anger, perceived stress and blood pressure. More specifically, research should focus on the effects of stress/psychosocially driven coping mechanisms/health behaviours and their relationship to blood pressure and hypertension.

23.2 Exploratory Sex-Based Findings

The exploratory sex-based analysis revealed several interesting points of significance. First, female participants improved on more psychometric measures compared to male counterparts in

the study. Second, only female participants had a fall in blood pressure from pre to post intervention in the study. Female participant's 24-hour and daytime systolic blood pressure fell by -3.09 mmHg and -3.47 mmHg, respectively, while male participants increased their 24-hour and daytime systolic blood pressure by 0.30 mmHg and 0.37 mmHg, respectively.

Baseline and change correlation analyses revealed some correlations of significance. However, follow-up multivariate linear regression analyses revealed specific associations of significance. After adjusting for baseline blood pressures and baseline psychometric values, post intervention trait anger temperament was found to be significantly positively associated with post intervention 24-hour systolic blood pressure among females (See column labelled 'Female' in Table 30 and Table 32), and post intervention perceived stress was negatively associated with post intervention 24-hour and nighttime systolic blood pressure among females and positively associated with 24-hour, daytime and nighttime systolic blood pressure among males (See column labelled 'Female' and 'Male' in Table 40, Table 41, Table 48).

These findings outline an interesting interaction between sex/gender, anger, perceived stress and blood pressure. It should be noted that this finding is exploratory and thus only hypothesis generating. Therefore, the following discussion regarding these findings is entirely speculative.

Trait Anger Temperament

Trait anger temperament measures the disposition to experience anger without specific provocation. In this study, higher trait anger temperament scores after the intervention were associated with higher 24-hour systolic blood pressure for female subjects. Every one point increase in trait anger temperament after the MBSR program (from total score range of 4 to 16 points) corresponded to an increase of 2.71 mmHg for 24-hour systolic post-intervention blood pressure.

This finding aligns with the study's raw psychometric data where only females demonstrated statistically significant reductions in trait anger temperament from pre to post intervention. The literature review on trait anger did outline possible sex differences suggesting a stronger effect among females.^{67, 69, 73-75} Additional evidence also suggests that males and females respond differently to stress reduction interventions.²¹⁵ Females have been cited to derive greater benefit from stress reduction,²¹⁶ more often reporting cognitive benefits over physical/athletic benefits

reported by males.^{217, 218} Studies using MBSR to treat hostility among inmates have reported greater pre-post improvements on self-report measures of hostility among female subjects.²¹⁹ Thus, this improved trait anger temperament finding among female subjects aligns with what is reported in the literature.

Only a handful of studies have examined trait anger temperament and blood pressure.^{55, 56, 66} These have been limited to studies on male adolescents or male war veterans and thus are limited in generalizability. Therefore this analysis of trait anger temperament is a novel contribution to the field.

Perceived Stress

The results for perceived stress reveal conflicting findings. Perceived stress was negatively associated with 24-hour and nighttime systolic blood pressure among females and positively associated with 24-hour, daytime and nighttime systolic blood pressure among males in the multivariate linear regression analyses.

The findings for the female participants are in opposition to all of the perceived stress literature which has only reported either no association or a positive association between perceived stress and blood pressure. Several steps were taken to evaluate the validity of these findings. A literature review was performed to determine whether stress reduction interventions had previously increased stress among women. No support could be found for this hypothesis. A review of the original data revealed no errors or outliers and reexamining the correlation matrices between post intervention perceived stress and post intervention blood pressure revealed no discernible pattern in the data (Figure 1 and Figure 2). Based on the contrast of this finding to the otherwise psychological and physiological beneficial outcomes observed for females in this study, the results of the literature review, the re-examination of the raw data and the high number of comparison procedures undertaken in these analyses it was determined that this result was most likely a chance finding.

Among males, results from the exploratory sex-based analysis revealed a positive association between perceived stress and blood pressure. This finding is consistent with approximately half of the literature that has studied perceived stress as measured by a validated questionnaire and blood pressure (Table 6). These results conform to an even greater number of studies from the

non-validated perceived stress literature (Table 6). However, the exploratory sex-based analysis also revealed that male participants did not change their blood pressure in this study. Thus, the validity of these results from the multivariate linear regression analyses for male subjects is questionable. Due to the lack of blood pressure lowering and high number of comparison procedures undertaken in these analyses it was determined that the perceived stress results for male subjects were also likely chance findings.

Exploratory Sex-Based Analyses: Summary

Findings from this exploratory sex-based analysis revealed a positive association between trait anger temperament and 24-hour systolic blood pressure among female subjects. For reasons discussed above, results from the perceived stress analyses were determined to likely be chance findings.

In terms of reconciling these findings with the proposed model of an indirect association between psychosocial variables and blood pressure linked via stress coping mechanisms/health behaviours, the exploratory nature of the sex-based analysis is limited in its scope of impact. More research on an adequately sized female sample must be performed in order to determine both the existence and validity of the association between trait anger temperament and blood pressure. It should be noted that no other anger construct was linked to blood pressure for males or females. Thus, this single finding does not significantly challenge the earlier proposed model of an indirect association between anger, perceived stress and blood pressure.

In fact, the lack of effect observed among the remaining anger constructs, despite the greater psychosocial improvement and blood pressure lowering observed among females, reinforces the proposed hypothesis of indirect association between psychosocial functioning and blood pressure. Interestingly, data from the Canadian National Population Health Survey which studied gender differences in health found that “social structural and psychosocial determinants of health were more important for females”²²⁰ over behavioural determinants. Sociological models such as the ‘tend and befriend’ stress response demonstrated by females and the ‘fight or flight’ stress response demonstrated by males²²¹ further underscores the possibility MBSR provided female subjects with stress coping mechanisms that better matched their stress behaviours compared to male subjects. Taken together, this information aligns directly with a hypothesis that an

intervention like MBSR could have a greater effect among females by possibly improving stress coping mechanisms and health behaviours linked to blood pressure.

In summary, findings from this exploratory sex-based analysis revealed a positive association between trait anger temperament and 24-hour systolic blood pressure among females. However, the exploratory nature of this analysis renders these findings hypothesis generating and explanations entirely speculative. The increased risk of type 1 error due to many comparison procedures challenges the strength of this finding. Therefore, while exciting, these results must be interpreted appropriately and confirmed with follow up research.

24 Limitations and Considerations

Study Design

The prospective longitudinal cohort within-group design is a limitation to this study. While this study was entirely neutral reporting no association between change in anger, perceived stress and blood pressure, only a randomized controlled trial design can best account for confounding and provide the most robust explanations for the study's findings. Moreover, despite the inclusion of baseline blood pressures and psychometric values in all regression analyses, without a randomly allocated placebo control group statistical measurement phenomena such as regression to the mean may also be exacerbated.²²² Thus, the prospective longitudinal cohort within-group design used in this thesis carries some inherent limitations that must be considered when evaluating the results of this study.

Participant Homogeneity

Subjects were recruited from the Toronto and greater Toronto area using advertising in the Toronto Star (online but majority in print), posters in Toronto General Hospital and Sunnybrook Health Sciences Centre and on a referral basis from physicians. A significant proportion of the participants learned about study through printed advertisements in the Toronto Star. Due to funding restraints and participant travel considerations, only select areas of Toronto received the printed advertisements. This recruitment strategy invariably selected individuals with a certain level of reading ability and income, thus potentially influencing the demographic of individuals

enrolled in study. It may also have impacted the racial/ethnic profile of the participants enrolled. Together this limits the generalizability of this study's findings.

Participant Motivation

Participants in this study were originally enrolled into the HARMONY trial, a study interested in evaluating a non-pharmacological treatment option for blood pressure lowering among untreated stage-1 hypertensives. Many participants in the trial had been informed by a health care provider that they may need pharmacotherapy in the future. Participants enrolling in the study were highly motivated to find non-drug alternatives for blood pressure lowering, as evidenced by their adherence to the MBSR program discussed earlier. These participants may not be an accurate reflection of the general population therefore also limiting the generalizability of these findings.

Biased Analyses

The analysis implemented in this study used complete pre-post datasets. Those with missing data were excluded. This approach inherently created a biased sample as only those participants who completed all required study visits were included in the analyses. Multiple imputation is one option to manage missing data; however, it is complex and computationally intensive.²²³ The decision to use multiple imputation is based in part on why data are missing. In this study, the missing data was not missing at random. More specifically, differences between subjects who completed all visits and subjects who did not complete all visits could not be explained by the same reason (i.e., a broken blood pressure monitor). Reasons for missing data are unclear, but may include participant responses to the intervention (i.e., some participants may not have liked the program) as well as changes in personal commitments that restricted their participation in the study. When data is not missing at random, multiple imputation carries a risk of introducing its own bias into a sample.²²³ Thus, based on both complexity of imputation and type of missing data in this study, multiple imputation was not used.

25 Future Directions

Evaluation of Indirect Mechanisms

Future directions should include evaluating the proposed model linking psychosocial health with blood pressure via stress coping mechanisms and health behaviours. More specifically, future

directions should involve evaluating the effectiveness of programs that target improved psychosocial health in addition to heart healthy behavioural changes with the goal of evaluating blood pressure change or control. Using ambulatory blood pressure as an outcome, one type of future research study could use a randomized controlled trial format where subjects could be randomized to lifestyle recommendations (usual care) or lifestyle recommendations plus MBSR. This type of study would enable a better understanding of the existence and extent of association between psychosocial health, heart healthy behaviours and blood pressure. If a linkage was confirmed, and specific psychosocial features could be identified that would make some patients more or less adherent to heart healthy behaviours linked to blood pressure, then it would be possible to refer (or even develop) psychosocial programs that may assist patients with adhering to lifestyle changes.

Preliminary research in this area has already begun among coronary heart disease patients. Using a randomized controlled trial format, cognitive behavioural therapy was demonstrated to decrease the rate of fatal and non-fatal first recurrent cardiovascular events as well as the number of acute myocardial infarctions.²²⁴ Although not examining blood pressure directly, this study underscores the strong potential existence of an indirect linkage between psychosocial health and blood pressure, and the need for future research to better understand the interplay of this relationship as it is mediated by stress coping mechanisms and health behaviours.

Angry/Stressed Populations

The participants examined in this study were healthy middle-aged adults with the exception of mildly elevated blood pressure. Based on anger literature suggesting that those scoring in the 75th percentile or greater have elevated blood pressure^{57, 66, 198} and that those scoring in the 90th percentile or greater have an increased risk for coronary artery disease and heart attacks,¹⁹⁷ it would be of interest to examine associations between perceived stress, anger and blood pressure among those suffering from more severe stress or anger issues. Thus, future directions should also include evaluating the linkages between psychosocial health, stress coping mechanisms/health behaviours and hypertension among those with more severe anger or stress issues.

Different Racial/Ethnic Populations

Majority of participants in this study self-identified as Caucasian (82%). As explained in the introduction, racial/ethnic contributions are still unclear; thus, different results may have been obtained had this study been completed on subjects from different racial/ethnic backgrounds. Therefore, future research should evaluate direct associations between anger, perceived stress and blood pressure, as well as indirect associations via stress coping mechanisms and health behaviours among subjects from different racial/ethnic backgrounds.

Physiological Measures of Stress

Psychosocial testing provides valuable subjective accounts of psychosocial functioning. However, objective physiological measures of stress (such as cortisol or inflammatory markers) could enable a better understanding the relationship between psychosocial variables and blood pressure. Although no direct associations between anger, perceived stress and blood pressure were identified in this study, any future research studying indirect linkages, more stressed/angry populations and other racial/ethnic groups would be bolstered by the addition of objective physiological stress measures. Thus, future research in this area should consider the inclusion of external physiological measures of stress.

Sex/Gender Based Analyses

The results of this study have underscored the critical need for more sex-based analysis in the area of psychosocial risk factors, health behaviours and blood pressure. Future directions must focus on including and studying males and females (together and separately) to address the existence of possible underlying sex differences.

26 Concluding Remarks

Previous research suggested an association between anger, perceived stress and blood pressure. These assertions were based almost exclusively on cross-sectional data that collected information at one time point and did not manipulate independent psychosocial variables of interest. Using a prospective longitudinal cohort within-group design with an intervention designed to affect the predictor variables of anger and perceived stress, findings from this study failed to demonstrate that change in anger and perceived stress were associated with change in ambulatory blood pressure among untreated stage-1 hypertensive subjects who completed an 8-week mindfulness-

based stress reduction program. Through the use of a stronger study design, this thesis has provided more robust evidence to a divided field and contributed to a better understanding of anger and perceived stress as suspected risk factors for hypertension development.

Anger, perceived stress and blood pressure improved from pre to post intervention but failed to demonstrate association with one another in the correlation and regression analyses. These results, combined with post-hoc analysis of study data and evidence from the literature, suggest a possible model whereby psychosocial variables are indirectly linked to blood pressure through stress coping mechanisms and health behaviours. Future efforts should focus on studying the effects of psychosocially driven stress coping mechanisms and health behaviours and their relationship to blood pressure and hypertension.

Table 2. Studies examining trait anger and blood pressure

Author (year)	Study type	Population	% identify as non-Caucasian	Sample size % male	Anger assessment tool	BPM type	Findings
Schneider (1986) ⁵⁸	Cross-sectional	HTN & WCH, undergraduate students	10%	33 91% male	STAXI	Automated home	No difference between groups
Friedman (2001) ⁹⁹	Cross-sectional	Mild HTN & NTN, middle aged adults	14%	283 100% male	STAXI	ABPM	No difference between groups
Lerman (1989) ⁷²	Cross-sectional	HTN & WCH middle aged adults	25%	98 57% male	STAXI	Non-standardized manual & ABPM	Trait anger more common among hypertensives
Durel (1989) ⁷³	Cross-sectional	NTN & unmedicated mild HTN, middle aged adults	53%	135 60% male	STAXI	ABPM & Automated office	Positive association (females only)
Webb (2002) ¹¹⁰	Cross-sectional	NTN, medicated HTN & unmedicated HTN, middle aged adults	100%	90 0% male	STAXI	Automated office	No difference between groups
Laude (1997) ⁶⁸	Cross-sectional	NTN & mild HTN, middle aged adults	0%	25 20% male	STAXI	Continuous finger BP	High trait anger subjects demonstrated significantly higher BP

Table 4. Methodological inconsistencies in the field of anger expression and blood pressure research

- Differing self-report anger measurement tools
- Unreliable or inconsistent blood pressure measurement tools
- Narrow subject populations
 - For example, primarily white middle aged females⁶⁹ or female university students⁷⁵
- Varying outcome measures
 - Treating blood pressure as a continuous versus dichotomous variable. For example, blood pressure change vs. proportion of anger expression between hypertensive and normotensive subjects.
- Varying anger constructs and subcomponents investigated
 - For example, studying only anger expressed in and anger expressed out and not other anger subcomponents⁶². Or even more narrow, studying only one component of anger expression, such as anger expressed in⁵⁰
- Studying only diastolic blood pressure (an issue comparing older studies to more recent works)^{52, 53}
- Incorrect use of anger definitions and categorizations as defined by Spielberger^{118, 225}
 - Example: Porter et al. in 1999 conducted a study titled “Anger expression and ambulatory blood pressure: A comparison of state and trait measures”. However, using Spielberger’s categorization, state and trait anger are subcomponents of anger

experience, not anger expression. State and trait anger *expression* do not exist in Spielberger's framework for understanding anger. The authors did reliably measure Anger Expression using the appropriate subscale of the STAXI. However, they subsequently combined those findings with other measures of state and trait anger creating "trait and state measures of anger expression", again a concept that does not exist in Spielberger's framework for understanding anger. This cherry picking approach to terminology, definitions and classifications creates considerable confusion.

- Example: Suls et al. published a meta-analysis in 1995 titled "Relationship of Trait Anger to Resting Blood Pressure – a meta analysis". Several different types of anger and hostility self-report questionnaires were included. To review, Spielberger's categories are:

Anger Experience

- State Anger
- Trait Anger

Anger Expression

- Anger Expressed In
- Anger Expressed Out
- Anger Control In
- Anger Control Out

In Suls et al.'s meta-analysis, studies that examined Spielberger's anger expression out subscale were categorized under "Anger Expression measures" and those that examined Spielberger's anger expression in subscale were categorized under "Anger Experience measures". These two categories (anger experience and anger expression) were then grouped together as "Trait Anger Measures" (see Table 6 in Suls et al.). Clearly, the authors did not use terminology or categorizations as defined by the original authors of those terms and categories. In fact, upon closer inspection, this meta-analysis did not look at trait anger in any capacity as defined by Spielberger. Yet, in the abstract it was stated that "anger experience was correlated with elevated BP" and even the study was titled "Relationship of Trait Anger to Resting Blood Pressure...". Upon breaking down the findings and re-framing them using Spielberger's categories, what these authors did find was that anger expressed in (what the authors defined as anger experience filed under the broad category of 'trait anger') was correlated to higher blood pressure. While one can appreciate the authors' attempts to add clarity to the literature, the misuse of definitions and categorizations makes interpreting findings extremely difficult.

Table 5. Studies examining anger expression and blood pressure

Author (year)	Study type	Population	% Not white	Sample size % male	Anger assessment tool	BPM type	Anger Expressed In (AXI)	Anger Expressed Out (AXO)
Harburg (1973) ⁵³	Cross-sectional	HTN & NTN, Middle aged adults	NOT REPORTED	125 100% male	HAX	Standardized manual	Positive association	Negative association
Gentry (1982) ⁵²	Cross-sectional	HTN & NTN, Middle aged adults	51%	1006 49% male	HAX	Standardized manual	Positive association	Negative association
Dimsdale (1986) ⁵⁰	Cross-sectional	NTN, Middle aged adults	45%	507 58% men	Modified HAX	Standardized manual	Positive association (Caucasian males only)	Negative association
Cottingham (1985) ⁵¹	Cross-sectional	NTN, borderline HTN & HTN, middle aged adults	NOT REPORTED	444 NOT REPORTED	Developed own long form anger Q	Standardized manual	Positive association	NOT REPORTED
Helmers (2000) ⁷⁰	Cross-sectional	Borderline HTN, middle aged adults	26%	194 66% male	STAXI	ABPM	Positive association (females only)	No association
Schneider (1986) ⁵⁸	Cross-sectional	HTN & WCH, undergraduate students	10%	33 91% male	STAXI	Automated home	AXI greater among hypertensives	No difference between groups

Hosseini (2011) ⁶⁷	Cross-sectional	HTN & NTN, middle aged adults	100%	300 33% male	STAXI	Manual (standardization level unclear)	HTN subjects more likely express anger inwards	No difference between groups
Haynes (1978) ⁷¹	Cross-sectional	All HTN statuses, Middle aged adults	NOT REPORTED	1822 43% male	HAX	Non-standardized manual	Positive association (females only)	No association
Julius (1986) ⁶¹	Longitudinal follow-up (7 years) * <i>post-hoc</i>	All HTN statuses, Middle aged adults	2%	696 NOT REPORTED	HAX	Non-standardized manual	No association	No association
Markovitz (1991) ⁶⁹	Longitudinal follow-up (3 years)	NTN at baseline, middle aged adults	7%	468 0% male	STAXI	Standardized manual	No association	No association
Everson (1998) ⁶²	Longitudinal follow-up (4 years)	NTN at baseline, middle aged adults	NOT REPORTED	537 100%	STAXI	Standardized manual	Positive association with increased incident risk for HTN	Positive association with increased incident risk for HTN
Friedman (2001) ⁹⁹	Cross-sectional	Mild HTN & NTN, middle aged adults	14%	283 100% male	STAXI	ABPM	No difference between groups	No difference between groups
Webb (2002) ¹¹⁰	Cross-sectional	NTN, medicated HTN & unmedicated HTN, middle aged adults	100%	90 0% male	STAXI	Automated office	No difference between groups	NOT REPORTED

Igna (2009) ²⁴	Cross-sectional	NTN, Middle aged adults	NOT REPORTED	705 100%	AX subscale of STAXI	Standardized manual	No association	Negative association
Hogan (2005) ⁷⁶	Cross-sectional	NTN & HTN middle aged adults	NOT REPORTED	268 50% male	BARQ	Automated office	NOT REPORTED	Negative association (females only)
Lerman (1989) ⁷²	Cross-sectional	HTN & WCH middle aged adults	25%	98 57% male	STAXI	Non-standardized Manual & ABPM	No difference between groups	Higher AXO among HTN subjects than WCH subjects
Harburg (1991) ⁷⁷	Cross-sectional	All HTN statuses, Middle aged adults	52%	720 100% male	STAXI	Standardized manual	No association	Positive association (males only)
Johnson (1984) ⁵⁷	Cross-sectional	NTN, high school students	42%	1060 57% male	STAXI	Automated office	Positive association	Positive association (Caucasian males only)
Ohira (2000) ¹¹⁵	Cross-sectional	All BP levels but primarily NTN, Middle aged males	100% (all Japanese)	790 100%	AX subscale from STAXI	Standardized manual	No association	Negative association (low coping group only)

Acronyms: Blood pressure measurement (BPM), anger expression (AX), anger expressed in (AXI), anger expressed out (AXO) hypertension (HTN), white coat hypertension (WCH), normotension (NTN), State-trait Anger Expression Inventory (STAXI), ambulatory blood pressure monitoring (ABPM), blood pressure (BP), diastolic blood pressure (DBP), systolic blood pressure (SBP), systolic ambulatory blood pressure (SABP), Harburg Anger-In/Anger-Out Scale (HAX), Behavioral Anger Response Questionnaire (BARQ)

Table 6. Studies examining perceived stress and blood pressure

Author (year)	Study type	Population	% Not white	Sample size % male	Stress assessment tool	BPM type	Finding
Huang (2011) ⁸³	Cross-sectional	HTN, middle aged adults	100%	307 52% male	Cohen's PSS	ABPM	Positive association
Wright (2011) ⁸⁴	Longitudinal follow-up (6 years)	NTN, middle aged adults	NOT REPORTED	170 100% male	Cohen's PSS	Standardized manual	Positive association
Logan (2012) ¹³⁴	Cross-sectional	NTN, middle aged adults	100%	102 40% male	Cohen's PSS	Automated office	No association
Ginty (2011) ¹³⁵	Cross-sectional	HTN status not reported (but likely NTN), university study students	NOT REPORTED	100 38% male	Cohen's PSS	Automated office	No association
Rod (2009) ⁸⁵	Longitudinal follow-up (10 years)	HTN status not reported , middle aged adults	NOT REPORTED	7066 42% male	Non validated PSS 2 item Q that addressed level of PS & frequency of PS	N/A (authors measured change in HTN status)	High PS association with HTN at F/U

Pollard (2007) ⁷⁸	Cross-sectional	NTN, middle aged adults	NOT REPORTED	33 0% male	Non validated PSS 1 item Q on PS, 1 = not at all stressed to 7 = very stressed.	Automated home	Positive association
Kario (2002) ⁷⁹	Cross-sectional	NTN, middle aged adults, PS at work vs. PS at home	100%	135 0% male	Non validated PSS 1 item Q on PS, 0 = low stress to 10 = high stress	ABPM	Higher work BP & sleep SBP among subjects reporting high PS at work vs. high PS at home
James (2001) ⁸⁰	Cross-sectional	NTN, young - middle aged adults, PS at work vs. PS at home	61.5%	91 0% male	Non validated PSS 1 item Q on PS, 0 = low stress to 10 = high stress	ABPM	Work-stressed subjects had higher BP than home-stressed subjects
Matthews (1992) ⁸¹	Cross-sectional	NTN, middle aged adults	0%	48 31 % male	Non validated PSS 1 item Q on PS, 1 = not at all to 5 = very much	ABPM	Positive association
Langewitz (1987) ⁸²	Cross-sectional	NTN, NOT REPORTED	NOT REPORTED	34 NOT REPORTED	Non validated PSS 1 item Q on PS, 1 = completely relaxed to 5 = extremely stressed	ABPM	Positive association

Table 7. Study inclusion and exclusion criteria. Adapted from the HARMONY protocol manuscript¹

<p>Inclusion criteria:</p> <ol style="list-style-type: none"> 1. Age 20 to 75 years. 2. Stage-1 hypertensive as determined by ABPM at baseline (daytime $\geq 135/85$ mmHg or 24-hour ABPM $\geq 130/80$ mmHg). 3. Willing and able to participate in the MBSR program. 4. Willing to be followed for safety BP checks if necessary. 5. Willing to accept a possible waiting period for MBSR. 6. Written informed consent. 7. Able to participate in mindfulness meditation program.
<p>Exclusion criteria:</p> <ol style="list-style-type: none"> 1. Use of antihypertensive medication within 6 months of the screening ABPM. 2. Screening office BP $> 180/100$ mmHg and ABPM $\geq 160/100$ mmHg. 3. Diabetes. 4. Secondary hypertension. 5. Renal disease (glomerular filtration rate < 60 ml/min or overt nephropathy). 6. History of heart attack. 7. Stroke or transient ischemic attack. 8. Re-vascularization procedure. 9. Active malignant disease (except non-melanoma skin cancer). 10. Epileptic seizure 6 months before the screening visit. 11. Congestive heart failure. 12. Severe liver disease. 13. Pregnancy or lactation period. 14. Participation in a clinical trial or receipt of investigational compound or treatment in the 3 months prior to the initial screening visit. 15. Planned elective surgery during the study period except for cataract surgery. 16. Inability or unwillingness to perform ABPMs. <p>ABPM = ambulatory blood pressure monitoring, MBSR = mindfulness-based stress reduction, BP = blood pressure.</p>

Table 8. Brief overview of the STAXI-2 scales and subscales. Adapted from STAXI-2 manual²

STAXI-2 scale/subscale	Number of questions	Scale/subscale range	Description of scale/subscale
State Anger (S-Ang)	15	15-60	Measures the intensity of angry feelings and the extent to which a person feels like expressing anger at a particular time
Feeling Angry (S-Ang/F)	5	5-20	Measures the intensity of the angry feelings the person is currently experiencing
Feel Like Expressing Anger Verbally (S-Ang/F)	5	5-20	Measures the intensity of current feelings related to the verbal expression of anger
Feel Like Expressing Anger Physically (S-Ang/F)	5	5-20	Measures the intensity of current feelings related to the physical expression of anger
Trait Anger (T-Ang)	10	10-40	Measures how often angry feelings are experienced over time
Angry Temperament (T-Ang/T)	4	4-16	Measures the disposition to experience angry without specific provocation
Angry Reaction (T-Ang/R)	4	4-16	Measures the frequency that angry feelings are experienced in situations that involve frustration and/or negative evaluations.
Anger Expression In (AXI)	8	8-32	Measures how often angry feelings are experienced but not expressed (suppressed)
Anger Expression Out (AXO)	8	8-32	Measures how often angry feelings are expressed in verbally or physically aggressive behaviour
Anger Control In (ACI)	8	8-32	Measures how often a person attempts to control angry feelings by calming down or cooling off
Anger Control Out (ACO)	8	8-32	Measures how often a person controls the outwards expression of angry feelings
Anger Expression Index (AXX)	32	0-96	Provides a general index of anger expression based on responses to the AXI, AXO, ACI and ACO items.

Table 9. Baseline demographic data

Characteristics	Result
Sex (% male)	37%
Age (years)	56 (11.4)
Waist circ. (cm)	90 (11.5)
BMI (kg/m ²)	28 (4.8)
Office SBP	136 (13.0)
Office DBP	86 (7.2)
24 HR SBP	135 (7.5)
24 HR DBP	81 (5.6)
Daytime SBP	141 (7.5)
Daytime DBP	86 (6.0)
Nighttime SBP	122 (10.3)
Nighttime DBP	71 (7.0)
Family History of Hypertension (%)	62%
Family history of premature coronary heart disease	28%
≥ Post-Secondary Education (% yes)	86%
Working (%)	80%
Drinks > 10/week	20%
Use relaxation techniques on a regular basis	17%
Vigorous exercise	64%
Moderate exercise	91%
Self-identify as Caucasian	82%
<i>Data is reported in mean (SD) for continuous variables. SBP = systolic blood pressure, DBP = diastolic blood pressure. All BP measured in mmHg. Vigorous exercise = participation in any weekly vigorous aerobic exercise lasting 20 ≥ minutes. Moderate exercise = participation in any weekly moderate exercise lasting ≥ 30 minutes.</i>	

Table 10. Baseline psychosocial data

Psychometric Scale	Score	%ile Normal Adults*
Trait Anger	17 (5)	45 th
Angry Temperament	7 (3)	65 th
Angry Reaction	8 (3)	35 th
Anger Expression In	17 (4)	60 th
Anger Expression Out	14 (3)	40 th
Anger Control In	22 (6)	45 th
Anger Control Out	23 (5)	45 th
Anger Expression Index (AX index)	34 (14)	50 th
Perceived Stress Scale	24 (8)	
<i>Data is reported in mean (SD). Percentile ranks were obtained from the State-Trait Anger Expression Inventory-2 Professional Manual. No such percentile ranking exists for the Perceived Stress Scale.</i>		

Table 11. MBSR class attendance and homework completion

Average No. MBSR sessions attended out of 8 (classes)	6 (1)
Average # minutes of MBSR homework/day (minutes)	32 (15.2)
<i>Data is reported in mean (SD).</i>	

Table 12. Correlations between baseline trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards, perceived stress and baseline 24-hour blood pressure

Anger Measure	All Subjects		Female		Male	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
T-Ang	-0.018	-0.043	-0.040	0.017	0.040	-0.139
T-Ang/T	-0.027	-0.052	-0.087	-0.030	0.078	-0.139
T-Ang/R	-0.064	-0.002	-0.030	0.065	-0.096	-0.118
AXI	-0.042	0.046	0.098	-0.002	-0.223	0.183
AXO	-0.120	-0.104	-0.079	-0.099	-0.163	-0.117
PS	0.081	0.098	0.203	0.155	-0.121	-0.035

Note. * $p < 0.10$, ** $p < 0.05$. T-Ang= Trait Anger, T-Ang/T = Trait Anger Temperament, T-Ang/R = Trait Anger Reaction, AXI = Anger Expression Inwards, AXO = Anger Expression Outwards, PS = Perceived Stress.

Table 13. Correlations between baseline trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards, perceived stress and baseline daytime blood pressure

Anger Measure	All Subjects		Female		Male	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
T-Ang	-0.050	-0.048	-0.093	0.032	0.015	-0.172
T-Ang/T	-0.082	-0.051	-0.142	0.002	-0.008	-0.148
T-Ang/R	-0.084	0.002	-0.040	0.107	-0.133	-0.171
AXI	-0.137	-0.032	0.004	-0.076	-0.340 **	0.072
AXO	-0.160	-0.115	-0.177	-0.136	-0.129	-0.081
PS	0.022	0.065	0.143	0.142	-0.174	-0.106

Note. * $p < 0.10$, ** $p < 0.05$. T-Ang= Trait Anger, T-Ang/T = Trait Anger Temperament, T-Ang/R = Trait Anger Reaction, AXI = Anger Expression Inwards, AXO = Anger Expression Outwards, PS = Perceived Stress.

Table 18. Changes in trait anger, trait anger temperament, trait anger reaction, anger expressed inwards, anger expressed outwards, perceived stress, anger control inwards, anger control outwards and the anger expression index from pre to post intervention

	Pre	Post	Average Change	Change p-value
Trait anger				
All Subjects	17.03 (5.26)	15.56 (4.00)	-1.47 (3.30)	0.0003 *
Female	16.82 (4.83)	15.55 (3.80)	-1.27 (3.18)	0.0076 *
Male	17.42 (6.05)	15.58 (4.44)	-1.85 (3.55)	0.0137 *
Trait anger temperament				
All Subjects	6.53 (2.67)	5.73 (1.91)	-0.80 (1.60)	0.0025 *
Female	6.37 (2.44)	5.57 (1.77)	-0.80 (1.62)	0.0241 *
Male	6.85 (3.08)	6.04 (2.14)	-0.81 (1.60)	0.0768
Trait anger reaction				
All Subjects	7.71 (2.57)	7.22 (2.28)	-0.50 (1.79)	0.0190 *
Female	7.73 (2.41)	7.42 (2.11)	-0.33 (1.75)	0.1944
Male	7.65 (2.90)	6.85 (2.57)	-0.81 (1.86)	0.0357 *
Anger expression inwards				
All Subjects	16.73 (4.43)	14.84 (3.77)	-1.89 (4.20)	0.0002 *
Female	16.10 (4.40)	14.45 (3.52)	-1.65 (3.55)	0.0021 *
Male	17.92 (4.34)	15.58 (4.18)	-2.35 (5.26)	0.0318 *
Anger expression outwards				
All Subjects	13.97 (3.18)	13.15 (2.60)	-0.83 (2.93)	0.0171 *
Female	13.82 (3.15)	12.80 (2.50)	-1.02 (2.87)	0.0163 *
Male	14.27 (3.28)	13.81 (2.71)	-0.46 (3.08)	0.4513
Perceived Stress				
All Subjects	24.04 (8.18)	20.77 (6.34)	-3.21 (7.24)	0.0001 *
Female	23.60 (8.51)	20.50 (6.58)	-2.98 (7.09)	0.0038 *
Male	24.77 (7.66)	21.24 (5.98)	-3.62 (7.60)	0.0160 *
Anger control inwards				
All Subjects	21.67 (5.82)	24.76 (4.68)	3.09 (4.45)	<.0001 *
Female	22.47 (5.83)	25.45 (4.87)	2.98 (4.30)	<.0001 *
Male	20.15 (5.60)	23.46 (4.09)	3.31 (4.81)	0.0017 *
Anger control outwards				
All Subjects	23.31 (5.42)	24.87 (5.01)	1.56 (3.59)	0.0003 *
Female	23.76 (5.07)	25.59 (4.81)	1.84 (3.60)	0.0008 *
Male	22.46 (4.83)	25.30 (5.18)	1.04 (3.57)	0.1507
Anger expression index				
All Subjects	33.73 (14.94)	26.36 (12.20)	-7.37 (10.66)	<.0001 *
Female	31.69 (15.54)	24.20 (12.21)	-7.49 (9.86)	<.0001 *
Male	37.58 (13.16)	30.42 (11.31)	-7.15 (12.24)	0.0063 *
* indicates significant difference from baseline to after the intervention (p < 0.05). All psychometric information represents raw score values.				

Table 19. Changes in the proportion of those scoring greater than or equal to the 75th percentile in trait anger, trait anger temperament, trait anger reaction, anger expressed inwards, anger expressed outwards, anger control inwards, anger control outwards and anger expression from pre to post intervention

	Proportion $\geq$ 75th percentile (PRE)	Proportion $\geq$ 75th percentile (POST)	p-value
Trait Anger			
All Subjects	24%	13%	0.0215*
Female	22%	12%	0.1250
Male	31%	19%	0.2500
Trait Anger Temperament			
All Subjects	32%	23%	0.0391*
Female	37%	20%	0.0078*
Male	35%	31%	1.0000
Trait Anger Reaction			
All Subjects	16%	7%	0.0391*
Female	15%	6%	0.2188
Male	19%	15%	1.0000
Anger Expression Inwards			
All Subjects	32%	12%	0.0015*
Female	41%	12%	0.0013*
Male	54%	27%	0.0654
Anger Expression Outwards			
All Subjects	17%	8%	0.0654
Female	24%	16%	0.3438
Male	23%	15%	0.6250
Anger Control Inwards			
All Subjects	19%	35%	0.0042*
Female	18%	27%	0.0225*
Male	12%	19%	0.6250
Anger Control Outwards			
All Subjects	27%	36%	0.1185
Female	29%	41%	0.1094
Male	12%	15%	1.0000
Anger Expression Index			
All Subjects	33%	12%	0.0004*
Female	35%	12%	0.0010*
Male	42%	31%	0.3750
<p>* indicates significant difference from baseline to after the intervention ($p < 0.05$)</p> <p>Note: According to the STAXI manual, scores differentiating $<$ or $\geq$ 75th percentile differ depending on the sex profile of the group under study (i.e., males and females together, females only or males only). Appropriate score cut-offs were used for each category in this table. As the cut-off values vary depending on sex-profile of the group, the group proportion of those scoring $\geq$ 75th percentile will not be the average of male and female proportions.</p>			

Table 20. Internal consistency measures using Cronbach's α at baseline and post intervention for trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards, perceived stress, anger control inwards and anger control outwards

	Baseline α Coefficient	Post intervention α Coefficient
Trait Anger	0.88	0.85
Trait Anger Temperament	0.89	0.85
Trait Anger Reaction	0.78	0.78
Anger Expression In	0.79	0.76
Anger Expression Out	0.74	0.62
Perceived Stress	0.90	0.86
Anger Control In	0.92	0.89
Anger Control Out	0.87	0.89
Only standardized scores for Cronbach's α have been reported. Note: in accordance with Nunnally & Bernstein's suggested value of 0.70 indicating sufficient internal consistency, no p-values have been assigned to α coefficients.		

Table 21. Changes in 24-hour, daytime and nighttime blood pressure from pre to post intervention

	Pre	Post	Average Change**	Change p-value
24-hour systolic				
All Subjects	134.82 (7.52)	132.78 (8.62)	-1.89 (6.83)	0.0124 *
Female	135.11 (7.77)	131.82 (7.66)	-3.09 (6.26)	0.0006 *
Male	134.29 (7.13)	134.53 (10.05)	0.30 (7.39)	0.8256
24-hour diastolic				
All Subjects	81.41 (5.60)	80.55 (7.32)	-0.73 (4.54)	0.1424
Female	81.43 (5.99)	80.18 (7.79)	-1.11 (4.52)	0.0741
Male	81.39 (4.91)	81.23 (7.09)	-0.03 (4.58)	0.9685
Daytime systolic				
All Subjects	140.60 (7.52)	138.40 (9.05)	-2.12 (7.09)	0.0072 *
Female	140.68 (7.49)	137.04 (8.00)	-3.47 (6.16)	0.0001 *
Male	140.45 (7.70)	140.09 (10.40)	0.37 (8.05)	0.8047
Daytime diastolic				
All Subjects	86.07 (6.01)	85.06 (7.91)	-0.92 (4.83)	0.0832
Female	86.14 (6.36)	84.71 (8.21)	-1.31 (4.70)	0.0436 *
Male	85.94 (5.43)	85.70 (7.43)	-0.20 (5.05)	0.8299
Nighttime systolic				
All Subjects	121.95 (10.34)	120.79 (9.94)	-0.92 (9.38)	0.3695
Female	123.36 (10.93)	120.60 (9.49)	-2.49 (9.81)	0.0652
Male	119.42 (8.79)	121.13 (10.88)	1.97 (7.88)	0.1819
Nighttime diastolic				
All Subjects	71.01 (7.04)	71.00 (7.40)	0.13 (5.43)	0.8266
Female	71.32 (7.06)	70.76 (7.48)	-0.42 (5.65)	0.5851
Male	70.45 (7.09)	71.43 (7.35)	1.13 (4.94)	0.2189
* indicates significant difference from baseline to after the intervention ($p < 0.05$). All blood pressure measured in mmHg				
** refers to the average of all blood pressure differences from pre to post intervention				

Table 22. Correlations between changes in trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards, perceived stress and change in 24-hour blood pressure from pre to post intervention

	All Subjects		Female		Male	
Anger Measure	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
T-Ang	-0.043	-0.008	0.043	0.010	-0.088	-0.001
T-Ang/T	0.135	0.093	0.195	0.103	0.040	0.082
T-Ang/R	-0.148	-0.058	-0.150	-0.055	-0.075	-0.023
AXI	0.042	0.147	0.137	0.257 *	-0.018	0.038
AXO	0.041	0.021	0.152	-0.002	-0.156	0.037
PS	-0.031	0.020	-0.173	-0.049	0.197	0.155

Note. * p<0.10, **p<0.05. T-Ang= Trait Anger, T-Ang/T = Trait Anger Temperament, T-Ang/R = Trait Anger Reaction, AXI = Anger Expression Inwards, AXO = Anger Expression Outwards, PS = Perceived Stress.

Table 23. Correlations between changes in trait anger, trait anger temperament, trait anger reaction, anger expression inwards, anger expression outwards, perceived stress and change in daytime blood pressure from pre to post intervention

	All Subjects		Female		Male	
Anger Measure	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
T-Ang	-0.091	-0.024	-0.020	-0.001	-0.111	-0.027
T-Ang/T	0.148	0.073	0.220	0.072	0.047	0.024
T-Ang/R	-0.164	-0.066	-0.168	-0.084	-0.087	-0.006
AXI	0.004	0.099	0.183	0.279 *	-0.123	-0.088
AXO	-0.019	0.005	0.124	0.029	-0.251	-0.053
PS	-0.039	0.004	-0.139	-0.007	0.113	0.037

Note. * $p < 0.10$, ** $p < 0.05$. T-Ang= Trait Anger, T-Ang/T = Trait Anger Temperament, T-Ang/R = Trait Anger Reaction, AXI = Anger Expression Inwards, AXO = Anger Expression Outwards, PS = Perceived Stress.

Table 25. Correlations between changes in anger control inwards, anger control outwards, anger expression index and change in 24-hour blood pressure from pre to post intervention

Anger Measure	All Subjects		Female		Male	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
ACI	0.011	0.027	-0.240	-0.168	0.328	0.330
ACO	-0.112	-0.104	-0.227	-0.171	0.118	0.050
AXX	0.061	0.087	0.282 *	0.228	-0.210	-0.119

Note. * $p < 0.10$, ** $p < 0.05$. ACI = Anger Control Inwards, ACO = Anger Control Outwards, AXX = Anger Expression Index.

Table 26. Correlations between changes in anger control inwards, anger control outwards, anger expression index and change in daytime blood pressure from pre to post intervention

Anger Measure	All Subjects		Female		Male	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
ACI	0.051	0.073	-0.272 *	-0.165	0.423 **	0.428 **
ACO	-0.113	-0.071	-0.242 *	-0.159	0.120	0.110
AXX	0.013	0.034	0.310 **	0.239	-0.316	-0.251

Note. * $p < 0.10$, ** $p < 0.05$. ACI = Anger Control Inwards, ACO = Anger Control Outwards, AXX = Anger Expression Index.

Table 27. Correlations between changes in anger control inwards, anger control outwards, anger expression index and change in nighttime blood pressure from pre to post intervention

Anger Measure	All Subjects		Female		Male	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
ACI	-0.168	-0.116	-0.213	-0.155	-0.122	-0.062
ACO	-0.151	-0.149	-0.149	-0.106	-0.092	-0.200
AXX	0.171	0.152	0.183	0.140	0.160	0.178

Note. * $p < 0.10$, ** $p < 0.05$. ACI = Anger Control Inwards, ACO = Anger Control Outwards, AXX = Anger Expression Index.

Table 28. Effects of post intervention trait anger on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.87 (0.13)	<.0001	1.02 (0.12)	<.0001	0.97 (0.17)	<.0001	0.92 (0.14)	<.0001			1.26 (0.28)	0.001
Pre MBSR T-Ang*	-0.10 (0.27)	0.71	-0.08 (0.18)	0.64	-0.26 (0.33)	0.44	-0.19 (0.23)	0.42			0.01 (0.53)	0.98
Post MBSR T-Ang	0.11 (0.39)	0.78	-0.01 (0.26)	0.97	0.08 (0.48)	0.86	-0.27 (0.34)	0.43			0.17 (0.85)	0.85
Age	-0.07 (0.08)	0.41	-0.11 (0.05)	0.04	-0.22 (0.10)	0.04	-0.19 (0.07)	0.01			-0.05 (0.11)	0.65
BMI*	0.03 (0.18)	0.85	-0.14 (0.12)	0.23	-0.14 (0.17)	0.42	-0.17 (0.11)	0.14			0.16 (0.50)	0.75
Sex (female)	-2.67 (1.95)	0.18	-0.28 (1.31)	0.83								
FHx premature CVD (Yes)	-3.13 (1.98)	0.12	-3.32 (1.33)	0.02	-2.88 (2.30)	0.22	-3.03 (1.62)	0.07			-4.18 (3.19)	0.21
Drink $\geq$ 10 drinks/wk (Yes)*	3.43 (2.27)	0.14	3.28 (1.52)	0.04	4.73 (2.69)	0.09	6.02 (1.91)	0.004			-0.10 (3.05)	0.97
Relaxation regularly (Yes)*	-0.82 (2.70)	0.76	-1.57 (1.81)	0.39	-1.50 (2.33)	0.52	-2.78 (1.65)	0.10			0.00	
Race (Caucasian)	-1.18 (2.63)	0.66	-2.96 (1.76)	0.10	1.21 (2.61)	0.65	-2.86 (1.85)	0.13			-1.85 (4.60)	0.69
Education (> high school)	3.15 (2.58)	0.23	0.94 (1.72)	0.59	2.60 (2.62)	0.33	1.61 (1.85)	0.39			3.75 (7.67)	0.63
Regular Exercise (Yes)*	-5.41 (2.94)	0.07	-2.83 (1.96)	0.16	-2.02 (2.98)	0.50	-1.37 (2.07)	0.51			-6.67 (5.62)	0.26

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 29. Effects of post intervention trait anger on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.78 (0.13)	<.0001	0.89 (0.11)	<.0001	0.90 (0.16)	<.0001	0.83 (0.12)	<.0001				
Pre MBSR T-Ang*	-0.03 (0.28)	0.93	-0.06 (0.19)	0.75	-0.12 (0.35)	0.74	-0.10 (0.24)	0.67				
Post MBSR T-Ang	-0.03 (0.41)	0.93	-0.10 (0.28)	0.73	-0.15 (0.51)	0.76	-0.53 (0.35)	0.15				
Age	-0.09 (0.09)	0.30	-0.15 (0.06)	0.01	-0.23 (0.11)	0.04	-0.24 (0.07)	0.002				
BMI*	0.06 (0.18)	0.73	-0.11(0.13)	0.40	-0.06 (0.17)	0.73	-0.12 (0.12)	0.30				
Sex (female)	-4.52 (2.03)	0.03	-1.21 (1.39)	0.39								
FHx premature CVD (Yes)	-2.77 (2.07)	0.19	-3.01 (1.42)	0.04	-3.94 (2.46)	0.12	-3.93 (1.68)	0.03				
Drink $\geq$ 10 drinks/wk (Yes)*	2.98 (2.37)	0.21	3.19 (1.62)	0.055	2.52 (2.89)	0.39	4.71 (2.00)	0.03				
Relaxation regularly (Yes)*	1.55 (2.82)	0.58	-0.10 (1.94)	0.96	1.85 (2.48)	0.46	-0.85 (1.71)	0.62				
Race (Caucasian)	-2.03 (2.74)	0.46	-3.05 (1.89)	0.11	0.82 (2.76)	0.77	-2.43 (1.91)	0.21				
Education (> high school)	2.09 (2.69)	0.44	1.03 (1.83)	0.58	2.60 (2.79)	0.36	2.41 (1.92)	0.22				
Regular Exercise (Yes)*	-5.37 (3.08)	0.09	-3.58 (2.10)	0.10	-1.49 (3.12)	0.64	-1.42 (2.17)	0.52				

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. Family history (FHx), cardiovascular disease (CVD).

Table 30. Effects of post intervention trait anger temperament on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.89 (0.13)	<.0001	1.00 (0.12)	<.0001	1.05 (0.16)	<.0001	0.87 (0.15)	<.0001			1.22 (0.28)	0.001
Pre MBSR T-Ang/T*	-0.71 (0.55)	0.21	-0.45 (0.38)	0.24	-1.76 (0.62)	0.01	-0.77 (0.52)	0.15			-0.66 (0.93)	0.50
Post MBSR T-Ang/T	1.14 (0.82)	0.17	0.40 (0.56)	0.47	2.71 (0.95)	0.01	0.47 (0.80)	0.56			1.33 (1.56)	0.41
Age	-0.08 (0.08)	0.34	-0.11 (0.05)	0.04	-0.29 (0.10)	0.01	-0.19 (0.07)	0.01			-0.04 (0.11)	0.73
BMI*	0.04 (0.17)	0.81	-0.13 (0.12)	0.26	-0.21 (0.15)	0.17	-0.19 (0.12)	0.11			0.18 (0.40)	0.66
Sex (female)	-2.10 (1.95)	0.29	-0.12 (1.32)	0.93								
FHx premature CVD (Yes)	-3.05 (1.93)	0.12	-3.24 (1.31)	0.02	-1.63 (2.00)	0.42	-2.33 (1.64)	0.17			-3.91 (3.03)	0.22
Drink $\geq$ 10 drinks/wk (Yes)*	3.64 (2.26)	0.11	3.52 (1.53)	0.03	5.32 (2.36)	0.03	5.97 (1.95)	0.005			-0.57 (3.17)	0.86
Relaxation regularly (Yes)*	-1.38 (2.69)	0.61	-1.86 (1.82)	0.31	-2.97 (2.15)	0.18	-2.98 (1.77)	0.10			0.00	
Race (Caucasian)	-1.82 (2.60)	0.49	-3.33 (1.76)	0.06	-0.05 (2.29)	0.98	-3.30 (1.88)	0.09			-2.59 (4.55)	0.58
Education (> high school)	2.84 (2.56)	0.27	0.66 (1.72)	0.70	-1.34 (2.58)	0.61	-0.19 (2.13)	0.93			6.11 (6.90)	0.39
Regular Exercise (Yes)*	-5.35 (2.88)	0.07	-2.89 (1.95)	0.14	-3.23 (2.67)	0.24	-1.58 (2.16)	0.47			-6.93 (5.09)	0.20

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 31. Effects of post intervention trait anger temperament on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.79 (0.13)	<.0001	0.87 (0.11)	<.0001	0.95 (0.16)	<.0001	0.80 (0.14)	<.0001				
Pre MBSR T-Ang/T*	-0.56 (0.58)	0.34	-0.42 (0.41)	0.30	-1.47 (0.70)	0.04	-0.62 (0.56)	0.28				
Post MBSR T-Ang/T	0.77 (0.86)	0.38	0.31 (0.60)	0.61	2.05 (1.07)	0.07	0.04 (0.85)	0.96				
Age	-0.09 (0.09)	0.28	-0.14 (0.06)	0.02	-0.28 (0.10)	0.01	-0.23 (0.08)	0.01				
BMI*	0.07 (0.18)	0.70	-0.10 (0.13)	0.44	-0.12 (0.16)	0.48	-0.15 (0.13)	0.26				
Sex (female)	-4.15 (2.06)	0.049	-1.08 (1.41)	0.45								
FHx premature CVD (Yes)	-2.63 (2.04)	0.20	-2.91 (1.40)	0.04	-2.58 (2.27)	0.27	-3.06 (1.77)	0.09				
Drink $\geq$ 10 drinks/wk (Yes)*	3.27 (2.39)	0.18	3.46 (1.64)	0.04	3.10 (2.68)	0.26	4.73 (2.12)	0.03				
Relaxation regularly (Yes)*	1.12 (2.83)	0.69	-0.34 (1.97)	0.86	0.64 (2.42)	0.79	-0.93 (1.92)	0.63				
Race (Caucasian)	-2.60 (2.73)	0.35	-3.48 (1.90)	0.07	-0.42 (2.56)	0.87	-3.11 (2.01)	0.13				
Education (> high school)	1.80 (2.70)	0.51	0.77 (1.84)	0.68	-1.06 (2.92)	0.72	0.58 (2.30)	0.80				
Regular Exercise (Yes)*	-5.41 (3.05)	0.08	-3.66 (2.09)	0.09	-2.42 (2.97)	0.42	-1.62 (2.35)	0.50				

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. Family history (FHx), cardiovascular disease (CVD).

Table 32. Effects of post intervention trait anger temperament as a multilevel categorical predictor variable divided into quartiles on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.89 (0.13)	<.0001	1.00 (0.12)	<.0001	1.01 (0.17)	<.0001	0.92 (0.16)	<.0001			1.67 (0.28)	0.0001
Pre MBSR T-Ang/T*	-0.64 (0.58)	0.28	-0.40 (0.39)	0.31	-1.57 (0.62)	0.02	-0.83 (0.49)	0.11			0.67 (0.85)	0.45
Post MBSR T-Ang/T												
Q1 vs Q4	-5.11 (3.78)	0.18	-1.81 (2.54)	0.48	-9.81 (3.89)	0.02	-2.16 (3.09)	0.49			0.26 (6.77)	0.97
Q2 vs Q4	-2.89 (4.01)	0.48	-1.15 (2.71)	0.67	-9.69 (4.27)	0.03	-3.55 (3.38)	0.30			10.17 (6.64)	0.16
Q3 vs Q4	-2.64 (3.24)	0.42	-2.06 (2.19)	0.35	-5.81 (3.08)	0.07	-2.31 (2.42)	0.35			0.96 (6.08)	0.88
Age	-0.09 (0.08)	0.30	-0.11 (0.05)	0.04	-0.25 (0.10)	0.02	-0.19 (0.07)	0.02			-0.10 (0.10)	0.32
BMI*	0.03 (0.18)	0.88	-0.15 (0.12)	0.23	-0.18 (0.16)	0.28	-0.20 (0.12)	0.12			0.35 (0.39)	0.40
Sex (female)	-2.31 (1.99)	0.25	-0.06 (1.35)	0.96								
FHx premature CVD (Yes)	-3.10 (1.99)	0.13	-3.43 (1.35)	0.01	-2.07 (2.14)	0.34	-2.66 (1.68)	0.13			-6.77 (2.75)	0.03
Drink ≥ 10 drinks/wk (Yes)*	3.49 (2.53)	0.17	3.15 (1.71)	0.07	6.77 (2.89)	0.03	6.44 (2.30)	0.01			-2.33 (3.49)	0.52
Relaxation regularly (Yes)*	-1.05 (2.85)	0.71	-1.55 (1.92)	0.42	-3.11 (2.37)	0.20	-3.20 (1.89)	0.10			0.00	
Race (Caucasian)	-1.37(2.85)	0.63	-2.92 (1.93)	0.14	-0.32 (2.59)	0.90	-3.66 (2.09)	0.09			7.10 (5.04)	0.19
Education (> high school)	2.48 (2.67)	0.36	0.76 (1.79)	0.68	0.04 (2.57)	0.99	0.13 (2.02)	0.95			3.79 (5.13)	0.48
Regular Exercise (Yes)*	-5.23 (2.99)	0.09	-2.85 (2.01)	0.16	-4.02 (3.02)	0.20	-2.25 (2.33)	0.34			-7.82 (4.29)	0.10

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD). Q1= bottom 25th percent quartile, Q2=second quartile, Q3=third quartile, Q4= top 25th percent quartile.

Table 33. Effects of post intervention trait anger temperament as a multilevel categorical predictor variable divided into quartiles on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.79 (0.13)	<.0001	0.87 (0.11)	<.0001	0.94 (0.16)	<.0001	0.86 (0.14)	<.0001			1.59 (0.26)	0.0001
Pre MBSR T-Ang/T*	-0.40 (0.61)	0.51	-0.41 (0.42)	0.34	-1.48 (0.68)	0.04	-0.91 (0.52)	0.09			1.01 (0.90)	0.29
Post MBSR T-Ang/T												
Q1 vs Q4	-2.95 (3.95)	0.46	-1.52 (2.74)	0.58	-8.11 (4.27)	0.07	-1.77 (3.25)	0.59			0.43 (7.02)	0.95
Q2 vs Q4	-0.15 (4.21)	0.97	-0.97 (2.91)	0.74	-9.06 (4.75)	0.07	-4.40 (3.59)	0.23			13.40 (7.02)	0.09
Q3 vs Q4	-0.59 (3.40)	0.86	-1.54 (2.36)	0.52	-4.17 (3.39)	0.23	-2.37 (2.55)	0.36			-0.68 (6.29)	0.92
Age	-0.10 (0.09)	0.27	-0.15 (0.06)	0.02	-0.26 (0.10)	0.02	-0.22 (0.08)	0.01			-0.12 (0.10)	0.27
BMI*	0.06 (0.19)	0.73	-0.11 (0.13)	0.41	-0.08 (0.17)	0.66	-0.15 (0.13)	0.27			0.53 (0.42)	0.23
Sex (female)	-4.49 (2.09)	0.04	-1.04 (1.44)	0.47								
FHx premature CVD (Yes)	-2.54 (2.09)	0.23	-3.04 (1.45)	0.04	-2.86 (2.37)	0.24	-3.32 (1.77)	0.07			-6.01 (2.84)	0.06
Drink $\geq$ 10 drinks/wk (Yes)*	2.98 (2.66)	0.27	3.21 (1.84)	0.09	5.12 (3.18)	0.12	5.52 (2.42)	0.03			-3.22 (3.72)	0.41
Relaxation regularly (Yes)*	1.60 (2.99)	0.59	-0.13 (2.08)	0.95	0.01 (2.59)	1.00	-1.43 (1.98)	0.48			0.00	
Race (Caucasian)	-1.92 (2.99)	0.52	-3.22 (2.08)	0.13	-1.32 (2.83)	0.64	-3.86 (2.17)	0.09			8.26 (5.40)	0.16
Education (> high school)	1.44 (2.80)	0.61	0.81 (1.93)	0.67	-0.17 (2.81)	0.95	0.45 (2.13)	0.83			4.24 (5.38)	0.45
Regular Exercise (Yes)*	-4.99 (3.16)	0.12	-3.63 (2.17)	0.10	-3.67 (3.30)	0.28	-2.70 (2.46)	0.28			-8.11 (4.43)	0.10

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD). Q1= bottom 25th percent quartile, Q2=second quartile, Q3=third quartile, Q4= top 25th percent quartile.

Table 34. Effects of post intervention trait anger reaction on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.86 (0.13)	<.0001	1.02 (0.12)	<.0001	1.01 (0.18)	<.0001	0.91 (0.15)	<.0001			1.33 (0.27)	0.0004
Pre MBSR T-Ang/R*	-0.08 (0.51)	0.88	-0.11 (0.34)	0.75	-0.19 (0.52)	0.71	-0.45 (0.38)	0.24			1.01 (1.03)	0.35
Post MBSR T-Ang/R	0.09 (0.61)	0.88	0.04 (0.41)	0.92	-0.58 (0.64)	0.37	-0.36 (0.46)	0.44			-0.78 (1.28)	0.55
Age	-0.07 (0.09)	0.44	-0.11 (0.06)	0.06	-0.25 (0.11)	0.03	-0.19 (0.07)	0.01			-0.06 (0.11)	0.57
BMI*	0.06 (0.20)	0.76	-0.12 (0.13)	0.35	-0.15 (0.19)	0.46	-0.13 (0.13)	0.33			0.49 (0.54)	0.38
Sex (female)	-2.68 (1.99)	0.18	-0.23 (1.34)	0.86								
FHx premature CVD (Yes)	-3.27 (2.01)	0.11	-3.42 (1.35)	0.01	-3.70 (2.26)	0.11	-3.39 (1.63)	0.048			-3.88 (3.11)	0.24
Drink $\geq$ 10 drinks/wk (Yes)*	3.25 (2.27)	0.16	3.06 (1.53)	0.052	4.74 (2.68)	0.09	6.17 (1.95)	0.004			-0.11 (2.96)	0.97
Relaxation regularly (Yes)*	-0.67 (2.73)	0.81	-1.37 (1.83)	0.46	-1.58 (2.30)	0.50	-2.47 (1.67)	0.15			0.00	
Race (Caucasian)	-1.07 (2.63)	0.69	-2.95 (1.77)	0.10	1.81 (2.53)	0.48	-2.70 (1.86)	0.16			-3.15 (4.70)	0.52
Education (> high school)	3.34 (2.66)	0.22	1.18 (1.79)	0.51	3.78 (2.61)	0.16	2.39 (1.90)	0.22			0.63 (6.13)	0.92
Regular Exercise (Yes)*	-5.28 (2.97)	0.08	-2.69 (1.99)	0.18	-2.32 (2.98)	0.44	-1.80 (2.11)	0.40			-5.07 (5.20)	0.35

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 35. Effects of post intervention trait anger reaction on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.77 (0.13)	<.0001	0.90 (0.11)	<.0001	0.95 (0.16)	<.0001	0.82 (0.13)	<.0001			1.14 (0.27)	0.001
Pre MBSR T-Ang/R*	-0.01 (0.53)	0.98	-0.10 (0.36)	0.78	-0.09 (0.55)	0.87	-0.40 (0.40)	0.32			1.08 (1.19)	0.38
Post MBSR T-Ang/R	0.08 (0.63)	0.90	-0.04 (0.44)	0.92	-0.71 (0.68)	0.31	-0.62 (0.48)	0.21			-0.86 (1.47)	0.57
Age	-0.08 (0.09)	0.38	-0.14 (0.06)	0.02	-0.26 (0.11)	0.03	-0.23 (0.07)	0.003			-0.08 (0.12)	0.50
BMI*	0.12 (0.20)	0.57	-0.08 (0.14)	0.58	-0.01 (0.20)	0.95	-0.07 (0.14)	0.63			0.53 (0.62)	0.41
Sex (female)	-4.48 (2.07)	0.04	-1.10 (1.42)	0.44								
FHx premature CVD (Yes)	-2.94 (2.09)	0.17	-3.11 (1.44)	0.04	-4.80 (2.40)	0.06	-4.23 (1.71)	0.02			-2.36 (3.45)	0.51
Drink $\geq$ 10 drinks/wk (Yes)*	2.76 (2.37)	0.25	2.93 (1.63)	0.08	2.50 (2.84)	0.39	4.99 (2.05)	0.02			0.75 (3.35)	0.83
Relaxation regularly (Yes)*	1.78 (2.84)	0.53	0.11 (1.97)	0.96	1.83 (2.41)	0.46	-0.61 (1.74)	0.73			0.00	
Race (Caucasian)	-2.09 (2.73)	0.45	-3.09 (1.89)	0.11	1.19 (2.66)	0.66	-2.45 (1.92)	0.21			-5.20 (5.17)	0.33
Education (> high school)	2.46 (2.76)	0.38	1.34 (1.91)	0.49	3.77 (2.74)	0.18	3.06 (1.98)	0.13			-0.21 (6.98)	0.98
Regular Exercise (Yes)*	-5.17 (3.10)	0.10	-3.42 (2.13)	0.12	-1.75 (3.10)	0.58	-1.98 (2.24)	0.38			-5.52 (5.88)	0.37

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 36. Effects of post intervention anger expressed inward on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.87 (0.13)	<.0001	1.01 (0.12)	<.0001	0.92	<.0001	0.92 (0.15)	<.0001			1.25 (0.27)	0.001
Pre MBSR AXI*	0.12 (0.22)	0.58	-0.02 (0.15)	0.90	0.11	0.67	-0.01 (0.19)	0.94			0.21 (0.35)	0.57
Post MBSR AXI	0.30 (0.24)	0.23	0.21 (0.17)	0.21	0.37	0.21	0.36 (0.22)	0.11			-0.17 (0.35)	0.64
Age	-0.05 (0.08)	0.52	-0.10 (0.05)	0.07	-0.15	0.14	-0.12 (0.07)	0.10			-0.03 (0.11)	0.77
BMI*	0.04 (0.17)	0.80	-0.13 (0.12)	0.26	-0.09	0.60	-0.14 (0.12)	0.23			0.26 (0.42)	0.55
Sex (female)	-2.36 (1.90)	0.22	-0.07 (1.30)	0.96								
FHx premature CVD (Yes)	-3.65 (1.95)	0.07	-3.50 (1.33)	0.01	-3.89	0.08	-3.30 (1.64)	0.054			-4.04 (3.07)	0.21
Drink $\geq$ 10 drinks/wk (Yes)*	2.81 (2.20)	0.21	2.93 (1.51)	0.06	3.84	0.13	5.02 (1.91)	0.01			-1.02 (3.69)	0.79
Relaxation regularly (Yes)*	0.40 (2.74)	0.88	-0.99 (1.87)	0.60	0.37	0.88	-1.22 (1.78)	0.50			0.00	
Race (Caucasian)	-0.40 (2.56)	0.88	-2.60 (1.74)	0.14	1.17	0.63	-3.07 (1.87)	0.11			-2.03 (5.42)	0.71
Education (> high school)	2.75 (2.53)	0.28	0.89 (1.71)	0.60	2.23	0.35	0.82 (1.79)	0.65			1.94 (4.97)	0.70
Regular Exercise (Yes)*	-5.00 (2.84)	0.08	-2.42 (1.93)	0.22	-1.06	0.72	-1.13 (2.16)	0.60			-7.62 (5.30)	0.18

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 37. Effects of post intervention anger expressed inward on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.80 (0.13)	<.0001	0.90 (0.11)	<.0001	0.92 (0.16)	<.0001	0.85 (0.14)	<.0001			1.09 (0.25)	0.001
Pre MBSR AXI*	0.19 (0.23)	0.43	0.03 (0.16)	0.85	0.12 (0.27)	0.66	-0.08 (0.21)	0.73			0.35 (0.38)	0.37
Post MBSR AXI	0.12 (0.26)	0.63	0.11 (0.18)	0.54	0.29 (0.31)	0.36	0.33 (0.25)	0.19			-0.36 (0.38)	0.36
Age	-0.08 (0.09)	0.35	-0.13 (0.06)	0.03	-0.18 (0.10)	0.08	-0.16 (0.08)	0.052			-0.05 (0.12)	0.70
BMI*	0.06 (0.18)	0.76	-0.10 (0.13)	0.44	-0.04 (0.17)	0.82	-0.11 (0.13)	0.43			0.34 (0.45)	0.47
Sex (female)	-4.29 (2.01)	0.04	-1.05 (1.40)	0.45								
FHx premature CVD (Yes)	-3.27 (2.07)	0.12	-3.21 (1.44)	0.03	-4.64 (2.35)	0.06	-3.61 (1.83)	0.06			-2.65 (3.22)	0.43
Drink $\geq$ 10 drinks/wk (Yes)*	2.39 (2.34)	0.31	2.81 (1.63)	0.09	1.89 (2.72)	0.49	3.72 (2.16)	0.10			-0.80 (3.95)	0.84
Relaxation regularly (Yes)*	2.77 (2.91)	0.35	0.60 (2.03)	0.77	3.49 (2.51)	0.18	0.51 (2.00)	0.80			0.00	
Race (Caucasian)	-1.71 (2.70)	0.53	-2.92 (1.89)	0.13	0.36 (2.62)	0.89	-2.81 (2.07)	0.19			-4.33 (5.69)	0.46
Education (> high school)	1.79 (2.67)	0.51	0.98 (1.86)	0.60	1.93 (2.53)	0.45	1.16 (2.00)	0.57			1.21 (5.33)	0.83
Regular Exercise (Yes)*	-5.07 (3.02)	0.10	-3.22 (2.10)	0.13	-1.00 (3.08)	0.75	-1.46 (2.45)	0.56			-9.15 (5.69)	0.13

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 38. Effects of post intervention anger expressed outward on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.85 (0.14)	<.0001	1.02 (0.12)	<.0001	0.95 (0.18)	<.0001	0.93 (0.16)	<.0001			1.28 (0.29)	0.001
Pre MBSR AXO*	-0.11 (0.33)	0.74	-0.03 (0.21)	0.88	-0.24 (0.34)	0.49	-0.14 (0.27)	0.59			0.19 (0.47)	0.70
Post MBSR AXO	0.23 (0.40)	0.57	0.00 (0.27)	0.99	0.41 (0.44)	0.36	-0.18 (0.34)	0.59			0.09 (0.57)	0.88
Age	-0.06 (0.09)	0.50	-0.11 (0.06)	0.06	-0.17 (0.10)	0.11	-0.16 (0.07)	0.04			-0.06 (0.12)	0.64
BMI*	0.06 (0.19)	0.74	-0.14 (0.12)	0.27	-0.09 (0.18)	0.63	-0.17 (0.13)	0.21			0.15 (0.43)	0.73
Sex (female)	-2.48 (1.97)	0.21	-0.25 (1.34)	0.85								
FHx premature CVD (Yes)	-3.07 (1.98)	0.13	-3.41 (1.34)	0.01	-2.97 (2.19)	0.19	-2.90 (1.66)	0.09			-4.03 (3.37)	0.25
Drink $\geq$ 10 drinks/wk (Yes)*	3.51 (2.27)	0.13	3.10 (1.54)	0.0495	4.89 (2.78)	0.09	5.41 (2.14)	0.02			0.13 (3.02)	0.97
Relaxation regularly (Yes)*	-0.79 (2.74)	0.78	-1.40 (1.85)	0.45	-1.03 (2.39)	0.67	-2.43 (1.86)	0.20			0.00	
Race (Caucasian)	-1.35 (2.64)	0.61	-3.01 (1.79)	0.10	1.14 (2.57)	0.66	-2.97 (2.01)	0.15			-1.71 (4.90)	0.73
Education (> high school)	3.09 (2.59)	0.24	0.96 (1.73)	0.58	2.51 (2.47)	0.32	1.25 (1.87)	0.51			3.36 (5.49)	0.55
Regular Exercise (Yes)*	-5.69 (3.02)	0.07	-2.57 (2.04)	0.21	-2.87 (3.25)	0.39	-0.57 (2.50)	0.82			-7.37 (5.30)	0.19

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 39. Effects of post intervention anger expressed outward on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.77 (0.14)	<.0001	0.90 (0.11)	<.0001	0.91 (0.16)	<.0001	0.81 (0.14)	<.0001				
Pre MBSR AXO*	-0.11 (0.35)	0.75	-0.03 (0.23)	0.90	-0.26 (0.37)	0.48	-0.20 (0.28)	0.49				
Post MBSR AXO	0.06 (0.42)	0.89	-0.05 (0.28)	0.86	0.36 (0.47)	0.45	-0.24 (0.36)	0.51				
Age	-0.08 (0.09)	0.35	-0.14 (0.06)	0.02	-0.19 (0.11)	0.08	-0.20 (0.08)	0.01				
BMI*	0.08 (0.19)	0.68	-0.10 (0.13)	0.45	-0.02 (0.18)	0.91	-0.12 (0.14)	0.39				
Sex (female)	-4.48 (2.06)	0.03	-1.22 (1.43)	0.40								
FHx premature CVD (Yes)	-2.72 (2.07)	0.20	-3.10 (1.43)	0.03	-3.69 (2.34)	0.13	-3.44 (1.77)	0.06				
Drink $\geq$ 10 drinks/wk (Yes)*	3.05 (2.39)	0.21	2.96 (1.64)	0.08	2.93 (3.00)	0.34	4.27 (2.29)	0.07				
Relaxation regularly (Yes)*	1.46 (2.87)	0.61	0.13 (1.99)	0.95	2.10 (2.56)	0.42	-0.62 (1.96)	0.76				
Race (Caucasian)	-2.22 (2.76)	0.43	-3.15 (1.92)	0.11	0.33 (2.73)	0.90	-2.89 (2.11)	0.18				
Education (> high school)	2.04 (2.70)	0.45	1.07 (1.85)	0.57	2.10 (2.61)	0.43	1.60 (2.00)	0.43				
Regular Exercise (Yes)*	-5.28 (3.17)	0.10	-3.17 (2.18)	0.15	-2.41 (3.47)	0.49	-0.57 (2.67)	0.83				

* Baseline covariates. NOTE1: Grey fill indicates an overall null model. Family history (FHx), cardiovascular disease (CVD).

Table 40. Effects of post intervention perceived stress on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.85 (0.11)	<.0001	1.00 (0.10)	<.0001	0.81 (0.12)	<.0001	0.94 (0.12)	<.0001	0.40 (0.26)	0.13	1.17 (0.23)	0.0001
Pre MBSR PS*	0.11 (0.12)	0.36	0.07 (0.08)	0.40	0.32 (0.12)	0.01	0.14 (0.09)	0.15	-0.05 (0.22)	0.81	0.14 (0.17)	0.43
Post MBSR PS	-0.03 (0.16)	0.84	-0.06 (0.11)	0.60	-0.35 (0.15)	0.03	-0.25 (0.13)	0.07	0.83 (0.32)	0.02	0.18 (0.20)	0.38
Age	-0.09 (0.08)	0.29	-0.12 (0.05)	0.02	-0.22 (0.09)	0.02	-0.19 (0.07)	0.01	0.26 (0.13)	0.07	0.04 (0.10)	0.73
BMI*	0.05 (0.17)	0.76	-0.16 (0.12)	0.16	-0.08 (0.15)	0.62	-0.19 (0.12)	0.12	0.18 (0.46)	0.70	0.05 (0.32)	0.88
Sex (female)	-3.44 (1.70)	0.048	-1.12 (1.16)	0.34								
FHx premature CVD (Yes)	-3.16 (1.89)	0.10	-3.63 (1.30)	0.01	-2.50 (2.03)	0.23	-2.86 (1.66)	0.10	-6.01 (3.34)	0.09	-5.65 (2.61)	0.047
Drink $\geq$ 10 drinks/wk (Yes)*	3.29 (2.15)	0.13	2.86 (1.47)	0.06	3.37 (2.44)	0.18	5.06 (2.00)	0.02	3.76 (3.60)	0.31	0.87 (2.58)	0.74
Relaxation regularly (Yes)*	0.43 (2.55)	0.87	-0.47 (1.74)	0.79	-0.90 (2.15)	0.68	-1.91 (1.76)	0.29	0.00		0.00	
Race (Causasian)	-0.85 (2.35)	0.72	-2.73 (1.61)	0.10	1.10 (2.20)	0.62	-3.24 (1.81)	0.08	-5.77 (4.37)	0.21	-2.01 (3.65)	0.59
Education (> high school)	-0.31 (2.31)	0.89	-0.81 (1.57)	0.61	-0.07 (2.22)	0.97	-0.10 (1.80)	0.96	3.23 (5.83)	0.59	-0.55 (3.88)	0.89
Regular Exercise (Yes)*	-3.73 (2.80)	0.19	-1.99 (1.92)	0.30	-0.49 (2.80)	0.86	-1.38 (2.22)	0.54	-17.19 (7.16)	0.03	-4.98 (4.11)	0.24

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 41. Effects of post intervention perceived stress on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.86 (0.11)	<.0001	0.96 (0.10)	<.0001	0.89 (0.13)	<.0001	0.95 (0.12)	<.0001	0.30 (0.28)	0.30	1.08 (0.23)	0.0003
Pre MBSR PS*	0.16 (0.12)	0.19	0.10 (0.09)	0.26	0.32 (0.12)	0.02	0.12 (0.10)	0.23	-0.05 (0.27)	0.86	0.29 (0.20)	0.16
Post MBSR PS	-0.05 (0.17)	0.74	-0.10 (0.12)	0.38	-0.33 (0.17)	0.055	-0.27 (0.14)	0.063	0.91 (0.36)	0.02	0.15 (0.23)	0.52
Age	-0.11 (0.09)	0.22	-0.14 (0.06)	0.01	-0.27 (0.09)	0.01	-0.22 (0.07)	0.01	0.27 (0.15)	0.10	0.05 (0.11)	0.67
BMI*	0.07 (0.18)	0.71	-0.09 (0.12)	0.45	-0.08 (0.16)	0.61	-0.14 (0.13)	0.29	0.16 (0.51)	0.76	0.21 (0.36)	0.56
Sex (female)	-5.27 (1.81)	0.01	-1.97 (1.24)	0.12								
FHx premature CVD (Yes)	-3.17 (2.02)	0.12	-3.50 (1.38)	0.01	-3.63 (2.19)	0.11	-3.42 (1.76)	0.06	-5.02 (3.72)	0.20	-5.06 (2.95)	0.11
Drink $\geq$ 10 drinks/wk (Yes)*	2.33 (2.29)	0.31	2.31 (1.56)	0.15	1.08 (2.61)	0.68	3.62 (2.11)	0.10	3.35 (4.02)	0.42	0.12 (2.90)	0.97
Relaxation regularly (Yes)*	3.37 (2.71)	0.22	1.08 (1.85)	0.56	2.70 (2.30)	0.25	-0.03 (1.86)	0.99	0.00		0.00	
Race (Causasian)	-1.42 (2.50)	0.57	-2.79 (1.72)	0.11	0.88 (2.38)	0.71	-3.30 (1.92)	0.10	-6.27 (4.94)	0.22	-2.68 (4.03)	0.52
Education (> high school)	-0.35 (2.46)	0.89	-0.33 (1.67)	0.84	-0.22 (2.39)	0.93	0.16 (1.91)	0.93	4.01 (6.19)	0.53	2.32 (4.43)	0.61
Regular Exercise (Yes)*	-3.58 (2.98)	0.23	-2.69 (2.03)	0.19	-0.69 (2.97)	0.82	-1.26 (2.35)	0.60	-19.30 (8.13)	0.03	-7.24 (4.64)	0.14

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 42. Effects of post intervention trait anger on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.85 (0.12)	<.0001	0.83 (0.09)	<.0001	0.95 (0.17)	<.0001	0.81 (0.13)	<.0001	0.84 (0.21)	0.002	0.89 (0.18)	0.0003
Pre MBSR T-Ang*	-0.02 (0.30)	0.94	-0.15 (0.19)	0.44	0.20 (0.42)	0.64	-0.24 (0.28)	0.39	-0.42 (0.72)	0.57	-0.35 (0.49)	0.48
Post MBSR T-Ang	0.14 (0.44)	0.76	0.35 (0.28)	0.22	-0.37 (0.61)	0.56	0.25 (0.41)	0.55	0.92 (1.17)	0.44	1.00 (0.81)	0.24
Age	0.02 (0.09)	0.83	-0.03 (0.06)	0.61	-0.18 (0.13)	0.17	-0.10 (0.08)	0.23	0.29 (0.15)	0.07	0.08 (0.11)	0.46
BMI*	0.06 (0.20)	0.77	-0.13 (0.12)	0.29	-0.24 (0.22)	0.28	-0.19 (0.14)	0.18	1.07 (0.66)	0.13	0.23 (0.47)	0.64
Sex (female)	-1.56 (2.13)	0.47	-0.06 (1.39)	0.97								
FHx premature CVD (Yes)	-1.93 (2.18)	0.38	-2.47 (1.40)	0.08	-3.91 (2.94)	0.20	-2.21 (1.96)	0.27	1.35 (4.01)	0.74	-1.05 (2.96)	0.73
Drink $\geq$ 10 drinks/wk (Yes)*	4.58 (2.52)	0.08	3.00 (1.65)	0.08	3.51 (3.54)	0.33	5.99 (2.43)	0.02	-0.57 (3.99)	0.89	-1.39 (2.86)	0.64
Relaxation regularly (Yes)*	-3.97 (3.02)	0.19	-3.83 (1.94)	0.054	-3.98 (3.08)	0.21	-4.55 (2.11)	0.04	0.00		0.00	
Race (Causasian)	1.83 (2.91)	0.53	-2.81 (1.85)	0.14	3.78 (3.44)	0.28	-2.45 (2.31)	0.30	-2.51 (5.42)	0.65	-3.56 (4.15)	0.41
Education (> high school)	3.30 (2.85)	0.25	0.80 (1.81)	0.66	1.67 (3.37)	0.62	-0.17 (2.23)	0.94	13.94 (10.18)	0.20	8.46 (7.43)	0.28
Regular Exercise (Yes)*	-6.10 (3.23)	0.07	-0.65 (2.07)	0.76	-1.90 (3.86)	0.63	-1.89 (2.49)	0.45	-13.52 (7.82)	0.11	0.53 (5.21)	0.92

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 43. Effects of post intervention trait anger temperament on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.85 (0.12)	<.0001	0.82 (0.10)	<.0001	0.96 (0.17)	<.0001	0.77 (0.13)	<.0001	0.85 (0.21)	0.002	0.82 (0.18)	0.001
Pre MBSR T-Ang/T*	-0.26 (0.62)	0.67	-0.31 (0.40)	0.44	-0.90 (0.86)	0.31	-0.71 (0.60)	0.24	-1.10 (1.30)	0.41	-0.61 (0.92)	0.52
Post MBSR T-Ang/T	0.81 (0.91)	0.37	0.45 (0.60)	0.46	1.95 (1.30)	0.15	1.02 (0.92)	0.28	1.94 (2.14)	0.38	1.40 (1.56)	0.39
Age	0.01 (0.09)	0.87	-0.05 (0.06)	0.43	-0.21 (0.12)	0.11	-0.12 (0.08)	0.15	0.30 (0.15)	0.06	0.10 (0.12)	0.42
BMI*	0.06 (0.20)	0.75	-0.13 (0.13)	0.32	-0.29 (0.21)	0.18	-0.20 (0.13)	0.15	1.34 (0.56)	0.03	0.50 (0.41)	0.24
Sex (female)	-1.19 (2.16)	0.58	0.10 (1.42)	0.95								
FHx premature CVD (Yes)	-1.90 (2.15)	0.38	-2.53 (1.41)	0.08	-2.29 (2.79)	0.42	-2.08 (1.89)	0.28	2.25 (3.88)	0.57	0.29 (2.94)	0.92
Drink $\geq$ 10 drinks/wk (Yes)*	4.46 (2.54)	0.09	3.12 (1.69)	0.07	4.38 (3.36)	0.20	5.76 (2.34)	0.02	-1.13 (4.22)	0.79	-2.12 (3.21)	0.52
Relaxation regularly (Yes)*	-4.18 (3.04)	0.18	-4.27 (1.98)	0.04	-4.93 (3.07)	0.12	-4.80 (2.12)	0.03	0.00		0.00	
Race (Causasian)	1.57 (2.90)	0.59	-2.73 (1.88)	0.15	2.37 (3.25)	0.47	-2.34 (2.23)	0.30	-3.95 (5.26)	0.47	-5.26 (4.22)	0.24
Education (> high school)	3.23 (2.85)	0.26	0.54 (1.85)	0.77	-1.71 (3.64)	0.64	-1.25 (2.46)	0.61	12.73 (9.19)	0.19	4.94 (6.94)	0.49
Regular Exercise (Yes)*	-5.97 (3.20)	0.07	-0.73 (2.09)	0.73	-3.09 (3.73)	0.41	-1.96 (2.46)	0.43	-12.02 (7.45)	0.13	2.38 (5.05)	0.65

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 44. Effects of post intervention trait anger temperament as a multilevel categorical predictor variable divided into quartiles on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.83 (0.12)	<.0001	0.83 (0.10)	<.0001	0.89 (0.18)	<.0001	0.80 (0.14)	<.0001	0.74 (0.23)	0.01		
Pre MBSR T-Ang/T*	-0.54 (0.63)	0.40	-0.24 (0.42)	0.57	-0.62 (0.86)	0.48	-0.31 (0.60)	0.61	-0.53 (1.39)	0.71		
Post MBSR T-Ang/T												
Q1 vs Q4	-4.69 (4.10)	0.26	-1.49 (2.76)	0.59	-5.29 (5.33)	0.33	-1.11 (3.75)	0.77	-6.29 (11.34)	0.59		
Q2 vs Q4	-7.50 (4.37)	0.09	-1.46 (2.94)	0.62	-8.61 (5.95)	0.16	-1.61 (4.13)	0.70	-4.26 (10.37)	0.69		
Q3 vs Q4	-4.00 (3.54)	0.26	-1.51 (2.38)	0.53	-4.73 (4.24)	0.28	-0.78 (2.96)	0.80	-11.62 (10.24)	0.28		
Age	0.01 (0.09)	0.92	-0.05 (0.06)	0.42	-0.17 (0.13)	0.20	-0.10 (0.08)	0.24	0.32 (0.16)	0.07		
BMI*	0.05 (0.20)	0.79	-0.13 (0.13)	0.30	-0.23 (0.22)	0.31	-0.18 (0.15)	0.23	1.65 (0.66)	0.03		
Sex (female)	-0.88 (2.16)	0.69	0.11 (1.46)	0.94								
FHx premature CVD (Yes)	-2.16 (2.17)	0.32	-2.68 (1.46)	0.07	-3.33 (2.90)	0.26	-2.59 (2.02)	0.21	0.75 (4.31)	0.87		
Drink $\geq$ 10 drinks/wk (Yes)*	5.36 (2.79)	0.06	3.04 (1.89)	0.12	5.34 (3.99)	0.19	5.96 (2.84)	0.046	-4.99 (5.98)	0.42		
Relaxation regularly (Yes)*	-4.93 (3.15)	0.12	-4.17 (2.10)	0.054	-4.79 (3.28)	0.16	-4.62 (2.35)	0.06	0.00			
Race (Caucasian)	0.38 (3.12)	0.90	-2.59 (2.08)	0.22	2.19 (3.58)	0.55	-2.29 (2.53)	0.37	-0.90 (6.53)	0.89		
Education (> high school)	3.10 (2.90)	0.29	0.61 (1.94)	0.75	0.09 (3.53)	0.98	-0.03 (2.43)	0.99	12.02 (8.75)	0.20		
Regular Exercise (Yes)*	-6.93 (3.25)	0.04	-0.82 (2.18)	0.71	-4.18 (4.06)	0.31	-2.01 (2.79)	0.48	-11.66 (7.90)	0.17		

* Baseline covariates. NOTE1: Grey fill indicate s an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD). Q1= bottom 25th percent quartile, Q2=second quartile, Q3=third quartile, Q4= top 25th percent quartile.

Table 45. Effects of post intervention trait anger reaction on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.85 (0.12)	<.0001	0.84 (0.09)	<.0001	0.95 (0.17)	<.0001	0.85 (0.13)	<.0001	0.80 (0.20)	0.002	0.86 (0.18)	0.0004
Pre MBSR T-Ang/R*	-0.03 (0.56)	0.96	-0.22 (0.35)	0.54	0.49 (0.64)	0.45	-0.39 (0.44)	0.38	-0.10 (1.38)	0.95	0.14 (0.97)	0.89
Post MBSR T-Ang/R	0.05 (0.67)	0.94	0.60 (0.42)	0.16	-1.10 (0.79)	0.17	0.20 (0.54)	0.72	0.59 (1.73)	0.74	0.63 (1.23)	0.62
Age	0.01 (0.09)	0.91	-0.03 (0.06)	0.65	-0.23 (0.12)	0.08	-0.10 (0.08)	0.23	0.28 (0.15)	0.08	0.07 (0.11)	0.52
BMI*	0.02 (0.22)	0.93	-0.16 (0.14)	0.24	-0.38 (0.24)	0.12	-0.25 (0.16)	0.13	1.22 (0.75)	0.13	0.44 (0.52)	0.41
Sex (female)	-1.67 (2.18)	0.45	-0.39 (1.40)	0.78								
FHx premature CVD (Yes)	-1.83 (2.21)	0.41	-2.49 (1.40)	0.08	-3.33 (2.79)	0.24	-2.13 (1.94)	0.28	1.42 (4.24)	0.74	-0.84 (3.04)	0.79
Drink $\geq$ 10 drinks/wk (Yes)*	4.75 (2.52)	0.07	3.17 (1.64)	0.06	3.98 (3.40)	0.25	6.32 (2.43)	0.02	-0.04 (4.11)	0.99	-0.70 (2.94)	0.81
Relaxation regularly (Yes)*	-4.10 (3.04)	0.18	-3.55 (1.93)	0.07	-4.92 (2.92)	0.10	-4.66 (2.08)	0.03	0.00		0.00	
Race (Caucasian)	1.98 (2.90)	0.50	-2.65 (1.83)	0.15	4.04 (3.22)	0.22	-2.31 (2.26)	0.32	-2.68 (6.14)	0.67	-4.09 (4.65)	0.40
Education (> high school)	3.00 (2.95)	0.31	0.68 (1.85)	0.72	0.52 (3.24)	0.87	-0.05 (2.24)	0.98	9.72 (8.56)	0.28	3.51 (6.13)	0.58
Regular Exercise (Yes)*	-6.25 (3.27)	0.06	-0.58 (2.07)	0.78	-2.36 (3.71)	0.53	-2.14 (2.49)	0.40	-12.49 (8.10)	0.15	2.29 (5.05)	0.66

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 46. Effects of post intervention anger expressed inward on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.83 (0.12)	<.0001	0.82 (0.10)	<.0001	0.85 (0.21)	0.0003	0.79 (0.14)	<.0001	0.84 (0.20)	0.001	0.84 (0.19)	0.001
Pre MBSR AXI*	-0.10 (0.24)	0.69	-0.12 (0.16)	0.48	0.21 (0.36)	0.56	-0.03 (0.23)	0.90	0.06 (0.46)	0.90	0.03 (0.36)	0.94
Post MBSR AXI	0.52 (0.27)	0.055	0.28 (0.18)	0.12	0.15 (0.39)	0.71	0.14 (0.26)	0.60	0.52 (0.46)	0.27	0.36 (0.34)	0.32
Age	0.04 (0.09)	0.64	-0.03 (0.06)	0.55	-0.13 (0.13)	0.32	-0.08 (0.08)	0.31	0.28 (0.14)	0.08	0.08 (0.12)	0.49
BMI*	0.09 (0.19)	0.66	-0.12 (0.12)	0.34	-0.19 (0.22)	0.41	-0.17 (0.14)	0.24	1.23 (0.55)	0.046	0.42 (0.41)	0.33
Sex (female)	-1.17 (2.07)	0.57	0.11 (1.38)	0.93								
FHx premature CVD (Yes)	-2.07 (2.12)	0.33	-2.57 (1.40)	0.07	-3.98 (2.85)	0.17	-2.82 (1.97)	0.16	1.66 (3.82)	0.67	-0.34 (2.92)	0.91
Drink $\geq$ 10 drinks/wk (Yes)*	4.38 (2.44)	0.08	2.95 (1.67)	0.08	3.39 (3.42)	0.33	5.23 (2.38)	0.04	-1.29 (4.75)	0.79	-1.69 (3.81)	0.66
Relaxation regularly (Yes)*	-3.47 (3.09)	0.27	-3.97 (2.06)	0.06	-2.47 (3.47)	0.48	-3.91 (2.32)	0.10	0.00		0.00	
Race (Caucasian)	2.97 (2.81)	0.30	-2.00 (1.85)	0.29	3.17 (3.27)	0.34	-1.88 (2.25)	0.41	1.18 (6.63)	0.86	-1.59 (5.20)	0.77
Education (> high school)	2.99 (2.78)	0.29	0.75 (1.81)	0.68	0.22 (3.19)	0.94	0.34 (2.09)	0.87	8.20 (6.46)	0.23	1.14 (4.94)	0.82
Regular Exercise (Yes)*	-5.68 (3.10)	0.07	-0.39 (2.04)	0.85	-0.92 (4.05)	0.82	-1.55 (2.55)	0.55	-11.17 (7.86)	0.18	2.73 (5.71)	0.61

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 47. Effects of post intervention anger expressed outward on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.82 (0.12)	<.0001	0.82 (0.10)	<.0001	0.92 (0.19)	<.0001	0.78 (0.15)	<.0001	0.81 (0.19)	0.001	0.83 (0.18)	0.001
Pre MBSR AXO*	-0.17 (0.35)	0.62	0.11 (0.23)	0.63	0.01 (0.44)	0.98	0.17 (0.31)	0.59	-0.53 (0.56)	0.36	-0.25 (0.43)	0.58
Post MBSR AXO	0.49 (0.44)	0.27	0.03 (0.28)	0.93	0.29 (0.59)	0.63	-0.04 (0.39)	0.91	0.78 (0.71)	0.29	0.57 (0.54)	0.31
Age	0.04 (0.09)	0.66	-0.04 (0.06)	0.46	-0.15 (0.13)	0.25	-0.08 (0.08)	0.28	0.35 (0.15)	0.04	0.13 (0.12)	0.30
BMI*	0.10 (0.20)	0.62	-0.14 (0.13)	0.28	-0.23 (0.23)	0.33	-0.20 (0.14)	0.17	1.46 (0.56)	0.02	0.57 (0.41)	0.19
Sex (female)	-1.15 (2.15)	0.60	-0.07 (1.43)	0.96								
FHx premature CVD (Yes)	-1.65 (2.15)	0.45	-2.65 (1.42)	0.07	-3.19 (2.79)	0.26	-2.82 (1.86)	0.14	3.66 (4.01)	0.38	1.21 (3.04)	0.70
Drink $\geq$ 10 drinks/wk (Yes)*	5.00 (2.49)	0.0503	2.80 (1.69)	0.10	3.91 (3.62)	0.29	4.83 (2.58)	0.07	-0.52 (3.84)	0.89	-1.31 (2.97)	0.67
Relaxation regularly (Yes)*	-4.04 (3.03)	0.19	-3.67 (2.02)	0.08	-3.61 (3.21)	0.27	-3.57 (2.34)	0.14	0.00		0.00	
Race (Caucasian)	1.47 (2.89)	0.61	-2.35 (1.90)	0.22	3.31 (3.38)	0.34	-1.48 (2.38)	0.54	-4.88(5.26)	0.37	-5.71 (4.26)	0.20
Education (> high school)	3.15 (2.82)	0.27	0.81 (1.84)	0.66	0.63 (3.18)	0.84	0.46 (2.10)	0.83	7.91 (6.98)	0.28	1.54 (5.36)	0.78
Regular Exercise (Yes)*	-7.09 (3.29)	0.04	-0.87 (2.16)	0.69	-3.04 (4.13)	0.47	-1.70 (2.75)	0.54	-11.43 (7.41)	0.15	2.52 (4.99)	0.62

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 48. Effects of post intervention perceived stress on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR nighttime BP*	0.77 (0.10)	<.0001	0.82 (0.09)	<.0001	0.68 (0.12)	<.0001	0.82 (0.12)	<.0001	0.77 (0.17)	0.001	0.79 (0.15)	<.0001
Pre MBSR PS*	0.06 (0.13)	0.68	0.02 (0.09)	0.83	0.41 (0.16)	0.01	0.12 (0.11)	0.28	-0.34 (0.21)	0.12	-0.17 (0.16)	0.31
Post MBSR PS	-0.04 (0.18)	0.81	0.07 (0.11)	0.56	-0.52 (0.21)	0.02	-0.14 (0.15)	0.35	0.61 (0.27)	0.04	0.29 (0.20)	0.16
Age	0.00 (0.09)	0.99	-0.05 (0.06)	0.38	-0.12 (0.11)	0.30	-0.11 (0.08)	0.19	0.26 (0.13)	0.07	0.09 (0.10)	0.38
BMI*	0.06 (0.20)	0.78	-0.19 (0.13)	0.14	-0.10 (0.21)	0.65	-0.21 (0.15)	0.16	0.68 (0.43)	0.13	0.14 (0.32)	0.68
Sex (female)	-2.00 (1.98)	0.32	-0.47 (1.27)	0.71								
FHx premature CVD (Yes)	-1.97 (2.18)	0.37	-2.93 (1.41)	0.04	-3.24 (2.74)	0.25	-3.20 (1.98)	0.12	-0.74 (3.26)	0.82	-1.01 (2.48)	0.69
Drink $\geq$ 10 drinks/wk (Yes)*	5.00 (2.49)	0.0497	3.51 (1.64)	0.04	2.73 (3.37)	0.42	5.09 (2.50)	0.0503	6.53 (3.41)	0.07	2.85 (2.66)	0.30
Relaxation regularly (Yes)*	-4.30 (2.95)	0.15	-3.22 (1.91)	0.10	-4.71 (2.93)	0.12	-3.89 (2.15)	0.08	0.00		0.00	
Race (Caucasian)	0.94 (2.71)	0.73	-2.74 (1.75)	0.12	1.46 (2.99)	0.63	-2.92 (2.18)	0.19	-2.04 (4.14)	0.63	-4.85 (3.39)	0.17
Education (> high school)	-0.55 (2.65)	0.84	-1.34 (1.72)	0.44	0.43 (2.98)	0.89	0.04 (2.14)	0.99	-6.88 (5.71)	0.25	-7.38 (3.91)	0.08
Regular Exercise (Yes)*	-4.79 (3.22)	0.14	0.37 (2.09)	0.86	-1.11 (3.79)	0.77	-1.43 (2.69)	0.60	-4.28 (6.41)	0.51	6.63 (4.00)	0.12

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 49. Effects of post intervention anger control inward on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.87 (0.13)	<.0001	1.03 (0.12)	<.0001	0.92 (0.17)	<.0001	0.92 (0.15)	<.0001	0.62 (0.30)	0.06	1.21 (0.27)	0.001
Pre MBSR ACI*	-0.03 (0.20)	0.87	-0.02 (0.13)	0.89	0.11 (0.26)	0.67	-0.01 (0.19)	0.94	-0.54 (0.43)	0.24	-0.33 (0.28)	0.27
Post MBSR ACI	0.23 (0.25)	0.36	0.15 (0.17)	0.37	0.37 (0.28)	0.21	0.36 (0.22)	0.11	0.36 (0.73)	0.63	0.14 (0.48)	0.77
Age	-0.08 (0.08)	0.36	-0.11 (0.05)	0.045	-0.15 (0.10)	0.14	-0.12 (0.07)	0.10	0.12 (0.16)	0.45	-0.03 (0.11)	0.77
BMI*	0.04 (0.18)	0.81	-0.13 (0.12)	0.26	-0.09 (0.16)	0.60	-0.14 (0.12)	0.23	0.22 (0.66)	0.75	0.02 (0.43)	0.97
Sex (female)	-2.88 (1.95)	0.15	-0.37 (1.31)	0.78								
FHx premature CVD (Yes)	-2.91 (1.97)	0.15	-3.21 (1.33)	0.02	-3.89 (2.15)	0.08	-3.30 (1.64)	0.054	-2.82 (4.61)	0.55	-4.35 (3.10)	0.19
Drink $\geq$ 10 drinks/wk (Yes)*	3.43 (2.21)	0.13	3.18 (1.49)	0.04	3.84 (2.49)	0.13	5.02 (1.91)	0.01	0.62 (4.74)	0.90	0.18 (3.24)	0.96
Relaxation regularly (Yes)*	-1.26 (2.77)	0.65	-1.68 (1.87)	0.37	0.37 (2.35)	0.88	-1.22 (1.78)	0.50	0.00		0.00	
Race (Caucasian)	-0.77 (2.59)	0.77	-2.78 (1.75)	0.12	1.17 (2.42)	0.63	-3.07 (1.87)	0.11	-7.12 (5.98)	0.26	-2.17 (4.71)	0.65
Education (> high school)	3.76 (2.62)	0.16	1.38 (1.75)	0.43	2.23 (2.37)	0.35	0.82 (1.79)	0.65	7.42 (7.58)	0.35	2.98 (4.84)	0.55
Regular Exercise (Yes)*	-5.32 (2.88)	0.07	-2.64 (1.94)	0.18	-1.06 (2.91)	0.72	-1.13 (2.16)	0.60	-20.53 (10.39)	0.07	-8.66 (5.45)	0.14

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 50. Effects of post intervention anger control inward on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.78 (0.13)	<.0001	0.91 (0.11)	<.0001	0.92 (0.16)	<.0001	0.85 (0.14)	<.0001	0.39 (0.33)	0.27	1.03 (0.25)	0.001
Pre MBSR ACI*	-0.07 (0.22)	0.75	-0.07 (0.14)	0.64	0.12 (0.27)	0.66	-0.08 (0.21)	0.73	-0.62 (0.45)	0.19	-0.51 (0.30)	0.11
Post MBSR ACI	0.16 (0.26)	0.55	0.16 (0.18)	0.37	0.29 (0.31)	0.36	0.33 (0.25)	0.19	0.23 (0.81)	0.78	0.21 (0.53)	0.70
Age	-0.09 (0.09)	0.31	-0.14 (0.06)	0.02	-0.18 (0.10)	0.08	-0.16 (0.08)	0.052	0.06 (0.16)	0.71	-0.05 (0.11)	0.66
BMI*	0.06 (0.18)	0.74	-0.10 (0.13)	0.43	-0.04 (0.17)	0.82	-0.11 (0.13)	0.43	-0.07 (0.65)	0.91	-0.07 (0.46)	0.88
Sex (female)	-4.56 (2.05)	0.03	-1.24 (1.40)	0.38								
FHx premature CVD (Yes)	-2.67 (2.08)	0.20	-2.94 (1.42)	0.04	-4.64 (2.35)	0.06	-3.61 (1.83)	0.06	-2.95 (4.77)	0.55	-3.28 (3.18)	0.32
Drink $\geq$ 10 drinks/wk (Yes)*	2.96 (2.33)	0.21	3.04 (1.60)	0.06	1.89 (2.72)	0.49	3.72 (2.16)	0.10	1.22 (4.85)	0.81	1.13 (3.47)	0.75
Relaxation regularly (Yes)*	1.58 (2.92)	0.59	0.12 (2.02)	0.95	3.49 (2.51)	0.18	0.51 (2.00)	0.80	0.00		0.00	
Race (Caucasian)	-1.85 (2.72)	0.50	-2.91 (1.89)	0.13	0.36 (2.62)	0.89	-2.81 (2.07)	0.19	-8.15 (5.99)	0.20	-3.98 (4.91)	0.43
Education (> high school)	2.51 (2.75)	0.37	1.48 (1.88)	0.44	1.93 (2.53)	0.45	1.16 (2.00)	0.57	8.21 (7.80)	0.31	3.03 (5.09)	0.56
Regular Exercise (Yes)*	-5.32 (3.05)	0.09	-3.39 (2.09)	0.11	-1.00 (3.08)	0.75	-1.46 (2.45)	0.56	-26.64 (11.81)	0.04	-10.69 (5.72)	0.09

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 51. Effects of post intervention anger control inward on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR night BP*	0.83 (0.12)	<.0001	0.84 (0.09)	<.0001	0.85 (0.21)	0.0003	0.79 (0.14)	<.0001	0.83 (0.20)	0.002	0.86 (0.20)	0.001
Pre MBSR ACI*	0.28 (0.22)	0.20	0.15 (0.14)	0.32	0.21 (0.36)	0.56	-0.03 (0.23)	0.90	0.25 (0.41)	0.55	0.15 (0.32)	0.64
Post MBSR ACI	-0.04 (0.27)	0.88	-0.11 (0.18)	0.53	0.15 (0.39)	0.71	0.14 (0.26)	0.60	-0.32 (0.67)	0.65	-0.33 (0.51)	0.53
Age	0.00 (0.09)	0.98	-0.05 (0.06)	0.36	-0.13 (0.13)	0.32	-0.08 (0.08)	0.31	0.28 (0.15)	0.08	0.09 (0.12)	0.48
BMI*	0.09 (0.19)	0.64	-0.12 (0.12)	0.34	-0.19 (0.22)	0.41	-0.17 (0.14)	0.24	1.51 (0.64)	0.04	0.63 (0.46)	0.20
Sex (female)	-2.01 (2.11)	0.35	-0.15 (1.41)	0.92								
FHx premature CVD (Yes)	-1.53 (2.14)	0.48	-2.59 (1.42)	0.07	-3.98 (2.85)	0.17	-2.82 (1.97)	0.16	1.98 (4.25)	0.65	-0.58 (3.27)	0.86
Drink $\geq$ 10 drinks/wk (Yes)*	4.70 (2.42)	0.06	3.00 (1.63)	0.07	3.39 (2.42)	0.33	5.23 (2.38)	0.04	-1.24 (4.46)	0.79	-2.08 (3.34)	0.54
Relaxation regularly (Yes)*	-5.49 (3.05)	0.08	-4.50 (2.03)	0.03	-2.47 (3.47)	0.48	-3.91 (2.32)	0.10	0.00		0.00	
Race (Caucasian)	1.80 (2.84)	0.53	-2.70 (1.87)	0.15	3.17 (3.27)	0.34	-1.88 (2.25)	0.41	-3.92 (5.59)	0.50	-5.20 (4.41)	0.26
Education (> high school)	3.33 (2.85)	0.25	0.54 (1.87)	0.77	0.22 (3.19)	0.94	0.34 (2.09)	0.87	6.89 (6.80)	0.33	0.61 (5.11)	0.91
Regular Exercise (Yes)*	-5.87 (3.14)	0.07	-0.50 (2.07)	0.81	-0.92 (4.05)	0.82	-1.55 (2.55)	0.55	-10.88 (8.51)	0.23	2.18 (5.70)	0.71

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 52. Effects of post intervention anger control outward on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.87 (0.13)	<.0001	1.01 (0.12)	<.0001	0.97 (0.17)	<.0001	0.90 (0.15)	<.0001			1.32 (0.27)	0.0003
Pre MBSR ACO*	-0.04 (0.26)	0.89	0.01 (0.18)	0.97	0.39 (0.28)	0.17	0.31 (0.21)	0.16			-0.24 (0.44)	0.60
Post MBSR ACO	0.11 (0.27)	0.67	0.03 (0.18)	0.87	-0.38 (0.30)	0.22	-0.17 (0.23)	0.48			-0.15 (0.46)	0.74
Age	-0.08 (0.08)	0.36	-0.11 (0.05)	0.047	-0.20 (0.10)	0.053	-0.16 (0.07)	0.03			-0.06 (0.11)	0.62
BMI*	0.03 (0.18)	0.87	-0.14 (0.12)	0.26	-0.12 (0.16)	0.47	-0.15 (0.12)	0.21			0.13 (0.48)	0.79
Sex (female)	-2.83 (1.97)	0.16	-0.31 (1.34)	0.82								
FHx premature CVD (Yes)	-3.21 (1.98)	0.11	-3.38 (1.34)	0.02	-2.15 (2.27)	0.35	-2.27 (1.71)	0.20			-4.72 (2.99)	0.14
Drink $\geq$ 10 drinks/wk (Yes)*	3.30 (2.23)	0.14	3.08 (1.51)	0.046	5.01 (2.64)	0.07	5.92 (2.01)	0.01			-0.05 (3.32)	0.99
Relaxation regularly (Yes)*	-0.86 (2.72)	0.75	-1.41 (1.84)	0.45	-1.80 (2.34)	0.45	-2.79 (1.78)	0.13			0.00	
Race (Caucasian)	-1.04 (2.60)	0.69	-3.00 (1.76)	0.09	1.11 (2.55)	0.67	-3.51 (1.94)	0.08			-2.57 (4.39)	0.57
Education (> high school)	3.24 (2.62)	0.22	0.99 (1.76)	0.57	1.44 (2.63)	0.59	0.49 (2.00)	0.81			5.73 (5.75)	0.34
Regular Exercise (Yes)*	-5.41 (3.08)	0.09	-2.59 (2.08)	0.22	-0.29 (3.12)	0.93	-0.13 (2.35)	0.96			-9.49 (5.43)	0.11

* Baseline covariates. NOTE1: Grey fill indicate s an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 53. Effects of post intervention anger control outward on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.78 (0.13)	<.0001	0.89 (0.11)	<.0001	0.91 (0.16)	<.0001	0.78 (0.14)	<.0001			1.13 (0.25)	0.001
Pre MBSR ACO*	-0.04 (0.28)	0.89	-0.06 (0.19)	0.77	0.31 (0.30)	0.30	0.20 (0.23)	0.38			-0.59 (0.51)	0.27
Post MBSR ACO	0.08 (0.28)	0.79	0.11 (0.19)	0.56	-0.36 (0.32)	0.28	0.00 (0.25)	0.99			0.20 (0.50)	0.70
Age	-0.09 (0.09)	0.30	-0.14 (0.06)	0.02	-0.21 (0.10)	0.06	-0.21 (0.08)	0.01			-0.11 (0.13)	0.39
BMI*	0.06 (0.19)	0.77	-0.11 (0.13)	0.39	-0.06 (0.17)	0.73	-0.11 (0.13)	0.41			-0.09 (0.54)	0.88
Sex (female)	-4.58 (2.06)	0.03	-1.30 (1.41)	0.36								
FHx premature CVD (Yes)	-2.81 (2.07)	0.18	-3.09 (1.43)	0.04	-2.96 (2.44)	0.24	-3.20 (1.86)	0.10			-3.46 (3.23)	0.30
Drink $\geq$ 10 drinks/wk (Yes)*	2.89 (2.33)	0.22	2.97 (1.60)	0.07	2.87 (2.86)	0.33	4.46 (2.20)	0.052			2.21 (3.72)	0.56
Relaxation regularly (Yes)*	1.64 (2.84)	0.57	0.17 (1.97)	0.93	1.74 (2.51)	0.49	-0.68 (1.93)	0.73			0.00	
Race (Caucasian)	-2.05 (2.72)	0.46	-3.12 (1.89)	0.10	0.61 (2.71)	0.82	-3.46 (2.09)	0.11			-4.17 (4.75)	0.40
Education (> high school)	2.22 (2.74)	0.42	1.24 (1.87)	0.51	1.05 (2.81)	0.71	1.41 (2.18)	0.52			4.34 (6.25)	0.50
Regular Exercise (Yes)*	-5.41 (3.23)	0.10	-3.55 (2.22)	0.12	-0.31 (3.32)	0.93	-0.86 (2.58)	0.74			-10.81 (5.96)	0.09

* Baseline covariates. NOTE1: Grey fill indicate s an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 54. Effects of post intervention anger control outward on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR night BP*	0.85 (0.12)	<.0001	0.85 (0.10)	<.0001	0.97 (0.17)	<.0001	0.84 (0.13)	<.0001	0.91 (0.19)	0.001	0.97 (0.19)	0.0003
Pre MBSR ACO*	0.14 (0.29)	0.63	0.09 (0.19)	0.66	0.41 (0.36)	0.26	0.25 (0.24)	0.32	0.96 (0.60)	0.13	0.54 (0.46)	0.26
Post MBSR ACO	-0.04 (0.30)	0.90	-0.11 (0.20)	0.59	-0.35 (0.39)	0.37	-0.23 (0.27)	0.40	-0.99 (0.61)	0.13	-0.80 (0.49)	0.13
Age	0.01 (0.09)	0.92	-0.04 (0.06)	0.45	-0.20 (0.12)	0.12	-0.09 (0.08)	0.25	0.38 (0.14)	0.02	0.13 (0.11)	0.27
BMI*	0.08 (0.20)	0.70	-0.12 (0.13)	0.37	-0.24 (0.21)	0.28	-0.17 (0.14)	0.22	2.05 (0.67)	0.01	0.93 (0.48)	0.08
Sex (female)	-1.72 (2.16)	0.43	0.03 (1.43)	0.98								
FHx premature CVD (Yes)	-1.74 (2.18)	0.43	-2.59 (1.42)	0.07	-2.30 (2.88)	0.43	-2.02 (1.97)	0.31	3.16 (3.64)	0.40	-0.27 (2.75)	0.92
Drink $\geq$ 10 drinks/wk (Yes)*	4.71 (2.47)	0.06	3.03 (1.65)	0.07	4.92 (3.49)	0.17	6.20 (2.45)	0.02	-4.35 (4.33)	0.33	-3.70 (3.20)	0.27
Relaxation regularly (Yes)*	-4.52 (3.03)	0.14	-4.10 (1.98)	0.04	-5.22 (3.10)	0.10	-4.90 (2.17)	0.03	0.00		0.00	
Race (Causasian)	1.83 (2.88)	0.53	-2.55 (1.87)	0.18	2.51 (3.35)	0.46	-2.41 (2.30)	0.30	-4.12 (5.03)	0.43	-4.93 (4.00)	0.24
Education (> high school)	3.00 (3.88)	0.30	0.54 (1.87)	0.77	-0.35 (3.36)	0.92	-0.51 (2.30)	0.82	9.37 (7.17)	0.22	4.84 (5.74)	0.42
Regular Exercise (Yes)*	-5.61 (3.40)	0.11	-0.32 (2.22)	0.89	-0.61 (4.01)	0.88	-0.70 (2.64)	0.79	-8.05 (7.71)	0.32	2.41 (5.04)	0.64

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 55. Effects of post intervention anger expression index on post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR 24-hour BP*	0.87 (0.14)	<.0001	1.02 (0.12)	<.0001	0.95 (0.17)	<.0001	0.91 (0.16)	<.0001			1.27 (0.26)	0.0004
Pre MBSR AXX*	0.00 (0.08)	0.97	-0.01 (0.05)	0.85	-0.12 (0.09)	0.19	-0.09 (0.07)	0.23			0.13 (0.11)	0.27
Post MBSR AXX	0.00 (0.10)	0.97	0.00 (0.07)	0.98	0.16 (0.11)	0.17	0.06 (0.09)	0.48			0.01 (0.13)	0.91
Age	-0.07 (0.08)	0.39	-0.11 (0.05)	0.0504	-0.17 (0.10)	0.10	-0.15 (0.07)	0.049			-0.05 (0.11)	0.64
BMI*	0.04 (0.18)	0.84	-0.14 (0.12)	0.27	-0.11 (0.16)	0.52	-0.16 (0.12)	0.21			0.07 (0.41)	0.86
Sex (female)	-2.67 (1.98)	0.19	-0.27 (1.34)	0.84								
FHx premature CVD (Yes)	-3.25 (2.00)	0.11	-3.37 (1.35)	0.02	-2.25 (2.24)	0.32	-2.31 (1.71)	0.19			-4.80 (2.99)	0.14
Drink $\geq$ 10 drinks/wk (Yes)*	3.27 (2.25)	0.15	3.11 (1.52)	0.046	4.78 (2.60)	0.08	5.67 (2.02)	0.01			-0.74 (3.02)	0.81
Relaxation regularly (Yes)*	-0.70 (2.85)	0.81	-1.46 (1.92)	0.45	-1.78 (2.44)	0.47	-2.85 (1.90)	0.15			0.00	
Race (Causasian)	-1.06 (2.61)	0.69	-3.02 (1.76)	0.09	1.28 (2.48)	0.61	-3.12 (1.94)	0.12			-1.51 (4.35)	0.73
Education (> high school)	3.17 (2.60)	0.23	0.99 (1.74)	0.57	1.74 (2.54)	0.50	0.82 (1.95)	0.68			4.57 (5.36)	0.41
Regular Exercise (Yes)*	-5.30 (2.94)	0.08	-2.56 (1.98)	0.20	-1.86 (2.90)	0.53	-1.40 (2.21)	0.53			-9.96 (5.56)	0.10

* Baseline covariates. NOTE1: Grey fill indicate s an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 56. Effects of post intervention anger expression index on post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariate

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR daytime BP*	0.79 (0.13)	<.0001	0.90 (0.11)	<.0001	0.91 (0.16)	<.0001	0.79 (0.14)	<.0001			1.11 (0.24)	0.0006
Pre MBSR AXX*	0.03 (0.09)	0.77	0.01 (0.06)	0.82	-0.11 (0.09)	0.26	-0.08 (0.07)	0.31			0.20 (0.12)	0.11
Post MBSR AXX	-0.02 (0.10)	0.84	-0.03 (0.07)	0.68	0.16 (0.12)	0.18	0.02 (0.09)	0.80			-0.05 (0.14)	0.73
Age	-0.09 (0.09)	0.30	-0.14 (0.06)	0.02	-0.19 (0.10)	0.09	-0.20 (0.08)	0.02			-0.08 (0.11)	0.47
BMI*	0.05 (0.19)	0.78	-0.11 (0.13)	0.41	-0.05 (0.17)	0.78	-0.12 (0.13)	0.39			0.00 (0.43)	1.00
Sex (female)	-4.50 (2.07)	0.03	-1.26 (1.42)	0.38								
FHx premature CVD (Yes)	-2.91 (2.09)	0.17	-3.09 (1.44)	0.04	-3.02 (2.38)	0.22	-2.96 (1.85)	0.12			-3.70 (3.10)	0.26
Drink $\geq$ 10 drinks/wk (Yes)*	2.80 (2.35)	0.24	2.94 (1.62)	0.08	2.78 (2.79)	0.33	4.39 (2.19)	0.06			0.18 (3.22)	0.96
Relaxation regularly (Yes)*	1.92 (2.98)	0.52	0.22 (2.07)	0.92	1.66 (2.61)	0.53	-0.96 (2.05)	0.64			0.00	
Race (Causasian)	-2.00 (2.72)	0.47	-3.11 (1.89)	0.11	0.54 (2.62)	0.84	-3.02 (2.07)	0.16			-2.95 (4.49)	0.52
Education (> high school)	2.22 (2.71)	0.42	1.20 (1.86)	0.52	1.22 (2.67)	0.65	1.44 (2.12)	0.50			4.29 (5.68)	0.47
Regular Exercise (Yes)*	-5.37 (3.08)	0.09	-3.37 (2.12)	0.12	-1.64 (3.05)	0.59	-1.64 (2.41)	0.50			-11.87 (5.88)	0.07

* Baseline covariates. NOTE1: Grey fill indicate s an overall null model. NOTE2: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 57. Effects of post intervention anger expression index on post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	All Subjects				Females				Males			
	Systolic		Diastolic		Systolic		Diastolic		Systolic		Diastolic	
	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value	β (SE)	p-value
Pre MBSR night BP*	0.85 (0.12)	<.0001	0.85 (0.10)	<.0001	0.95 (0.18)	<.0001	0.83 (0.13)	<.0001	0.86 (0.20)	0.001	0.89 (0.19)	0.001
Pre MBSR AXX*	-0.10 (0.09)	0.24	-0.05 (0.06)	0.40	-0.11 (0.11)	0.33	-0.08 (0.08)	0.32	-0.12 (0.15)	0.44	-0.05 (0.11)	0.66
Post MBSR AXX	0.11 (0.11)	0.31	0.07 (0.07)	0.29	0.12 (0.14)	0.43	0.08 (0.10)	0.39	0.19 (0.18)	0.29	0.17 (0.13)	0.22
Age	0.02 (0.09)	0.87	-0.04 (0.06)	0.49	-0.17 (0.13)	0.18	-0.08 (0.08)	0.31	0.30 (0.14)	0.053	0.10 (0.11)	0.41
BMI*	0.10 (0.20)	0.61	-0.11 (0.13)	0.40	-0.22 (0.21)	0.31	-0.16 (0.14)	0.24	1.51 (0.58)	0.02	0.59 (0.41)	0.18
Sex (female)	-1.42 (2.14)	0.51	0.15 (1.42)	0.92								
FHx premature CVD (Yes)	-1.62 (2.16)	0.46	-2.55 (1.41)	0.08	-2.50 (2.86)	0.39	-2.10 (1.92)	0.28	2.40 (3.85)	0.54	-0.38 (2.87)	0.90
Drink $\geq$ 10 drinks/wk (Yes)*	4.94 (2.46)	0.0499	3.14 (1.65)	0.06	4.49 (3.44)	0.20	6.02 (2.39)	0.02	-1.16 (4.05)	0.78	-1.99 (3.04)	0.53
Relaxation regularly (Yes)*	-4.94 (3.13)	0.12	-4.26 (2.07)	0.045	-5.19 (3.28)	0.13	-5.02 (2.29)	0.04	0.00		0.00	
Race (Causasian)	1.60 (2.84)	0.58	-2.65 (1.86)	0.16	2.83 (3.30)	0.40	-2.32 (2.26)	0.31	-3.61 (5.08)	0.49	-4.35 (4.06)	0.31
Education (> high school)	2.93 (2.82)	0.30	0.47 (1.83)	0.80	0.19 (3.29)	0.95	-0.18 (2.17)	0.93	8.58 (7.21)	0.26	2.93 (5.44)	0.60
Regular Exercise (Yes)*	-5.68 (3.20)	0.08	-0.45 (2.09)	0.83	-2.16 (3.78)	0.57	-1.68 (2.46)	0.50	-10.36 (8.29)	0.24	1.76 (5.46)	0.75

* Baseline covariates. NOTE1: only 1 male subject regularly practiced relaxation, thus, that β coefficient is zero for males. Family history (FHx), cardiovascular disease (CVD).

Table 58. Standardized and unstandardized effects of post intervention primary predictor variables on post intervention 24-hour blood pressure for all subjects after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	Systolic Blood Pressure		Diastolic Blood Pressure	
	β (SE) Unstandardized	β (SE) Standardized	β (SE) Unstandardized	β (SE) Standardized
T-Ang	0.11 (0.39)	0.05 (0.18)	-0.01 (0.26)	-0.01 (0.14)
T-Ang/T	1.14 (0.82)	0.25 (0.18)	0.40 (0.56)	0.10 (0.14)
T-Ang/R	0.09 (0.61)	0.02 (0.16)	0.04(0.41)	0.01 (0.13)
AXI	0.30 (0.24)	0.13 (0.11)	0.21 (0.17)	0.11 (0.09)
AXO	0.23 (0.40)	0.07 (0.12)	0.00 (0.27)	0.00 (0.09)
PS	-0.03 (0.16)	-0.02 (0.11)	-0.06 (0.11)	-0.05 (0.09)

T-Ang= Trait Anger, T-Ang/T = Trait Anger Temperament, T-Ang/R = Trait Anger Reaction, AXI = Anger Expression Inwards, AXO = Anger Expression Outwards, PS = Perceived Stress.

Table 59. Standardized and unstandardized effects of post intervention primary predictor variables on post intervention daytime blood pressure for all subjects after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictors	Systolic Blood Pressure		Diastolic Blood Pressure	
	β (SE) Unstandardized	β (SE) Standardized	β (SE) Unstandardized	β (SE) Standardized
T-Ang	-0.03 (0.41)	-0.02 (0.18)	-0.10 (0.28)	-0.05 (0.14)
T-Ang/T	0.77 (0.86)	0.16 (0.18)	0.31 (0.60)	0.07 (0.14)
T-Ang/R	0.08 (0.63)	0.02 (0.16)	-0.04(0.44)	-0.01 (0.13)
AXI	0.12 (0.26)	0.05 (0.11)	0.11 (0.18)	0.05 (0.09)
AXO	0.06 (0.42)	0.02 (0.12)	-0.05 (0.28)	-0.02 (0.09)
PS	-0.05 (0.17)	-0.04 (0.12)	-0.10 (0.12)	-0.08 (0.09)

T-Ang= Trait Anger, T-Ang/T = Trait Anger Temperament, T-Ang/R = Trait Anger Reaction, AXI = Anger Expression Inwards, AXO = Anger Expression Outwards, PS = Perceived Stress.

Table 60. Baseline demographic data by sex

Characteristics	Male (n=38)	Female (n=63)
Age (years)	56 (12.4)	56 (10.9)
Waist circ. (cm)	96 (8.9)	87 (11.8)
BMI (kg/m ²)	28 (3.2)	28 (5.6)
Office SBP	133 (13.2)	137 (12.7)
Office DBP	85 (7.3)	86 (7.1)
24 HR SBP	134 (7.3)	135 (8.3)
24 HR DBP	82 (4.7)	82 (6.3)
Daytime SBP	140 (7.6)	141 (7.9)
Daytime DBP	87 (5.4)	87 (6.8)
Nighttime SBP	119 (9.6)	123 (11.6)
Nighttime DBP	70 (7.2)	72 (7.6)
Family History of Hypertension (%)	64%	62%
Family history of premature coronary heart disease	25%	30%
≥ Post-Secondary Education (% yes)	86%	85%
Working (%)	83%	77%
Drinks > 10/week	33%	11%
Use relaxation techniques on a regular basis	3%	25%
Vigorous exercise	78%	61%
Moderate exercise	92%	91%
Self-identify as Caucasian	86%	79%
<i>Data is reported in mean (SD) for continuous variables. SBP = systolic blood pressure, DBP = diastolic blood pressure. All BP measured in mmHg. Vigorous exercise = participation in any weekly vigorous aerobic exercise lasting 20 ≥ minutes. Moderate exercise = participation in any weekly moderate exercise lasting ≥ 30 minutes.</i>		

Table 61. Baseline psychosocial data by sex

Psychometric Scale	Score Males	%ile for Normal Male Adults**	Score Females	%ile for Normal Female Adults***
State Anger	17 (4.3)	60 th	16 (3.9)	55 th
Feeling angry	6 (1.9)	50 th	6 (2.0)	55 th
Feel like expressing anger verbally	6 (2.3)	75 th	5 (0.9)	50 th
Feel like expressing anger physically	5 (0.4)	50 th	5 (1.3)	50 th
Trait Anger	18 (6.3)	60 th	17 (4.5)	55 th
Angry Temperament	7 (3.1)	70 th	6 (2.3)	60 th
Angry Reaction	8 (3.0)	45 th	8 (2.3)	40 th
Anger Expression In	18 (4.5)	90 th	16 (4.3)	80 th
Anger Expression Out	14 (3.7)	40 th	13 (3.0)	30 th
Anger Control In	20 (5.4)	20 th	23 (5.6)	35 th
Anger Control Out	22 (4.8)	45 th	24 (5.4)	50 th
Anger Expression Index (AX index)	38 (13.0)	70 th	31 (14.6)	55 th
Perceived Stress Scale	25 (7.8)	N/A	23 (8.4)	N/A
<i>Data is reported in mean (SD). Percentile ranks were obtained from the State-Trait Anger Expression Inventory-2 Professional Manual. No such percentile ranking exists for the Perceived Stress Scale.</i>				

Table 62. Primary results summary: associations between primary post intervention anger measures and post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

	All Subjects		Females		Males	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Trait Anger	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Trait Anger Temperament	No sig assoc.	No sig assoc.	Sig positive assoc.	No sig assoc.		No sig assoc.
Trait Anger Reaction	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Anger Expression Inwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Anger Expression Outwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Perceived Stress	No sig assoc.	No sig assoc.	Sig negative assoc.	No sig assoc.	Sig positive assoc.	No sig assoc.

NOTE: Grey fill indicate s an overall null model.

Table 63. Primary results summary: associations between primary post intervention anger measures and post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

	All Subjects		Females		Males	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Trait Anger	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Trait Anger Temperament	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		
Trait Anger Reaction	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		
Anger Expression Inwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		
Anger Expression Outwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		
Perceived Stress	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	Sig positive assoc.	No sig assoc.

NOTE: Grey fill indicate s an overall null model.

Table 64. Secondary results summary: associations between post intervention primary anger measures and post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

	All Subjects		Females		Males	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Trait Anger	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Trait Anger Temperament	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Trait Anger Reaction	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Anger Expression Inwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Anger Expression Outwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Perceived Stress	No sig assoc.	No sig assoc.	Sig negative assoc.	No sig assoc.	Sig positive assoc.	No sig assoc.

Table 65. Exploratory results summary: associations between post intervention exploratory anger measures and post intervention 24-hour blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

	All Subjects		Females		Males	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Anger Control Inwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Anger Control Outwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Anger Expression Index	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.

NOTE: Grey fill indicate s an overall null model.

Table 66. Exploratory results summary: associations between post intervention exploratory anger measures and post intervention daytime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

	All Subjects		Females		Males	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Anger Control Inwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Anger Control Outwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.
Anger Expression Index	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.		No sig assoc.

NOTE: Grey fill indicate s an overall null model.

Table 67. Exploratory results summary: associations between post intervention exploratory anger measures and post intervention nighttime blood pressure after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

	All Subjects		Females		Males	
	Systolic	Diastolic	Systolic	Diastolic	Systolic	Diastolic
Anger Control Inwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Anger Control Outwards	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.
Anger Expression Index	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.	No sig assoc.

Table 68. Standardized and unstandardized effects of post intervention trait anger temperament on post intervention 24-hour systolic blood pressure for female subjects after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictor	24-hour Systolic	
	β (SE) Unstandardized	β (SE) Standardized
Trait anger temperament	2.71 (0.95)	0.62 (0.22)

Table 69. Standardized and unstandardized effects of post intervention perceived stress on post intervention systolic blood pressure for female subjects after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictor	24-hour Systolic		Nighttime Systolic	
	β (SE) Unstandardized	β (SE) Standardized	β (SE) Unstandardized	β (SE) Standardized
Perceived Stress	-0.35 (0.15)	-0.30 (0.13)	-0.52 (0.21)	-0.36 (0.14)

Table 70. Standardized and unstandardized effects of post intervention perceived stress on post intervention systolic blood pressure for male subjects after adjustment for baseline blood pressure, baseline psychosocial functioning and other covariates

Predictor	24-hour Systolic		Daytime Systolic		Nighttime Systolic	
	β (SE) Unstandardized	β (SE) Standardized	β (SE) Unstandardized	β (SE) Standardized	β (SE) Unstandardized	β (SE) Standardized
Perceived Stress	0.83 (0.32)	0.49 (0.19)	0.91 (0.36)	0.52 (0.21)	0.61 (0.27)	0.33 (0.15)

28 Figures

Figure 1. Scatter plot of post intervention 24-hour systolic blood pressure correlated with post intervention perceived stress scale score for female participants. Legend: A = 1 observation, B = 2 observations

Figure 2. Scatter plot of post intervention nighttime systolic blood pressure correlated with post intervention perceived stress scale score for female participants. Legend: A = 1 observation, B = 2 observations

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