A Meta-Analysis of Cardiovascular Reactivity to Stress in Persons With and Without High Blood Pressure

by

Talia A. Neal

A thesis submitted to the faculty of Radford University in partial fulfillment of the requirements for the degree of Master of Arts in the Department of Psychology

Thesis Advisor: Dr. Thomas Pierce

April 2022

Copyright 2022, Talia A. Neal

Thomas W. Pierce — 5/5/22

Dr. Thomas Pierce
Thesis Advisor — Date

M. G. — 5/5/22

Dr. Jeffery Aspelmeier
Committee Member — Date

Dayna M Hayes — 5/5/2022

Dr. Dayna Hayes.
Committee Member — Date

Abstract

This study reports a meta-analysis comparing groups with high blood pressure to those with normal blood pressure in terms of increases from baseline in systolic blood pressure, diastolic blood pressure, and heart rate while participants performed challenging or stressful tasks. This meta-analysis consists of information from published studies that were analyzed for differences between the two groups. The differences in participant reactions to challenging task conditions were measured by the mean changes in systolic blood pressure, diastolic blood pressure, and heart rate. Mean change scores were calculated by subtracting the mean baselines for a physiological measure from the mean value for a physiological measure recorded in a particular stressor condition. A series of regression analyses were conducted predicting mean change scores in heart rate (HR), systolic blood pressure (SBP), and diastolic blood pressure (DBP) for high blood pressure groups from the mean change scores for the normal blood pressure groups. For all three measures of cardiovascular function (i.e., HR, SBP, and DBP), slopes were found to be significantly different from 1.0 and y-intercepts were significantly different from zero. This pattern indicates that differences in cardiovascular reactivity between hypertensive and normotensive groups were largest in situations with lower stress levels.

Keywords: *Blood Pressure, Stressors, Cardiovascular Reactivity, Hypertensive, Normotensive, Group Differences.*

Table of Contents

Abstract 2

Table of Contents 3

Chapter 1: Introduction 5

Hypertension 5

Theoretical Perspectives on the Development of Hypertension 6

Genetic History 6

Lifestyle Differences 7

The Effects of Chronic Stress on Resting Levels of Blood Pressure 8

Urban Versus Suburban Versus Rural Environments and Hypertension 8

Occupational Stress and Hypertension 9

Unemployment and Cardiovascular Diseases 10

The Reactivity Hypothesis 10

The Present Study 11

Justification and Explanation of this Regression-Based Meta-Analysis 12

Hypotheses 13

Chapter 2: Method 15

Identification of Published Studies 15

Formation of the Data Set 15

Analysis of Baseline-Stressor Change Scores 16

Chapter 3: Results 17

Regression Analyses Involving Mean Residualized Change Scores for Systolic Blood Pressure 17

Regression Analyses Involving Mean Residualized Change Scores for Diastolic Blood Pressure 18

Regression Analyses Involving Mean Residualized Change Scores for Heart Rate 19

The Moderation Effect of the Type of Stressor on Group Differences in Cardiovascular Reactivity 21

Moderation Effect of Type of Stress for Heart Rate Reactivity 21

Moderation Effect of Type of Stress for Systolic Blood Pressure Reactivity 22

Moderation Effect of Type of Stress for Diastolic Blood Pressure Reactivity 24

Chapter 4: Discussion 26

Aims of the Study 26

Less Discriminant Reactivity to Stress 27

Study Limitations 28

Authors Did Not Report Means Separately 29

Future Directions 30

References..... 31

Chapter 1: Introduction

Hypertension

Hypertension, commonly known as high blood pressure, is an important health issue in the United States. Hypertension is defined as clinically elevated blood pressure levels that are associated with a number of disease states, including coronary heart disease and stroke (Fuchs & Whelton, 2020). Blood pressure is defined as the force of circulating blood against the body's arteries within the body (National Cancer Institute at the National Institutes of Health, 2021*)*. Blood pressure is measured in millimeters of mercury and is commonly reported as two values, where the higher value of systolic blood pressure represents the pressure within blood vessels when the heart contracts and the value for diastolic blood pressure represents blood pressure within blood vessels when the heart rests between heart beats (American Heart Association, 2021).

Hypertension is diagnosed when blood pressure is measured on 2 consecutive days and systolic blood pressure values on both days are 140 mmHg or greater and/or diastolic blood pressure values on both consecutive days are 90 mmHg or greater (WHO, 2021). It is predicted that 1.13 billion individuals globally have hypertension with two-thirds of this number residing in countries that are considered to be of low- and middle-income status (World Health Organization [WHO], 2021). As of 2015, one in four men and one in five women had hypertension, with fewer than one in five individuals across genders having their hypertension under control with medication and/or lifestyle changes (WHO, 2021). Hypertension is a significant factor in premature deaths; managing hypertension and reducing risk factors for hypertension helps prevent a wide array of health issues such as heart attack, heart disease, heart

failure, kidney damage, kidney disease, kidney failure, organ failure, tissue death, stroke, and death (Mills et al., 2020; WHO, 2021).

Theoretical Perspectives on the Development of Hypertension

Several ideas have been proposed for why individuals develop hypertension. This section covers three significant, overlapping factors: genetic history, lifestyle, and the reactivity hypothesis.

Genetic History

Blood pressure is of interest to clinicians and geneticists because it is a significant cardiovascular risk factor as well as a classic quantitative genetic trait (i.e., a measurable phenotype dependent on actions of genetic and environmental factors; McKusick, 1960; Miall & Oldham, 1963; Springer Nature, 2021). Thus, blood pressure changes are due to a combination of genetic (i.e., degree of family history of hypertension, cardiac malfunction, cardiac disease, etc.) and environmental factors (i.e., degree of stressors present, lifestyle differences, etc.; McKusick, 1960; Miall & Oldham, 1963; Springer Nature, 2021).

Family and twin studies have found that the fraction of the expression of hypertension explained by genes (i.e., heritability) is 35% to 65% (Kupper et al., 2005; Levy et al., 2007; Miall & Oldham, 1963; Moll et al., 1983; Rotimi et al., 1999; Snieder et al., 2000). Regarding the importance of the role of genes in hypertension, there is strong empirical evidence that hypertension is 2.4 times more common in persons who have two hypertensive parents (Wang et al., 2008). It was discovered that parental and maternal hypertension were found to be strongly and independently related to higher blood pressure levels and to the chance of developing hypertension across the lifespan (Wang et al., 2008). Higher blood pressure levels during the end of adolescence and significant increases in blood pressure measurements (specifically, systolic

blood pressure) during adult life in men who had a parent or parents with hypertension placed men at higher future risk for developing hypertension (Wang et al., 2008). Men who had both parents diagnosed with hypertension or men with one hypertensive parent before age 55 had a heightened risk of developing hypertension at younger ages.

Blood pressure heritability in families is again likely to be attributed both to shared environmental exposures and to genetic susceptibility. Regarding the environment, blood pressure concordance (i.e., the same traits of hypertension present in both twins) is higher between spouses than between non-spouses due to the likelihood to conform to similar lifestyle and behavioral patterns (Morton et al., 1980). Genetically, blood pressure concordance is higher between biological siblings than adoptive siblings who share the same household (Biron et al., 1976; Biron & Mongeau, 1978; Mongeau et al., 1986; Rice et al., 1989). Concordance is also higher in monozygotic (i.e., identical) twins compared to dizygotic (i.e., fraternal) twins (Pickering, 1968). Identical twins result from the fertilization of a single egg splitting into two; therefore, they share all genes and are of the same sex (National Human Genome Research Institute [NHGRI], 2021). On the other hand, fraternal twins are the product of two different eggs being fertilized during the same pregnancy; therefore, they share half of their genes like any siblings do and can be of different sexes (NHGRI, 2021). Thus, identical twins would share the exact copies of genomes for the susceptibility of hypertension despite the environments they are reared in, whereas fraternal twins would have a lower concordance rate for developing hypertension because their genes are not identical.

Lifestyle Differences

Lifestyle factors such as physical activity and fitness, dietary salt intake, and complex dietary changes with regards to fruits, vegetables, fats, and fiber in the diet have a significant

amount of influence on the development of hypertension (Beilin et al., 1999). For example, salt reduction was found to be helpful in decreasing blood pressure levels among hypertensive individuals as well as the maintenance of consistent healthy blood pressure levels in normotensive individuals (Beilin et al., 1999; Law et al., 1991; Webster et al., 2011).

Lower incidences of hypertension were found in vegetarian populations consuming large amounts of vegetables, fruits, and fiber with low levels of saturated fat (Beilin et al., 1999; Chuang et al., 2016). In studies involving maintenance of exercise programs and weight tracking, blood pressure is shown to decline and stabilize when consistent exercise is paired with weight reduction (Beilin et al., 1999; Choudhury & Lip, 2005; Guimarães et al., 2010). In this case, weight loss had dominant effects on blood pressure status, and effects of lowering blood pressure levels were stronger when weight loss was paired with exercise (Beilin et al., 1999; Choudhury & Lip, 2005; Guimarães et al., 2010). Within hypertensive populations, activities such as jogging, swimming, cycling, or walking 40 minutes approximately three times per week aid in long-term lowering of blood pressure and improved sensitivity to insulin (Beilin et al., 1999).

The Effects of Chronic Stress on Resting Levels of Blood Pressure

Urban Versus Suburban Versus Rural Environments and Hypertension

Exposure to chronic stressors such as violence, occupational stress, and social environment have been correlated with being a risk factor for hypertension (Rosenthal & Alter, 2012; Spruill, 2010). Blood pressure levels spike temporarily after exposure to stressors; however, long term exposure to stressors could lead to increases in blood pressure, causing hypertension (Rosenthal & Alter, 2012; Spruill, 2010). For example, rates of serious violence per thousand people living in urban areas was significantly greater than serious violence rates in rural areas and slightly higher than serious crime rates in suburban populations (Berg &

Lauritsen, 2016). Thus, one's immediate environment may serve as a potential risk factor for the development of hypertension through consistent exposure to stressors in an individual's immediate environment such as one's city of residence or workplace.

Occupational Stress and Hypertension

Rates of hypertension incidences are found to be significantly higher in jobs with higher levels of job stress. Babu et al. (2013) suggested that among Indian IT professionals, hypertension occurs a decade earlier when compared to the rest of the population in India as well as two decades earlier when compared to developing countries. A total of 46% of IT professionals were classified as pre-hypertensive (a state identifying one as at high-risk of developing hypertension; Babu et al., 2013; Chobanian et al., 2003). This is significant as hypertension is a risk factor for cardiovascular disease. Cardiovascular diseases (CVD) lead to 25% of deaths within the Indian continent, and there is a high incidence rate for CVD in both rural and urban areas (Babu et al., 2013). When compared to professionals who have worked for over 12 years, Babu et al. (2013) found higher rates of hypertension among professionals who were in their early years of joining IT (Babu et al., 2013). A relationship was found where the odds of being diagnosed with hypertension decreased as the number of years worked by IT professionals increased (Babu et al., 2013). Many reasons may explain this, such as professionals who develop hypertension in earlier years as the result of an interaction between cardiovascular risk factors and job stress either leave the workforce or gradually adapt and develop improved coping mechanisms for their stressful situation (Babu et al., 2013). Individuals with higher levels of work/environment-related stressors and autonomy-related stress tend to have higher hypertension prevalence when compared to those with lower stress levels (Babu et al., 2013).

Unemployment and Cardiovascular Diseases

Janlert. (1992) and Hammarstroem (1994) discovered a positive correlation with periods of unemployment and increases in systolic and diastolic blood pressure. A study by Fox and Goldblatt (1981) described a non-differentiated and slightly increased cardiovascular mortality as present for unemployed individuals, while other researchers such as Iversen and Sabroe (1987) had discovered a significantly increased prevalence rate of cardiovascular heart disease with increased length of unemployment periods (Fox & Goldblatt, 1981; Iversen & Sabroe, 1987). Research by Brackbill et al. (1995) and Iversen and Sabroe (1987) also found an association between increased incidence of hypertension and heart infarction with increases in unemployment periods.

The Reactivity Hypothesis

Cardiovascular reactivity (CR) is defined as changes in measures of cardiovascular function measures (i.e., heart rate, blood pressure, etc.) in the interval between baseline periods and physical/psychological challenge or external stressors being presented to the individual (Cinciripini, 2021; Kamarck & Lovallo, 2003; Maier et al., 2003; Vitaliano et al., 1993). Some individuals show heightened cardiovascular reactivity whereas others show lower levels for a variety of different reasons (i.e., hypertension, normal levels of blood pressure, stressor magnitude; Cinciripini, 2021; Huang et al., 2013; Lovallo, 2005). The identification of a person as having exaggerated responses varies from selecting individuals showing the largest responses in their group to those who show cardiovascular responses that exceed the metabolic demands of a situation (Sherwood et al., 1986; Zanstra & Johnston, 2011).

Schneiderman et al. (2004) described the classic reactivity hypothesis as stating that people who are considered high reactors in response to behavioral challenges or stressors will be

more likely over time to develop elevated blood pressure levels and forms of heart disease (i.e., conditions affecting heart structure and function; Allender et al., 2007; National Heart, Lung, & Blood Institute (NIH), 2021; Schneiderman et al., 2004). The high stress response could be considered a marker of increased risk for cardiovascular diseases, or it may contribute directly to the development of cardiovascular diseases (Manuck, 1994; Schneiderman et al., 2004). Large cardiovascular responses to stress measures such as elevated blood pressure or heart rate after exposure to a stressor contribute directly to the development of hypertension and cardiovascular disease (Manuck, 1994; Schneiderman et al., 2004). Thus, situations where high cardiovascular responses are evoked on a frequent basis in one's environment (i.e., work or home) may result in consistently higher resting levels of blood pressure through structural changes in the heart and blood vessels (Schneiderman et al., 2004).

The Present Study

To date no study has compared different levels of stress or challenge in a study comparing hypertensive and normotensive groups in order to examine whether the level of stress elicited in a situation moderates the size of hypertensive/normotensive differences in cardiovascular reactivity to stress. The purpose of this meta-analysis was to speak to this question by examining the relationship between the level of stress (as measured by mean normotensive increases in blood pressure) and group differences in cardiovascular reactivity. By use of regression this study identified the conditions that elicited the largest differences in cardiovascular reactivity in systolic blood pressure, diastolic blood pressure, and heart rate between hypertensive and normotensive groups. This regression-based meta-analysis also determined whether larger differences between the two groups (hypertensive individuals and those without hypertension) were observed under lower or higher stress conditions. Specifically,

this study extracted mean baseline-stressor change scores for both normotensive and hypertensive groups from a variety of stressor conditions reported in papers examining the effect of blood pressure status on cardiovascular reactivity. Each pair of mean baseline-stressor change scores formed a case within a regression analysis where the mean baseline-stressor change scores obtained from normotensive groups were used to predict mean baseline-stressor change scores for hypertensive groups.

Justification and Explanation of this Regression-Based Meta-Analysis

The rationale for this use of regression came from the methods used by Pierce et al. (2005), which examined whether the effect of a family history of hypertension on three measures of cardiovascular reactivity (systolic blood pressure, diastolic blood pressure, and heart rate) was moderated by variations in the size of cardiovascular responses resulting from stressor/task conditions (physical stressors, psychological stressors, and psychosocial stressors). Mean change scores for participants without a family history of hypertension were used to predict mean change scores for participants with a family history of hypertension. For all three measures of cardiovascular function, the y-intercept of the regression line was significantly greater than zero and the slope of the regression line was significantly less than 1.0. This pattern indicated that the largest family history differences in cardiovascular reactivity to stress were found when baseline-stressor change scores in groups without a family history of hypertension were smallest, reflecting a low level of situational stress. The authors interpreted this pattern to indicate that groups with a family history of hypertension do not modulate their cardiovascular responses to meet the specific needs of the situation to the same degree as do groups without a family history of hypertension.

The novel contribution of the present study is the application of this regression-based meta-analytic strategy to a comparison of groups different from those previously examined by Pierce et al. (2005). Pierce et al. (2005) compared cardiovascular responses in groups with and without a family history of hypertension (i.e., persons at risk for hypertension compared to persons not at risk for hypertension). This present study compared cardiovascular responses in groups that already had a diagnosis with hypertension to cardiovascular responses in groups that did not have a diagnosis of hypertension.

Hypotheses

A series of regression analyses was conducted using mean baseline-stressor change scores for systolic blood pressure, diastolic blood pressure, and heart rate for normotensive groups to predict mean baseline-stressor change scores in hypertensive groups. A slope of the regression line significantly greater than 1.0 would mean that higher magnitude stressors (as indicated by the presence of large baseline-stressor change scores for normotensive groups) are associated with larger differences in cardiovascular reactivity between hypertensive and normotensive groups. A slope of the regression line of 1.0 would indicate that the magnitude of stress does not moderate differences in cardiovascular reactivity between hypertensive and normotensive groups. Lastly, a slope of the regression line that is significantly less than 1.0 would indicate that higher magnitude stressors are associated with smaller group differences in cardiovascular reactivity between hypertensive and normotensive groups.

Consistent with the results of Pierce et al. (2005), it was predicted that for all three measures of cardiovascular function (heart rate, systolic blood pressure, and diastolic blood pressure), the slope of the regression line would be significantly less than 1.0. Also, the y-intercept of regression equations obtained for the three measures of cardiovascular function

would be significantly greater than zero. The pattern would indicate that the largest differences in cardiovascular reactivity between hypertensive and normotensive groups were observed in conditions with lower levels of stress. Likely, this would be due to the fact that hypertensive individuals display less variability in cardiovascular responses across levels of stress, challenge, or threat than do normotensive persons.

Chapter 2: Method

Identification of Published Studies

Collaborative efforts have generated a meta-analytic data set of peer-reviewed research papers comparing hypertensive to normotensive groups on cardiovascular responses to a variety of stressors. Research papers were identified from peer-reviewed research journals through databases such as PsycINFO and PubMed utilizing keyword searches of the following terms: "cardiovascular reactivity," "cardiovascular responsivity," "hypertension and cardiovascular reactivity," and "hypertension and cardiovascular responsivity," along with finding published studies through the reference sections of published research papers comparing hypertensive and normotensive populations. To be included in the meta-analysis research papers had to meet three conditions: (a) they provided comparisons of hypertensive individuals and normotensive individuals, (b) they separately provided mean change scores for hypertensive and normotensive individuals, and (c) they were published in a peer-reviewed journal.

Formation of the Data Set

Each case in the meta-analytic dataset was compromised of information taken from a comparison of hypertensive and normotensive groups in a single stressor condition extracted from a single study. For each paper, a separate case was generated for every stressor condition examined. Within a stressor condition, cases were also separated by gender, race, and the type of stressor (cognitive, physical, and social). This method allowed for the possibility of moderation analyses in which separate regression lines could be compared for (a) men and women, (b) Black and White participants, and (c) the type of stressor (cognitive, social, physical).

Cases of the dataset were made up of the mean baseline value, mean stressor value, and mean baseline-stressor change scores for hypertensive and normotensive groups for each of the

following cardiovascular measures: systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart/pulse rate (HR/PR) (Pierce et al., 2005). Not all studies reported the means for all three cardiovascular measures. Cases with change score values less than zero were not included in statistical analyses of this paper.

Analysis of Baseline-Stressor Change Scores

To control for differences in baseline values across studies, I obtained residualized mean change scores for each cardiovascular variable (i.e., SBP, DBP, HR) using the method employed by Pierce et al. (2005). Baseline values were regressed on mean change score values and unstandardized residuals for mean change scores were saved as variables for each of the three cardiovascular variables. For each cardiovascular variable, the mean change score was added to the residuals (i.e., SBP, DBP, HR) to produce values in the original change score units.

After obtaining residualized mean change scores, separate analyses for the three cardiovascular measures were conducted where mean residualized change scores for normotensive groups were entered into regression models predicting mean residualized change scores of hypertensive groups. The slope of the regression equation was tested against a comparison value of 1.0, and the y-intercept was tested against a comparison value of zero. Moderation analyses were conducted to examine the degree to which the slope of the regression line varied according to the type of stressor. Moderation analyses were conducted using the procedures outlined in Aiken and West (1991).

Chapter 3: Results

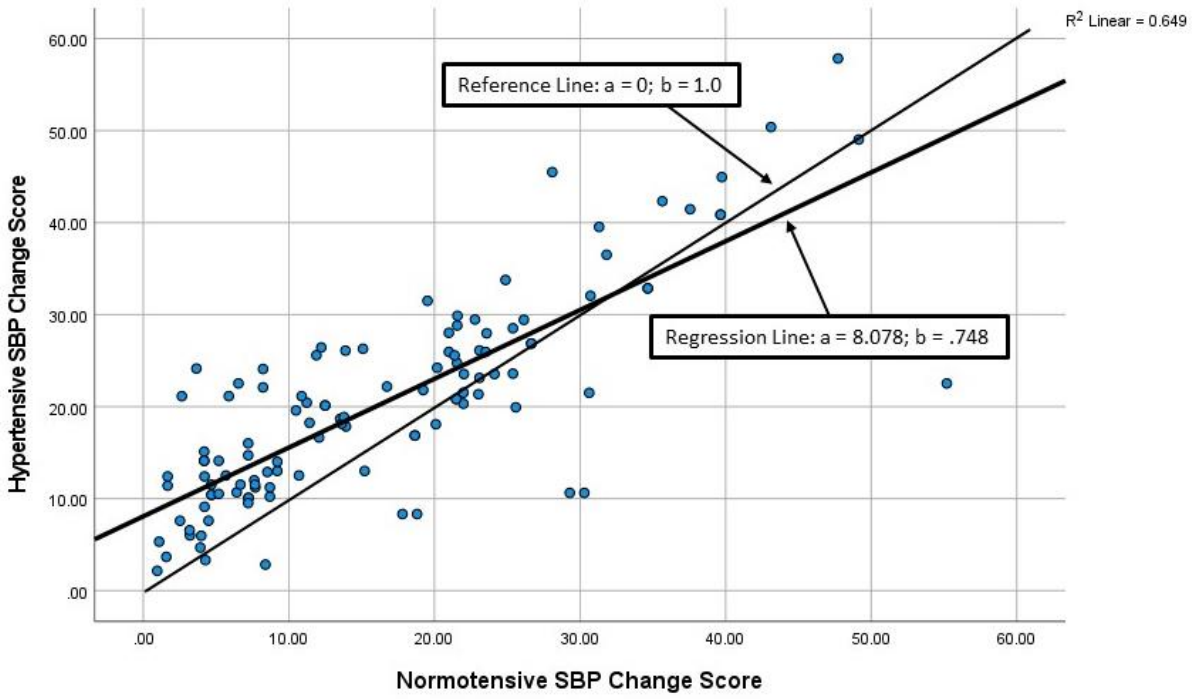
Regression Analyses Involving Mean Residualized Change Scores for Systolic Blood Pressure

Mean residualized systolic blood pressure change scores for normotensive groups were entered into a regression model predicting mean residualized systolic blood pressure change scores for hypertensive groups. The scatterplot and regression line for this analysis are presented in Figure 1. Change scores for normotensive groups accounted for a significant amount of variability in change scores for hypertensive groups, $F(1,112) = 207.272$, $p < 0.001$, $R^2 = 0.649$. The slope of the regression line ($b = 0.748$, $SE = 0.052$) was significantly less than one, $t(112) = -4.846$, $p < 0.001$, indicating that larger differences in systolic blood pressure reactivity to stress for hypertensive groups were observed when change scores for normotensive groups were small. The y-intercept for this regression model ($a = 8.078$, $SE = 1.054$) was significantly greater than zero, $t(112) = 7.664$, $p < 0.001$.

The same pattern of results was observed when change scores that had not been statistically adjusted for baseline differences were used. The regression equation predicting mean change scores for hypertensive groups from mean change scores for normotensive groups had a slope of 0.865 ($SE = 0.045$) and a y-intercept of 6.038 ($SE = 0.890$).

Figure 1

Scatterplot of Mean Residualized Change Scores for Systolic Blood Pressure for Hypertensive and Normotensive Groups



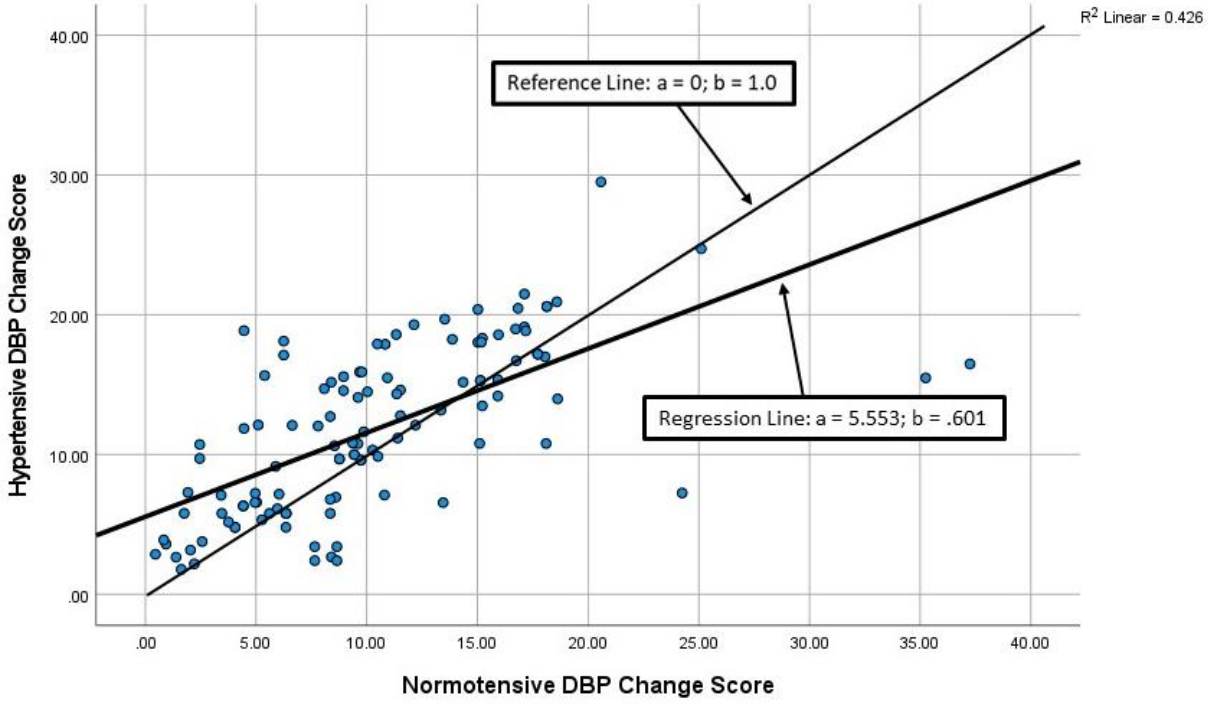
Regression Analyses Involving Mean Residualized Change Scores for Diastolic Blood Pressure

Mean residualized diastolic blood pressure change scores for normotensive groups were entered into a regression model predicting mean diastolic blood pressure change scores for hypertensive groups. The scatterplot and regression line for this analysis are presented in Figure 2. Change scores for normotensive groups accounted for a significant amount of variability in change scores for hypertensive groups, $F(1,112) = 83.281$, $p < 0.001$, $R^2 = 0.426$. The slope of the regression line ($b = 0.601$, $SE = 0.065$) was significantly less than one, $t(112) = -6.045$, $p < 0.001$. The y-intercept for this regression model ($a = 5.553$, $SE = 0.800$) was significantly greater than zero, $t(112) = 6.944$, $p < 0.001$.

The same pattern of results was observed when change scores that had not been statistically adjusted for baseline differences were used. The regression equation predicting mean change scores for hypertensive groups from mean change scores of normotensive groups had a slope of $b = 0.774$, $SE = 0.062$, and a y-intercept of $a = 3.687$, $SE = 0.738$.

Figure 2

Scatterplot of Mean Residualized Change Scores for Diastolic Blood Pressure for Hypertensive and Normotensive Groups



Regression Analyses Involving Mean Residualized Change Scores for Heart Rate

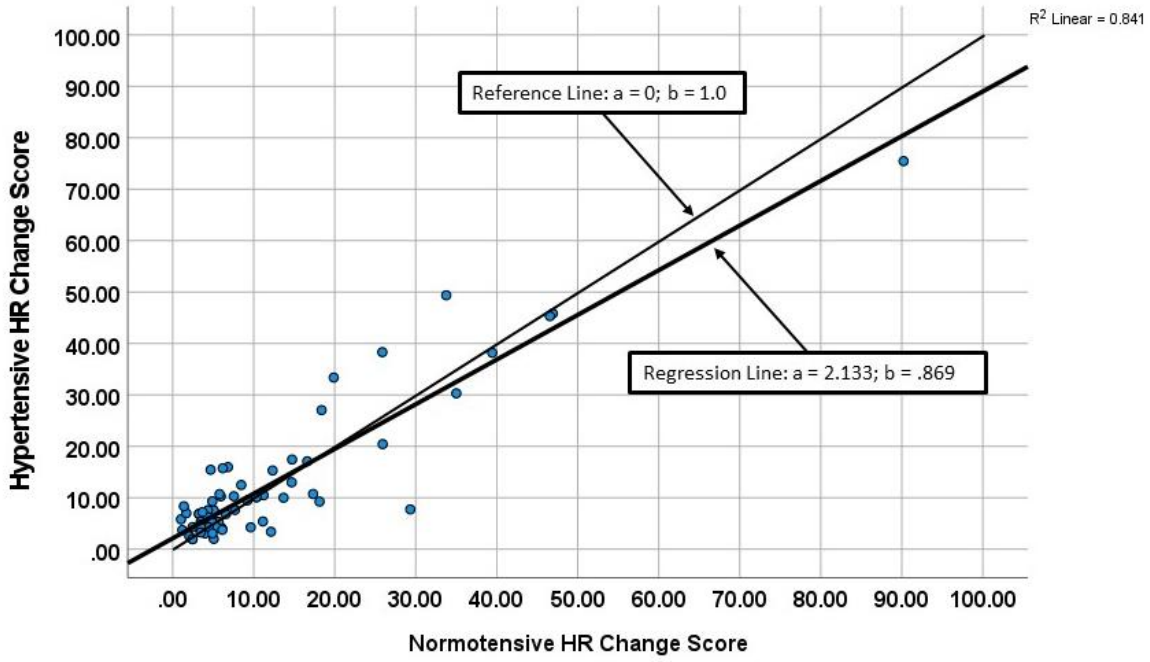
Mean residualized heart rate change scores for normotensive groups were entered into a regression model predicting mean residualized heart rate change scores for hypertensive groups. The scatterplot and regression line for this analysis are presented in Figure 3. Change scores for normotensive groups accounted for a significant amount of the variability in change scores for hypertensive groups, $F(1, 76) = 403.242$, $p < 0.001$, $R^2 = 0.841$. The slope of the regression line

(b = 0.869, SE = 0.043) was significantly less than one, $t(76) = -3.047$, $p < 0.001$. The y-intercept for this regression equation (a =2.133, SE = 0.744) was significantly greater than zero, $t(84) = 2.869$, $p = 0.005$.

When non-residualized change scores for heart rate were utilized, the y-intercept (a = 2.828, SE = 0.713) was significantly greater than zero. The slope of the regression equation (b= 0.837, SE = 0.043) was significantly less than 1.0. This pattern shows that hypertensive differences in heart rate reactivity change significantly across the range of baseline-stressor change scores seen in normotensive groups.

Figure 3

Scatterplot of Mean Residualized Change Scores for Heart Rate for Hypertensive and Normotensive Groups



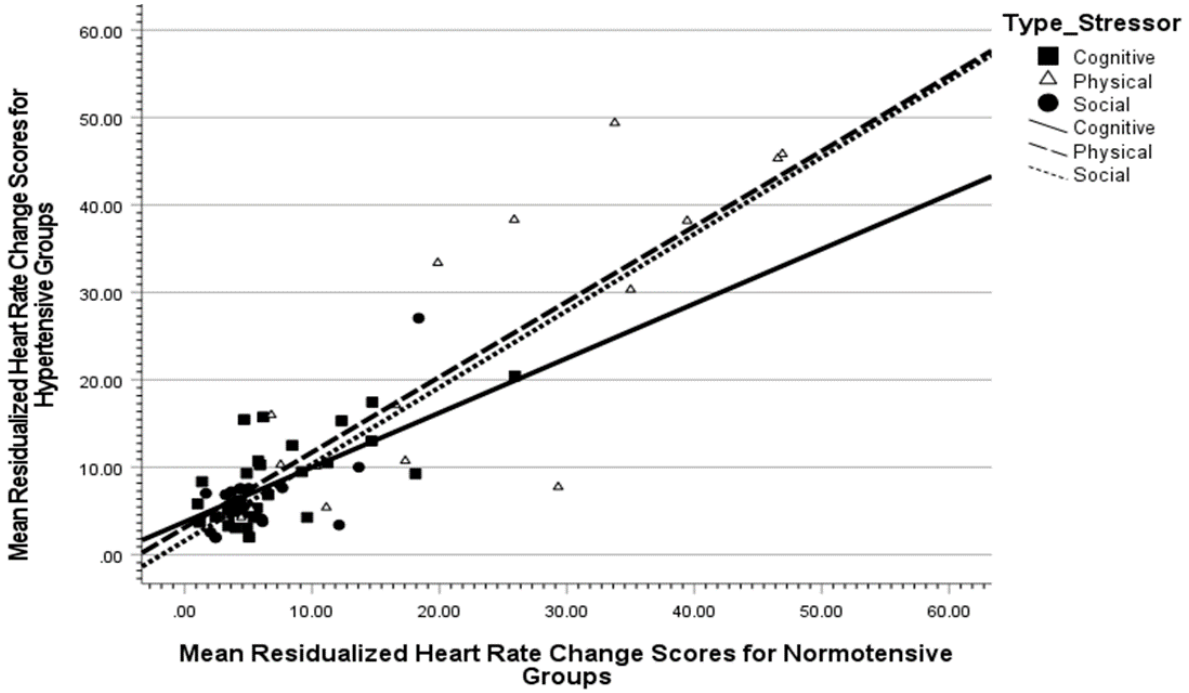
The Moderation Effect of the Type of Stressor on Group Differences in Cardiovascular Reactivity

Moderation Effect of Type of Stress for Heart Rate Reactivity

A moderation analysis was conducted assessing the ability of the type of stress (cognitive, physical, or social) to moderate the strength of the relationship between normotensive baseline-stressor change scores and hypertensive baseline-stressor change scores. Thirty-two, 20, and 26 cases were available for cognitive, physical, and social stressor conditions, respectively. The effect of Type of Stressor was captured through two effect coded variables. Values for the continuous predictor variable, residualized baseline-stressor change scores for normotensives, were centered. Two variables capturing variability associated with the moderation effect were constructed by multiplying each effect coded variable for Type of Stressor by the centered variable for normotensive baseline-stressor change scores. The moderation effect was tested by entering the two effect coded variables for Type of Stressor and the centered variable for normotensive baseline-stressor change scores in Block 1 of a regression model predictions residualized baseline-stressor change scores for hypertensives. Variables capturing the moderation effect were entered in Block 2 of the regression model. The unique contribution of the two moderation variables was not statistically significant, R^2 Change = 0.004, $F(2, 72) = 0.847$, $p = 0.433$, indicating that the type of stress does not moderate the slope of the regression line (see Figure 4).

Figure 4

Scatterplot of Mean Residualized Change Scores for Heart Rate for Hypertensive and Normotensive Groups



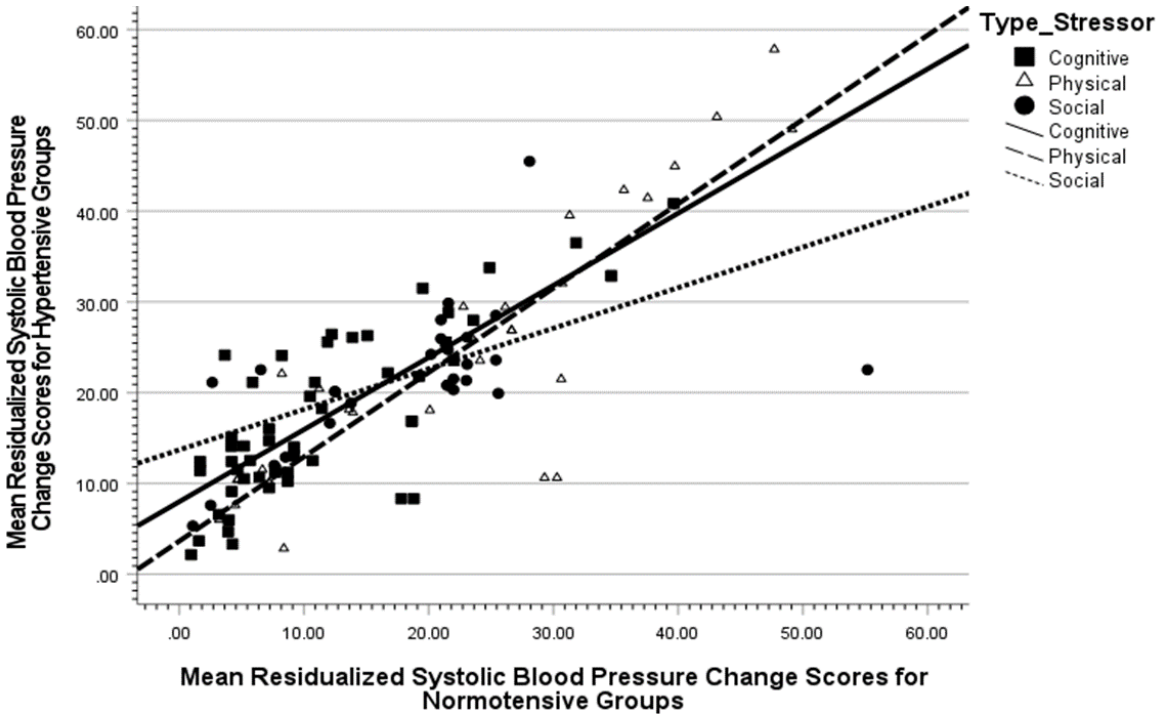
Moderation Effect of Type of Stress for Systolic Blood Pressure Reactivity

A moderation analysis was conducted assessing the ability of the type of stress (cognitive, physical, or social) to moderate the strength of the relationship between normotensive baseline-stressor change scores for systolic blood pressure and hypertensive baseline-stressor change scores. For this measure, 54, 31, and 28 cases were available for cognitive, physical, and social stressor conditions, respectively. The effect of Type of Stressor was captured through two effect coded variables. Values for the continuous predictor variable, residualized baseline-stressor change scores for normotensives, were centered. Two variables capturing variability associated with the moderation effect were constructed by multiplying each effect coded variable for Type of Stressor by the centered variable for normotensive baseline-stressor change scores.

The moderation effect was tested by entering the two effect coded variables for Type of Stressor and the centered variable for normotensive baseline-stressor change scores in Block 1 of a regression model predicting residualized baseline-stressor change scores for hypertensives. Variables capturing the moderation effect were entered in Block 2 of the regression model. The unique contribution of the two moderation variables was statistically significant, R^2 Change = 0.037, $F(2, 107) = 6.349$, $p = 0.002$, indicating that the type of stress does moderate the slope of the regression line. The slope of the regression line for social stressors ($b = 0.45$) was significantly lower than that for physical stressors ($b = 0.93$), $F(1, 109) = 6.79$, $p = .011$. The slope of the regression line for social stressors was significantly lower than that for cognitive stressors ($b = 0.79$), $F(1, 109) = 10.05$, $p = .002$. See Figure 5.

Figure 5

Scatterplot of Mean Residualized Change Scores for Systolic Blood Pressure for Hypertensive and Normotensive Groups

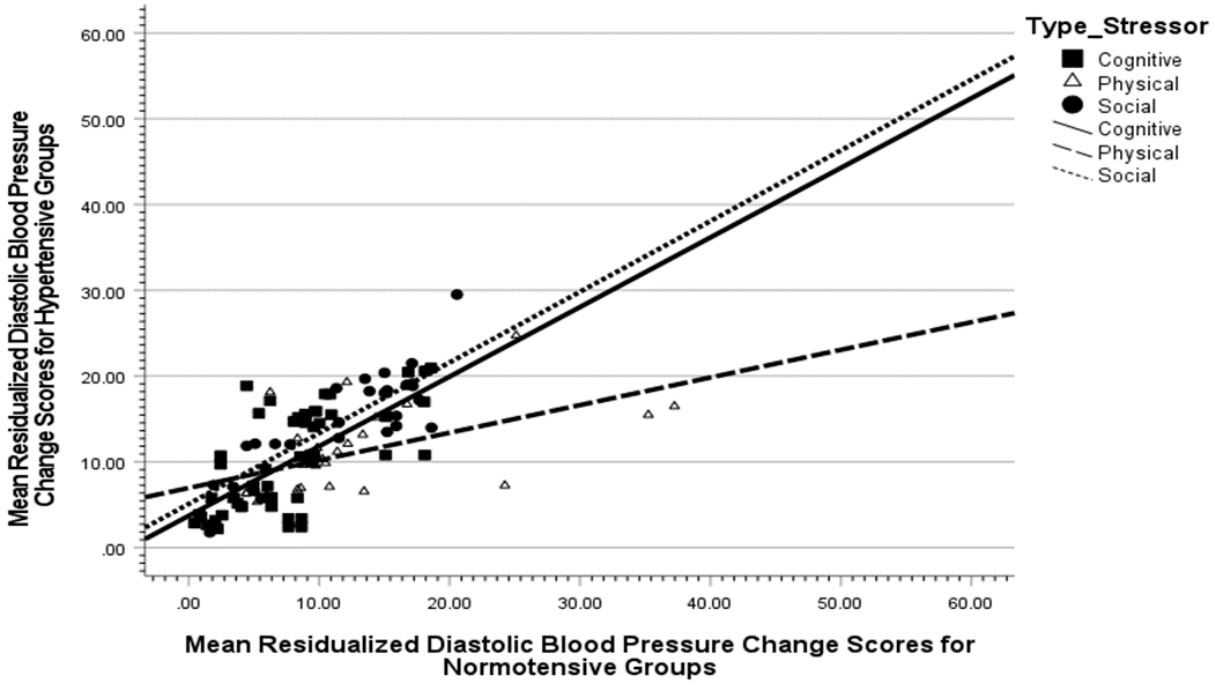


Moderation Effect of Type of Stress for Diastolic Blood Pressure Reactivity

A moderation analysis was conducted assessing the ability of the type of stress (cognitive, physical, or social) to moderate the strength of the relationship between normotensive baseline-stressor change scores for diastolic blood pressure and hypertensive baseline-stressor change scores. For this measure, 54, 30, and 29 cases were available for cognitive, physical, and social stressor conditions, respectively. The effect of Type of Stressor was captured through two effect coded variables. Values for the continuous predictor variable, residualized baseline-stressor change scores for normotensives, were centered. Two variables capturing variability associated with the moderation effect were constructed by multiplying each effect coded variable for Type of Stressor by the centered variable for normotensive baseline-stressor change scores. The moderation effect was tested by entering the two effect coded variables for Type of Stressor and the centered variable for normotensive baseline-stressor change scores in Block 1 of a regression model predicting residualized baseline-stressor change scores for hypertensives. Variables capturing the moderation effect were entered in Block 2 of the regression model. The unique contribution of the two moderation variables was statistically significant, R^2 Change = 0.063, $F(2, 107) = 7.646$, $p < 0.001$, indicating that the type of stress moderates the slope of the regression line. The slope of the regression line for physical stressors ($b = 0.32$) was significantly lower than the slope of the regression line for cognitive stressors ($b = 0.81$), $F(1, 80) = 9.77$, $p = .002$. The slope of the regression line for physical stressors was significantly lower than that for social stressors ($b = 0.82$), $F(1, 55) = 10.93$, $p = .002$. See Figure 6.

Figure 6

Scatterplot of Mean Residualized Change Scores for Diastolic Blood Pressure for Hypertensive and Normotensive Groups



Chapter 4: Discussion

Aims of the Study

Previous research has not addressed whether the largest differences in cardiovascular reactivity between groups with and without hypertension are observed in high stress or low stress conditions. This study examined the relationship between the level of stress (as measured by mean normotensive increases in blood pressure) and group differences in cardiovascular reactivity. This regression-based meta-analysis tested whether larger group differences in cardiovascular reactivity between the two groups (i.e., hypertensive individuals and those without hypertension) are observed in either lower or higher stress conditions. A series of regression analyses were conducted utilizing mean baseline-stressor change scores for cardiovascular measures of systolic blood pressure, diastolic blood pressure, and heart rate for normotensive groups to predict mean baseline-stressor change scores in hypertensive groups. This method of regression-based meta-analysis was first used by Pierce et al. (2005), who had investigated how the cardiovascular reactivity of those with and without a family history of hypertension are moderated in high and low stress challenge/task conditions.

For all measures of cardiovascular reactivity (systolic blood pressure, diastolic blood pressure, and heart rate), slopes of the regression line predicting change scores for hypertensive groups from change scores for normotensive groups were found to be significantly less than 1.0 and the y-intercept was found to be significantly greater than zero. This pattern indicates that the largest differences in cardiovascular reactivity between hypertensive and normotensive groups were observed in conditions with lower stress levels, as indicated by the presence of small baseline-stressor change scores in normotensive groups. This pattern was seen in (a) individual analyses conducted for cognitive, social, and physical stressors, and (b) analyses utilizing change

scores that were not adjusted for baseline values. This indicates that persons with high blood pressure moderate their cardiovascular responses across the range of stressors examined (i.e., high and low stress situations/challenges) to a lesser degree than do persons without high blood pressure.

A set of moderation analyses for the three measures of cardiovascular function was conducted to examine the ability of the type of stress (cognitive, physical, or social) to moderate the strength of the relationship between residualized change scores for hypertensive groups and residualized change scores for normotensive groups. The patterns of results were inconsistent for heart rate, systolic blood pressure, and diastolic blood pressure. For heart rate, no moderation effect of the type of stressor was observed. For systolic blood pressure, a significant moderation effect of the type of stressor was observed in which the slope of the regression line for physical stressors was significantly lower than the stress of the regression lines for cognitive or social stressors. For diastolic blood pressure, a significant moderation effect of the type of stressor was observed, but for this measure, the slope of the regression line for social stressors was significantly lower than the slope of the regression lines for cognitive or physical stressors. The reason for these inconsistent results is unclear, but should be investigated further in future research.

Less Discriminant Reactivity to Stress

The primary implication of this pattern is that situations that elicit small increases in blood pressure and heart rate in persons with normal blood pressure are associated with much larger increases in those variables in persons with high blood pressure. This results in a situation where the blood pressure of hypertensives goes up more frequently in the presence of these low-level stressors. It may be this increased frequency of moderately sized responses, rather than the

magnitude of responses, that has led to a resetting of resting levels of blood pressure to these higher values.

Study Limitations

As suggested by Pierce et al. (2005), researchers must consider important points of this study's methodology when interpreting results for this specific method of regression based meta-analysis. First, group means are the only values that contribute to the regression and moderation analyses conducted on residualized and non-residualized change score data. This means that variability of scores within each group is not available for analysis. This precludes the ability to provide effect size data of the number of standard deviations separating mean change scores for hypertensive and normotensive groups. Second, despite the fact that sample sizes used to compute group means for one case could differ from the sample sizes used to compute group means for another case, each case is given equal weight in the determining of regression equations. For example, studies with low sample sizes possess the same influence over the regression equations generated as do studies with large sample sizes. Third, studies containing larger numbers of stressor conditions could have greater influence on the regression analyses conducted. In this study, however, no one study contributed more than seven cases. Furthermore, in cases from individual studies, the pattern of results was found to be consistent with the pattern of results found utilizing all cases.

As the final and perhaps the most important limitation of this study, the results, like those of many other meta-analyses, were subject to a prevalent issue in meta-analytic research known as the File Drawer Problem. The File Drawer Effect, first noted by Robert Rosenthal (1979), referenced a publication bias problem in science where a significant number of results remain unpublished, especially those with negative results. Results largely influence whether or not the

study is published by assessing if results are statistically significant, practically significant, and agree with expectations of researchers (Rosenthal, 1979). If a study does not meet these criteria, it is deemed unworthy of publication; thus, researchers are largely left with exposure to studies with significant results and without knowledge of the results of unpublished studies. Negative, neutral, or statistically non-significant research findings are tucked away into file drawers, essentially becoming inaccessible to meta-analytic researchers (Praveen et al., 2016; Rosenthal, 1979). According to Praveen et al. (2016), for every published significant result, there are 19 non-significant studies that are unpublished.

Perhaps, if it were not for the file drawer problem, in a world with access to many more unpublished studies, the results of this meta-analysis could have shifted based on the unpublished data of other researchers. If the file drawer problem did not exist, this study would be subject to change from the results of unpublished studies on hypertensive versus normotensive individuals' stress responses. Because only peer-reviewed published studies were utilized, and unpublished studies were not accessible, there was exposure to the file drawer problem in this study.

Authors Did Not Report Means Separately

Within the selected meta-analyses, many authors did not report means separately for a variety of different categories, some of which included separate means for male and female groups; racial groups (Black, White, Asian, Hispanic, etc.); body mass index; exercise habits; diet; and smoking, among others. Many authors do not think to report these, and participants in these subcategories are combined to form larger groups. For example, racial groups and male and female groups are often grouped together. If authors had reported means separately for the subcategories, additional moderation analyses could have been conducted examining whether the slopes of regression lines differ significantly from one category to another.

Future Directions

The results of this study show that differences in cardiovascular reactivity between persons with and without hypertension are largest in situations with lower levels of stress. This pattern indicates that hypertensive individuals modulate cardiovascular responses across low to high stressor situations to a lesser degree than those without hypertension. This signals an opportunity to apply techniques in stress management and cognitive behavioral therapy to change the perceptions of low-level stressors before the development of hypertension so they elicit smaller cardiovascular responses. Future studies can provide additional information on how diet, racial group, and/or gender differences provide different rates of likelihood toward the development of hypertension based on a multitude of cultural, environmental, and genetic factors.

References

Aiken, L. S., & West, S. G. (1991). *Multiple regression: Testing and interpreting interactions*. Sage Publications.

Akpolat, T., Erdem, Y., Derici, U., Erturk, S., Caglar, S., Hasanoglu, E., Karatan, O., Sindel, S., & Turgan, C. (2012). Use of home sphygmomanometers in Turkey: A nation-wide survey. *Hypertension Research*, *35*(3), 356-361.

Allender, S., Peto, V., Scarborough, P., Boxer, A., & Rayner, M. (2007). *Coronary heart disease statistics*. British Heart Foundation Health Promotion Research Group.

American Heart Association. (2021, August 10). *Understanding blood pressure readings*. www.heart.org. Retrieved October 20, 2021, from https://www.heart.org/en/health-topics/high-blood-pressure/understanding-blood-pressure-readings

Babu, G. R., Mahapatra, T., & Detels, R. (2013). Job stress and hypertension in younger software professionals in India. *Indian Journal of Occupational and Environmental Medicine*, *17*(3), 101-107.

Beilin, L. J., Puddey, I. B., & Burke, V. (1999, September 1). *Lifestyle and hypertension*. OUP Academic. https://academic.oup.com/ajh/article/12/9/934/351716

Berg, M. T., & Lauritsen, J. L. (2016). Telling a similar story twice? NCVS/UCR convergence in serious violent crime rates in rural, suburban, and urban places (1973–2010). *Journal of Quantitative Criminology*, *32*(1), 61-87.

Biron, P., Mongeau, J. G., & Bertrand, D. (1976). Familial aggregation of blood pressure in 558 adopted children. *Canadian Medical Association Journal*, *115*(8), 773-774.

Biron, P., & Mongeau, J. G. (1978). Familial aggregation of blood pressure and its components. *Pediatric Clinics of North America*, *25*(1), 29-33.

Blascovich, J. J., & Katkin, E. S. (1993). *Cardiovascular reactivity to psychological stress & disease* (pp. xv-242). American Psychological Association.

Brackbill, R. M., Siegel, P. Z., & Ackermann, S. P. (1995). Self-reported hypertension among unemployed people in the United States. *BMJ*, *310*(6979), 568.

Chobanian, A. V., Bakris, G. L., Black, H. R., Cushman, W. C., Green, L. A., Izzo, J. L., Jones, D. W., Materson, B. J., Oparil, S., Wright, J. T., & Roccella, E. J. (2003). Seventh report of the Joint National Committee on prevention, detection, evaluation, and treatment of high blood pressure. *Hypertension*, *42*(6), 1206-1252.

Choudhury, A., & Lip, G. Y. H. (2005). Exercise and hypertension. *Journal of Human Hypertension*, *19*(8), 585-587.

Chuang, S. Y., Chiu, T. H., Lee, C. Y., Liu, T. T., Tsao, C. K., Hsiung, C. A., & Chiu, Y. F. (2016). Vegetarian diet reduces the risk of hypertension independent of abdominal obesity and inflammation: A prospective study. *Journal of Hypertension*, *34*(11), 2164-2171.

Cinciripini, P. M. (2021, June 15). *NIOSHTIC-2 Publications Search - 20027354 - Cognitive stress and cardiovascular reactivity: Relationship to hypertension.* Centers for Disease Control and Prevention. https://www.cdc.gov/niosh/nioshtic-2/20027354.html

Dickersin, K., & Chalmers, I. (2011). Recognizing, investigating and dealing with incomplete and biased reporting of clinical research: From Francis Bacon to the WHO. *Journal of the Royal Society of Medicine*, *104*(12), 532-538.

Ehret, G. B., Bakris, G. L., & Forman, J. P. (2017). Genetic factors in the pathogenesis of hypertension. *UpToDate.* https://www. uptodate. com/contents/genetic-factors-in-the-pathogenesis-of-hypertension

Fox, A. J., & Goldblatt, P. O. (1981). *Longitudinal study 1971-1975*. Office of Population Censuses and Surveys, Her Majesty's Stationery Office, London.

Fuchs, F. D., & Whelton, P. K. (2020). High blood pressure and cardiovascular disease. *Hypertension*, *75*(2), 285-292.

Guimarães, G. V., Ciolac, E. G., Carvalho, V. O., D'Avila, V. M., Bortolotto, L. A., & Bocchi, E. A. (2010). Effects of continuous vs. interval exercise training on blood pressure and arterial stiffness in treated hypertension. *Hypertension Research*, *33*(6), 627-632.

Hammarstroem, A. (1994). Health consequences of youth unemployment. *Public Health*, *108*(6), 403-412.

Howell, D. C. (2002). *Statistical methods for psychology* (5th ed.). Thomson Wadsworth.

Huang, C. J., Webb, H. E., Zourdos, M. C., & Acevedo, E. O. (2013). Cardiovascular reactivity, stress, and physical activity. *Frontiers in Physiology*, *4*, 314.

Iversen, L., & Sabroe, S. (1987). Plant closures, unemployment, and health: Danish experiences from the declining ship-building industry. In *Unemployment, social vulnerability, and health in Europe* (pp. 31-47). Springer.

Janlert, U. (1992). Unemployment and blood pressure in Swedish building labourers. *Journal of Internal Medicine*, *231*(3), 241-246.

Kamarck, T. W., & Lovallo, W. R. (2003). Cardiovascular reactivity to psychological challenge: Conceptual and measurement considerations. *Psychosomatic Medicine*, *65*(1), 9-21.

Kupper, N., Willemsen, G., Riese, H., Posthuma, D., Boomsma, D. I., & de Geus, E. J. (2005). Heritability of daytime ambulatory blood pressure in an extended twin design. *Hypertension*, *45*(1), 80-85.

Ladbrook, D. A. (1988). Why are crime rates higher in urban than in rural areas?—Evidence from Japan. *Australian & New Zealand Journal of Criminology*, *21*(2), 81-103.

Law, M. R., Frost, C. D., & Wald, N. J. (1991). By how much does dietary salt reduction lower blood pressure? III--Analysis of data from trials of salt reduction. *British Medical Journal*, *302*(6780), 819-824.

Levy, D., Larson, M. G., Benjamin, E. J., Newton-Cheh, C., Wang, T. J., Hwang, S. J., Vasan, R.S., & Mitchell, G. F. (2007). Framingham Heart Study 100K Project: Genome-wide associations for blood pressure and arterial stiffness. *BMC Medical Genetics*, *8*(1), 1-11.

Liu, X., Gu, W., Li, Z., Lei, H., Li, G., & Huang, W. (2017). Hypertension prevalence, awareness, treatment, control, and associated factors in Southwest China: An update. *Journal of Hypertension*, *35*(3), 637-644.

Lovallo, W. R. (2005). Cardiovascular reactivity: Mechanisms and pathways to cardiovascular disease. *International Journal of Psychophysiology*, *58*(2-3), 119-132.

Lumen Learning. (2021). *Boundless biology*. Lumen. Retrieved October 5, 2021, from https://courses.lumenlearning.com/boundless-biology/chapter/laws-of-inheritance/

Maier, K. J., Waldstein, S. R., & Synowski, S. J. (2003). Relation of cognitive appraisal to cardiovascular reactivity, affect, and task engagement. *Annals of Behavioral Medicine*, *26*(1), 32-41.

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

Manuck, S. B. (1994). Cardiovascular reactivity in cardiovascular disease: “Once more unto the breach.” *International Journal of Behavioral Medicine, 1*(1), 4-31.

Matthews, K. A., Woodall, K. L., & Allen, M. T. (1993). Cardiovascular reactivity to stress predicts future blood pressure status. *Hypertension*, *22*(4), 479-485.

McKusick, V. A. (1960). Genetics and the nature of essential hypertension. *Circulation*, *22*(5), 857-863.

Miall, W. E., & Oldham, P. D. (1963). The hereditary factor in arterial blood-pressure. *British Medical Journal*, *1*(5323), 75-80.

Mills, K. T., Stefanescu, A., & He, J. (2020). The global epidemiology of hypertension. *Nature Reviews Nephrology*, *16*(4), 223-237.

Moll, P. P., Harburg, E., Burns, T. L., Schork, M. A., & Ozgoren, F. (1983). Heredity, stress and blood pressure, a family set approach: The Detroit Project revisited. *Journal of Chronic Diseases*, *36*(4), 317-328.

Mongeau, J. G., Biron, P., & Sing, C. F. (1986). The influence of genetics and household environment upon the variability of normal blood pressure: The Montreal Adoption Survey. *Clinical and Experimental Hypertension. Part A: Theory and Practice*, *8*(4-5), 653-660.

Mongeau, J. G. (1989, September). Heredity and blood pressure. *Seminars in Nephrology*, *9*(3), 208-216.

Morton, N. E., Gulbrandsen, C. L., Rao, D. C., Rhoads, G. G., & Kagan, A. (1980). Determinants of blood pressure in Japanese-American families. *Human Genetics*, *53*(2), 261-266.

National Cancer Institute at the National Institutes of Health. (2021). *NCI dictionary of cancer terms*. National Cancer Institute. Retrieved October 20, 2021, from https://www.cancer.gov/publications/dictionaries/cancer-terms/def/blood-pressure

National Criminal Justice Reference Service. (2017). *2017 NCVRW Urban Rural 508 - NCJRS*. NCJRS.gov. Retrieved January 11, 2022, from https://www.ncjrs.gov/ovc_archives/ncvrw/2017/images/en_artwork/Fact_Sheets/2017NCVRW_UrbanRural_508.pdf

National Heart, Lung, & Blood Institute (NIH). (2021). *Coronary heart disease | Nhlbi, NIH*. Coronary Heart Disease. Retrieved October 27, 2021, from https://www.nhlbi.nih.gov/health-topics/coronary-heart-disease

National Human Genome Research Institute. (2021). *Identical twins*. National Institutes of Health. Retrieved October 6, 2021, from https://www.genome.gov/genetics-glossary/identical-twins

Oldham, P. D., Pickering, G., Roberts, J. F., & Sowry, G. S. C. (1960). The nature of essential hypertension. *The Lancet*, *275*(7134), 1085-1093.

Pandit, A. U., Tang, J. W., Bailey, S. C., Davis, T. C., Bocchini, M. V., Persell, S. D., Federman, A.D., & Wolf, M. S. (2009). Education, literacy, and health: Mediating effects on hypertension knowledge and control. *Patient Education and Counseling*, *75*(3), 381-385.

Pickering, G.W. (1968). *High blood pressure* (2nd ed.). Grune & Stratton Inc.

Pierce, T. W., Grim, R. D., & King, J. S. (2005). Cardiovascular reactivity and family history of hypertension: A meta-analysis. *Psychophysiology*, *42*(1), 125-131.

Praveen, G., Anitha, A., & Ramesh, M. (2016). Negating the negativity: Spotlight on “file drawer effect” in health care research. *Journal of Indian Association of Public Health Dentistry*, *14*(2), 243-243.

Quinney, R. (1966). Structural characteristics, population areas, and crime rates in the United States. *The Journal of Criminal Law, Criminology, and Police Science*, *57*(1), 45-52.

Rice, T., Vogler, G. P., Pérusse, L., Bouchard, C., Rao, D. C., & Williams, R. R. (1989). Cardiovascular risk factors in a French Canadian population: Resolution of genetic and familial environmental effects on blood pressure using twins, adoptees, and extensive information on environmental correlates. *Genetic Epidemiology*, *6*(5), 571-588.

Rosenthal, R. (1979). The file drawer problem and tolerance for null results. *Psychological Bulletin*, *86*(3), 638-641.

Rosenthal, T., & Alter, A. (2012). Occupational stress and hypertension. *Journal of the American Society of Hypertension*, *6*(1), 2-22.

Rotimi, C. N., Cooper, R. S., Cao, G., Ogunbiyi, O., Ladipo, M., Owoaje, E., & Ward, R. (1999). Maximum-likelihood generalized heritability estimate for blood pressure in Nigerian families. *Hypertension*, *33*(3), 874-878.

Schneiderman N., Schwartz, G. E., Raichle, K., Ewart, C. K., Helgeson, V. S., Johnsen, E. L., Cameron, L. D., Lawler, S., Stone, A. A., Broderick, J. E., Cvengros, J. A., Wonderlich, S. A., Freiburger, P., Beer, A., Glanz, K., Christensen, A. J., Diviak, K. R., Sussman, S., Skara, S., Suls, J., et al. (2004) Ss. In A. J. Christensen, R. Martin, & J. M. Smyth (Eds.), *Encyclopedia of health psychology*. Springer. https://doi.org/10.1007/978-0-387-22557-9_19

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

Shi, D., Li, J., Wang, Y., Wang, S., Liu, K., Shi, R., Zhang, Q., & Chen, X. (2017). Association between health literacy and hypertension management in a Chinese community: A retrospective cohort study. *Internal and Emergency Medicine*, *12*(6), 765-776.

Snieder, H., Hayward, C. S., Perks, U., Kelly, R. P., Kelly, P. J., & Spector, T. D. (2000). Heritability of central systolic pressure augmentation: A twin study. *Hypertension, 35*(2), 574-579.

Springer Nature. (2021). *Quantitative trait*. Nature news. Retrieved October 3, 2021, from https://www.nature.com/subjects/quantitative-trait

Spruill, T. M. (2010). Chronic psychosocial stress and hypertension. *Current Hypertension Reports, 12*(1), 10-16.

Sundin, Ö., Öhman, A., Palm, T., & Ström, G. (1995). Cardiovascular reactivity, Type A behavior, and coronary heart disease: Comparisons between myocardial infarction patients and controls during laboratory-induced stress. *Psychophysiology, 32*(1), 28-35.

Treiber, F. A., Kamarck, T., Schneiderman, N., Sheffield, D., Kapuku, G., & Taylor, T. (2003). Cardiovascular reactivity and development of preclinical and clinical disease states. *Psychosomatic Medicine, 65*(1), 46-62.

U.S. Department of Justice. (2015, September 29). *Criminal victimization, 2014*. U.S. Department of Justice, Bureau of Justice Statistics. Retrieved January 29, 2022, from http://cran.r-project.org

U.S. Department of Justice National Criminal Justice Reference Service. (2017). *2017 NCVRW urban and rural victimization - NCJRS*. ncjrs.gov. Retrieved November 10, 2021, from https://www.ncjrs.gov/ovc_archives/ncvrw/2017/images/en_artwork/Fact_Sheets/2017NCVRW_UrbanRural_508.pdf

Vitaliano, P. P., Russo, J., Bailey, S. L., Young, H. M., & McCann, B. S. (1993). Psychosocial factors associated with cardiovascular reactivity in older adults. *Psychosomatic Medicine, 55*(2), 164-177.

Wang, N. Y., Young, J. H., Meoni, L. A., Ford, D. E., Erlinger, T. P., & Klag, M. J. (2008). Blood pressure change and risk of hypertension associated with parental hypertension: The Johns Hopkins Precursors Study. *Archives of Internal Medicine*, *168*(6), 643-648.

Wang, Q., Xu, L., Sun, L., Li, J., Qin, W., Ding, G., Zhang, J., Zhu, J., Xie, S., Yu, Z., & Zhou, C. (2018). Rural–urban difference in blood pressure measurement frequency among elderly with hypertension: A cross-sectional study in Shandong, China. *Journal of Health, Population and Nutrition*, *37*(1), 1-10.

Webster, J. L., Dunford, E. K., Hawkes, C., & Neal, B. C. (2011). Salt reduction initiatives around the world. *Journal of Hypertension*, *29*(6), 1043-1050.

Wolf, M. S., Gazmararian, J. A., & Baker, D. W. (2005). Health literacy and functional health status among older adults. *Archives of Internal Medicine*, *165*(17), 1946-1952.

World Health Organization. (2021, August 25). *Hypertension*. Retrieved January 11, 2022, from https://www.who.int/news-room/fact-sheets/detail/hypertension

Zahid, H., Amin, A., Amin, E., Waheed, S., Asad, A., Faheem, A., Jawaid, A., Afzal, A., Misbah, S., & Majid, K. (2017). Prevalence and predictors of use of home sphygmomanometers among hypertensive patients. *Cureus*, *9*(4).

Zanstra, Y. J., & Johnston, D. W. (2011). Cardiovascular reactivity in real life settings: Measurement, mechanisms and meaning. *Biological Psychology*, *86*(2), 98-105.

Zimmerman, E., & Woolf, S. (2014). *Understanding the relationship between education and health.* Institute of Medicine of The National Accademies. https://nam.edu/wp-content/uploads/2015/06/BPH-UnderstandingTheRelationship1.pdf. Accessed 17 August 2021.