THE EFFECTS OF EXERCISE ON CARDIOVASCULAR REACTIVITY AND RECOVERY TO DAILY HASSLES

THESIS

Presented to the Graduate Council of
Texas State University- San Marcos
in Partial Fulfillment
of the Requirements

for the Degree

Master of ARTS

by

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San Marcos, Texas
May 2009

ACKNOWLEDGEMENTS

I would like to thank my parents, Karen and Larry White, for their continued support of this project and for their encouragement. I am grateful for all they have done for me and for their unwavering confidence in me. I would also like to thank my committee members Alex Nagurney and Marc Turner. I would especially like to thank my committee chair, Reiko Graham. Her assistance on this project was indispensible. My friends and co-workers also deserve acknowledgement for their support and patience during the past two years. I would also like to thank Jovan Wilborn for his support in helping me to "practice what I preach". His help in sustaining my sanity and in taking care of life's nuisances was priceless.

This manuscript was submitted on April 3, 2009.

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ABSTRACT

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Objective: The purpose of this study was to examine the influence of exercise and gender on cardiovascular reactivity, specifically systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate (HR), and recovery to daily hassles. ***Design***: Participants completed three, 10-minute periods: a baseline condition, an interview in which they were asked to describe and discuss a daily hassle, and a recovery period, while SBP, DBP, and HR were recorded. Means for the three measures were calculated for these periods and differences between gender and exercise levels were evaluated while controlling for anger expression and body mass index (BMI). ***Results and***

Conclusion: Those that exercised had a significantly slower SBP and DBP recovery. Additionally, there was a marginally significant interaction between gender and exercise on SBP, DBP and HR reactivity; however, no other effects were found. While exercise was not found to directly interact with cardiovascular reactivity when individuals recounted daily hassles, the interaction of exercise with gender implies some moderating effect of exercise on cardiovascular reactivity; however, further research is necessary. That exercise was significantly related to cardiovascular recovery suggests that exercise can buffer cardiovascular responses to minor stressors, which might reduce the risk for cardiovascular disease.

CHAPTER I

INTRODUCTION

Coronary heart disease and hypertension are major health problems in the United States today. Risk factors such as high blood pressure and being overweight have long been connected to both coronary heart disease and to the future development of hypertension. Both the direct and indirect benefits of exercise have been addressed in numerous studies (eg. Chafin, Christenfeld, & Gerin, 2008; Paffenbarger, Wing, & Hyde, 1995; Taylor & Katomeri, 2006). Exercise has been used to reduce the risk for cardiovascular disease and hypertension through weight loss and improved overall health. Recently, exercise has also been addressed as a buffer of other possible risk factors to cardiovascular disease, such as the influence of stress (Georgiades, Sherwood, Gullette, Babyak, Hinderliter, et al., 2000). Cardiovascular reactivity and recovery to stress, specifically, have been implicated in the development of these cardiovascular diseases. Because of the increasing rates of cardiovascular diseases in America, research on the cardiovascular reactivity and recovery to stress is imperative.

Cardiovascular reactivity refers to the changes in cardiovascular variables, such as heart rate and blood pressure, experienced in response to a stressor. Cardiovascular recovery is measured as the return of cardiovascular levels to baseline after a stressor. As discussed below, it has been well documented that cardiovascular reactivity and recovery are influenced by major stressors (Ottaviani, Shapiro, Davydov, & Goldstein, 2008;

Powch & Houston, 1996; Schwerdtfeger, Konermann, & Schonhofen, 2008), however, little research has been done to establish the same relationship between cardiovascular reactivity and recovery to daily hassles. Similarly, there has been support of the notion that exercise can act as a buffer to cardiovascular reactivity and recovery to major stress (Chafin, Christenfeld, & Gerin, 2008; Forcier, Stroud, Papandonatos, Hitsman, Reiches, Krishnamoorthy, et al., 2006; Georgiades et al., 2008; Ledwidge, 1980), but again, there is limited research linking any protective effects of exercise to cardiovascular reactivity and recovery to daily hassles. Because of the lack of research in cardiovascular reactivity and recovery in response to daily hassles, the present study will attempt to extend the finding that exercise can buffer the negative effects of major stressors to all types of stress.

Types of Stress

Stressors play a major part in everyday life. Two types of stressors have been identified as detrimental to both physical and mental health: major stressors and daily hassles. Major stressors consist of important life events that cause significant distress, such as a death in the family or a job change (Blankstein & Flett, 1992). Major stressors generally occur much more infrequently than daily hassles and tend to be more severe in nature. Daily hassles have been defined by Kanner, Coyne, Schaefer, & Lazarus (1981) to be those demands that characterize everyday dealings within one's life that are irritating, frustrating, and distressing (Blankstein & Fleet, 1992; Iwasaki, 2001). Blankstein & Flett (1992) characterized daily hassles to be stressors that occur with some frequency within a one month interval. These include such things as arguments, lost articles and poor weather. Both major stressors and minor stressors may result in psychological and physical consequences, although the majority of the research related to cardiovascular reactivity and recovery has focused on major stressors.

Given that research has shown that cardiovascular reactivity and recovery are related to major stressors, it is likely that daily hassles also elicit cardiovascular reactivity and recovery. Kanner et al. (1981) originally introduced the idea that major stressors may change the way that daily hassles are interpreted or even what daily hassles may be presented in life. By measuring major events such as a parents' divorce or the death of a relative, daily stressors such as waiting in line, and pleasurable events, such as listening to music, Wagner, Compas, & Howell (1988) supported this notion by showing that major events or stressors tended to produce an increase in daily stress, which in turn produced an increase in psychological symptoms of stress. However, daily hassles have resoundingly been found to be a better predictor of psychological and physical symptoms, such as overall health status, somatic symptoms and energy levels, than are major stressors (DeLongis et al., 1982; Holahan et al., 1984; Kanner et al., 1981; Monroe, 1983; Oppenheimer & Prinz, 1985, as cited in Wagner, Compas, & Howell, 1988). That daily hassles are generally more influential to psychological and physical symptoms further supports the idea that daily hassles are possibly more important for cardiovascular health than major stressors. Therefore, an understanding of how minor daily hassles affect cardiovascular variables such as blood pressure and heart rate is necessary.

Physical symptoms of stress

Stress will generally increase heart rate, blood pressure, and muscle tension. Stressors will initially activate the sympathetic arm of the autonomic nervous system, which triggers activity in the hypothalamic-pituitary-adrenal (HPA) axis (Kolb &

Whishaw, 2005). The HPA axis has been identified as a mechanism by which the biological response to stress may influence health. The HPA axis is a hormonal response system that produces stress hormones such as cortisol when we are stressed (Kolb & Whishaw, 2005). Major stressors have been linked to an increase in cardiovascular and HPA axis increases (Chrousos & Gold, 1992, as cited in Forcier et al., 2006; McEwen, 1998; Traustadottir, Bosch, & Matt, 2003). There is even evidence that the HPA cortical and hypothalamic-pituitary-gonadal (HPG) axes interact during stress (Granberry et al., 1990; Kant et al., 1983, as cited in Dishman et al., 2000). This interaction will be revisited subsequently in the context of gender differences in the manifestation of stress response. As the HPA axis and, subsequently cortisol levels, contribute to the body's homeostatic state and are also implicated in the parasympathetic influence of the heart, the regulation of these systems have an important contribution to the overall health of a person (Chida & Hamer, 2008; Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology, 1996).

Elevated cortisol levels have been strongly implicated in the involvement of the HPA axis with stress. Cortisol is released when corticotrophin-releasing hormones (CRH) released from the hypothalamus signal the release of adrenocorticotropin hormones (ACTH, Kolb & Whishaw, 2005). CRH is released when either a physical or psychological stressor signals the neurons in the paraventricular nucleus of the hypothalamus (Miller, Chen, & Zhou, 2007). CRH then travels through the hypophyseal portal circulation to the anterior pituitary gland to signal ACTH. ACTH in turn travels through the peripheral circulation to the adrenal glands, which release cortisol.

Deactivation of the HPA system occurs when cortisol binds to hypothalamic receptors (Kolb & Whishaw, 2005).

Cortisol is a key factor in the regulation of glucose storage and its utilization. The link between cortisol and stress may also be a factor in the increase in glucose availability during a stressful situation (Miller, Chen, & Zhou, 2007). Stress has been found to be linked to an increase in glucose availability, which helps to meet the metabolic needs of the physical response to stress. It has also been suggested that elevated cortisol levels may be a risk factor of higher levels of systolic blood pressure (Schwerdtfeger, Konermann, & Schonhofen, 2008, as cited in Ahmed et al., 2004). An increased risk to coronary heart disease, diabetes, and hypertension have all been related to continuous elevated cortisol levels and hyper-reactivity to the HPA-axis (Traustadottir, Bosch, & Matt, 2003), suggesting that a reduction in cortisol levels and reactivity to the HPA-axis may lead to a decreased risk to these disorders.

Stress and cardiovascular reactivity

Evidence has shown standard physical risk factors, such as unhealthy eating and a sedentary lifestyle, are not always able to explain cases of chronic heart disease in a number of different cases (Kannel & Schatzkin, 1983, as cited in Powch & Houston, 1996). Because of this, other more psychological factors have been explored to help explain these cases. Stress, for example, has been shown to be related to cardiovascular diseases. In fact, those who show an inflated cardiovascular response to stress are at risk for cardiovascular disease and hypertension (Krantz & Manuk, 1984, 1986, as cited in Chafin, Christenfeld & Gerin, 2008; Lovallo & Wilson, 1992). One common risk factor for myocardial ischemia, coronary heart disease, and the future development of hypertension is a larger than normal cardiovascular response to mental stress (Matthews et al., 1993; Wood et al., 1984, as cited in Georgiades et al., 2000). Cardiovascular reactivity to stress has been suggested through many studies to be an independent risk factor for hypertension, and can even influence hypertension development up to 45 years later (Chaney & Eyman, 1988; Dlin, Hanne, Silverbert, & Bar-Or, 1983; Treiber et al., 2003; Wilson & Meyer, 1981, as cited in Stewart, Janicki, & Kamarck, 2006). Folkow (1990, as cited in Stewart, Janicki, & Kamarck, 2006) and Obrist (1981, as cited in Stewart, Janicki, & Kamarck, 2006) suggest that a continued exaggerated cardiovascular response to stress may be a contributing factor to long term hypertension, possibly because of sustained or repeating high blood pressure or by impairing endothelium dependent vasodilation.

The literature relating to cardiovascular reactivity to stress overlooks the effects of minor stressors such as daily hassles. Because daily hassles incorporate a significant portion of stress throughout the life, it is important to study how cardiovascular reactivity to daily hassles may manifest itself. As major stressors and minor stressors share similarities in the discernible physical and psychological symptoms (Kanner et al., 1981; Wagner, Compas, & Howell, 1988), it is likely that cardiovascular reactivity will show a similar response to daily hassles as to major stressors.

Stress and cardiovascular recovery

Cardiovascular recovery is measured as the time it takes a participant's cardiovascular variables like heart rate and blood pressure to return to baseline levels from an aroused cardiovascular state after the induction of stress or the level of elevation in cardiovascular variables that remains during the recovery period (Linden, Earle, Gerin,

& Christenfeld, 1997, as cited in Stewart, Janicki, & Kamarack, 2006). For example, heart rate recovery is estimated by the period of time after a stressful event that it takes for one's heart rate to return to baseline pre-stress levels. Recovery of the cardiovascular system from a stressful encounter is also a mechanism that has been deemed as noteworthy relative to both physical as well as psychological health. Cardiovascular recovery from stress may even have equal, if not greater, importance to cardiovascular health than cardiovascular reactivity (Glynn, Christenfeld, & Gerin, 2002; Linden, Earle, Gerin, & Christenfeld, 1997; McEwen, 1998; Schwartz et al., 2003, as cited in Forcier et al., 2006). Slow recovery may be indicative of poor coping skills, which can prolong the experience of stress (Cameron & Meichenbaum, 1982, as cited in Jamieson, Flood, & Lavoie, 1994). Prolonged stress can lead to poor health because of the extended amount of time that the body is in an aroused state (Johannson & Frankenhaeuser, 1973, as cited in Jamieson, Flood, & Lavoie, 1994). Compared to those with a normal recovery time, those that show a slower recovery from an elevated cardiovascular response to stress may have an enlarged risk to cardiovascular disease (Borghi et al., 1986; Gerin & Pickering, 1995; Haynes et al., 1991; Steptoe & Marmot, 2005, as cited in Chafin, Christenfeld & Gerin, 2008).

Again, the majority of the literature relating to cardiovascular recovery to stress has been conducted on major stressors. Slow cardiovascular recovery to major stressors has been linked to poor cardiovascular health, possibly because of the length of time the body is in an aroused state. Because daily hassles are generally more frequent in nature, thereby causing a longer state of arousal, daily hassles may be linked to cardiovascular recovery in a similar manner as major stressors. This would be especially true when there is slow recovery to a stressor as the body would have less of a chance to return to a normal state of cardiovascular operation.

Personality factors and cardiovascular health

As mentioned previously, psychological variables have been repeatedly shown to be related to cardiovascular disease. "Type A" behavior has been suggested to be related to cardiovascular disease; however, studies have shown conflicting results, possibly due to research limitations (i.e. Allred & Smith, 1991; Barefoot, Dahlstrom, & Williams, 1983; Christensen & Smith, 1993; Hardy & Smith, 1988). Hostility, a major attribute of Type A behavior, has been more consistently linked to cardiovascular disease (Barefoot et al., 1989; Rozanski, Blumenthal, & Kaplan, 1999). Hostility related variables, such as cynicism, mistrust and a disposition for hostile behaviors, specifically, have been found to be related to cardiovascular reactivity (Smith, 1992, as cited in Powch & Houston, 1996). More recently, anger has also been identified as a variable related to Type A behavior that may potentially be correlated to cardiovascular disease (Seigman, 1994). Anger expression and hostility have both been two of the most widely studied variables related to cardiovascular disease. Most studies confirm a positive correlation between those who exhibit anger and hostility to an increased risk of cardiovascular disease (Gallo & Matthews, 2003; Kop, 1999; Krantz & McCeney, 2002; Rozanski, Blumenthal, & Kaplan, 1999; Smith & Ruiz, 2002, as cited in Smith, et al., 2004).

Although hostility and anger are sometimes presented as one entity because of the similarities they share, they are, in fact, two different traits. Hostility has been defined as a "negative attitude towards others, consisting of enmity, denigration, and ill will" (Smith, 1994, p. 26). Anger, on the other hand, has been defined as "an unpleasant emotion ranging in intensity from irritation or annoyance to fury or rage (Smith, 1994, p. 25). Both of these constructs involve a tendency to use aggressive behavior to release frustration and are highly correlated, but should certainly be viewed as different entities. Hostility tends to be a more chronic characteristic while anger tends to be short-lived, although generally reoccurring. By treating hostility and anger as separate mechanisms, personality variables can be more deeply evaluated and isolated.

One model relating hostility to cardiovascular disease suggests that a person scoring high in hostility may respond to stressors with a more prominent increase in blood pressure and neuroendocrine hormones such as cortisol. As mentioned previously, continuous repetition of this increase due to repeated stress can lead to cardiovascular disease and hypertension (Christensen & Smith, 1993). That hostility is related to cardiovascular disease has been well documented (Booth-Kewley & Friedman, 1987; Miller et al., 1996; Rozanski, Blumenthal, & Kaplan, 1999, as cited in Prkachin & Silverman, 2002; Vella, Kamarck, & Shiffman, 2008). Vella, Kamarck, & Shiffman (2008) supported the finding that heart rate and blood pressure tend to increase in those rated high in hostility during a stressful event. Christensen & Smith (1993) showed that hostile individuals have significantly higher blood pressure during self-disclosure of troubling events compared to those who score lower on hostility measures. Specifically, those who show cynical hostility experience a higher level of interpersonal stress and show greater systolic blood pressure reactivity in situations of high stress than do those who do not show high levels of cynical hostility (Kamarck, Manuck & Jennings, 1990; Powch & Houston, 1996; Wright, Contrada, Glass, 1985, as cited in Kamarck Manuck & Jennings, 1990). Additonally, hostility has been found to be correlated with an increase in unhealthy behaviors (Lipkus et al., 1994; Leiker & Hailey, 1988, as cited in Prkachin & Silverman, 2002' Scherwitz et al., 1992), further exacerbating the chances for disease and early death. That "Type A" personality characteristics and hostility itself have been more consistently associated with an increased SBP reactivity than that of DBP, it is important to investigate these variables separately.

Hostility has also been found to be related to slower cardiovascular recovery. Neumann, et al. (2004) suggest that the relationship between hostility and cardiovascular recovery could be due to an extended sympathetic activation and a declined ability for vagal control of the heart. When specific methods were used to enhance recovery, such as distraction, blood pressure recovery was enhanced (Neumann, et al., 2004; Patel, 1975; A. Schwartz et al., 2000, as cited in Neumann, et al., 2004), suggesting that future research of alternative treatments to stress may be successful at reducing the detriments seen by prolonged cardiovascular recovery to stress.

Anger expression, also, has been continually linked to cardiovascular disease (Prkachin, Mills, Zwaal & Husted, 2001). The measure of anger expression differs from other measures of anger as it directly targets those who outwardly express their anger in an enraged manner. By using an interview designed to have participants "re-live" an angry experience, Prkachin et al. (2001) was able to demonstrate that systolic blood pressure (SBP), diastolic blood pressure (DBP), and heart rate were higher during the interview than at baseline, and that the anger interview provoked a raise in these cardiovascular responses for a longer duration than other stressors, such as physical or mental tasks. This suggests that reliving an angry emotion can potentially be more influential in cardiovascular disease than other emotions or stressors, like mental arithmetic. Typically, stressors used in experiments, such as math tasks, mirror image tracing tasks, or cold pressor tasks, are do not directly relate to real life experiences. For this reason, an interview was used to elicit the same feelings and reactions experienced during a minor stressor. Prkachin et al. (2001) evaluated their anger interview against a mental arithmetic task and a dynamic handgrip task to assess the validity of the interview. This study supported previous conclusions (e.g., Anderson & Lawler, 1995; Ironson et al., 1992; Lamensdorf & Linden, 1992) that men and women sustain a relatively large blood pressure change in response to recalling and discussing an experience of anger or a stressor. Because of these results, the assumption is made that an interview of daily hassles or minor stressors can be used to illicit similar responses to those experienced when the daily hassle occurs.

Delayed cardiovascular recovery can be related to anger expression as well. Suchday et al. (2004) found that resentful and reflective anger are related to delayed cardiovascular recovery to provocation. During frustrating role play tasks, a positive correlation was found between these types of anger and a delayed cardiovascular recovery. Similarly, both suppressed and expressed anger have also been found to be correlated with a prolonged cardiovascular recovery to stress or provocation (Brosschot & Thayer, 1998, as cited in Suchday et al., 2004; Faber & Burns, 1996; Suarez & Williams, 1990).

The influence of hostility and anger has been well documented relative to cardiovascular reactivity and recovery. The implication of this influence is that those who display high levels of anger and/or hostility may be at a higher risk for cardiovascular disease and hypertension. Because of this influence, when studying the relationship between exercise and cardiovascular reactivity and recovery, it is important to control for these variables.

Gender influences

It is possible that cardiovascular reactivity and recovery to stress may be a factor in the elevated risk of CVD in males (Stoney et al., 1987, as cited in Traustadottir, Bosch, & Matt, 2003). Males have consistently shown a higher rate of cardiovascular disease than do females. Age-adjusted rates for hypertension have shown males to be 1½ times more likely to have hypertension than females (Harlan, 1987, as cited in Girdler, Pedersen, Stern & Light, 1993). Because of this inequality of gender, it is important to examine the effects of exercise, not just overall but also while isolating gender. Possibly, men would sustain greater protective effects of exercise than women as these statistics imply a greater potential for a reduction in cardiovascular disease. Because of this possibility, it is important to assess the moderating effects of exercise for both men and women separately.

Potentially, female hormones offer a protective effect to cardiovascular diseases such as hypertension, specifically for stress induced cardiovascular reactivity. Differing reproductive hormone levels in women during laboratory investigations have found a correlation in determining stress response (Davis & Matthews, 1990; Saab, Matthews, Stoney, & McDonald, 1989, as cited in Matthews et al., 1991). Several studies have since examined cardiovascular reactivity relative to women's menstrual phase; for the follicular phase, which is 0-2 weeks after the last period, and for the leutal phase, which is 2-4 weeks after the last period. Findings from studies examining this possibility have been very inconsistent; however. Some indicate that women have a greater cardiovascular reactivity during the luteal phase (e.g., Hastrup & Light, 1984), some indicate that women have a greater cardiovascular reactivity during the follicular phase (i.e. Polefrone & Manuck, 1988), and some indicate no differences at all (i.e. Collins, Eneroth, & Landgren, 1985; Weidner & Helmig, 1990). This suggests a need to explore the relationship between gender and cardiovascular reactivity and recovery to stress, including the mechanisms that may influence cardiovascular reactivity and recovery to stress.

Women have been shown to exhibit larger ACTH and cortisol responses to stress compared to men (Born et al., 1995; Greenspan et al., 1993; Heuser et al., 1994; Luisi et al., 1998, as cited in Traustadottir, Bosch, & Matt, 2003). Female rats tend to show higher levels of HPA cortical activity relative to male rats. Additionally, estrogen and progesterone levels may differently influence the regulation of ACTH responses during different stages of estrous in normally cycling rats and humans (Loucks et al., 1989; Vuau & Meaney, 1991, as cited in Dishman et al., 2000). Findings that cortisol and ACTH are essential in the physical process of reacting to stress as well as the link between raised cortisol levels and cardiovascular disease indicate that women may be more susceptible to an elevated physical response to stress.

Most of these data come from a response to physical stressors, such as the immobilization of rats or a novel foot shock. The data on psychological stressors, however, tend to be more conflicting. For example, Kirschbaum et al. (1992; 1998, as cited in Traustadottir, Bosch, & Matt, 2003) and Earle et al. (1999, as cited in Traustadottir, Bosch, & Matt, 2003) contend that men tend to show a greater cortisol response during a stressful situation. When comparing a Stroop word task and a mirror trace task that were randomly described as either masculine or feminine, Matthews et al. (1991) showed that men had higher systolic blood pressure levels, regardless of the task. Others, however, challenge that this would depend on the stressor. Frankenhaeuser (1983, as cited in Davis & Matthews, 1996) alleged that an actual gender matched challenge, such as matching males to achievement demands, would yield an increased reactivity for that specific gender. Davis and Matthews (1996) tested this hypothesis by using having participants either empathize with (the feminine condition) or try to persuade (the masculine condition) a confederate when discussing corporal punishment. They found that participants with high instrumentality, which is a masculine trait, exhibited a similar SBP increase to the persuasion and empathy tasks, while those high in expressiveness, the feminine trait, tended to show a larger SBP increase to the persuasion task, rather than the empathy task. This indicates that just labeling a task as masculine or feminine is not effective in creating a reliable gender difference; however, actual tasks attributed to gender competency may influence reactivity levels, specifically in those who express typically feminine traits. Because only an increase in SBP was found, it is important to separate analysis of SBP and DBP to determine if they may react differently by gender.

Further contributing to the variability in cardiovascular response to stress by gender, Seeman et al. (1995; 2001, as cited in Traustadottir, Bosch, & Matt, 2003), indicate that women show an increased cortisol response relative to men during a driving simulation task. Additionally, women tend to show an extended recovery from a challenge or stressor compared to men (Born et al., 1995; Deuschle et al., 1997; Heuser et al., 1994; Wilkinson et al., 1997, as cited in Traustadottir, Bosch, & Matt, 2003).

Because most studies have indicated that men exhibit a larger cardiovascular response, more research is needed. Further research on cardiovascular reactivity and recovery to daily hassles will help establish gender trends, as there is currently little research showing gender differences within cardiovascular reactivity and cardiovascular recovery in the context of daily hassles.

When psychological stressors are used, men tend to show higher reactivity to acute stress and chronic stressors (Girdler, Hinderliter, & Light, 1990; Stoney, Matthews, McDonald, & Johnson, 1988, as cited in Matthews, Gump, & Owens, 2001), although women are generally exposed to more stress, especially relationship stress (Davis, Matthews, & Twamley, 1999, as cited in Matthews, Gump, & Owens, 2001). Matthews, Gump, and Owens (2001), however, found that men had higher levels of systolic blood pressure and cortisol than did women, regardless of whether the type of stress was chronic or not. Men were also found to have prolonged recovery to stress in their systolic blood pressure and epinephrine levels, even when covariates were adjusted for. Though it has been shown that males tend to show a more prolonged recovery to stress than females, it has also been documented that females tend to show a slower recovery to stressors than males (Fredrickson, Levenson, & Cartensen, 1992, as cited in Jamieson, Flood, & Lavoie, 1994). These conflicting results suggest that further research on the cardiovascular response to stressors needs to be conducted.

Exercise as a buffer

Given the array of mechanisms by which cardiovascular disease can be influenced, it is important to identify a common defense or treatment for this increasingly devastating disease. As we have heard in recent years, exercise is crucial to your physical health as it reduces chances for cardiovascular disease, improves lung capacity, and provides many other physical benefits. Endurance training, by using aerobic exercise, has been found to reduce the biological response to stress. Those who are more active tend to have a lower cardiovascular response to stress compared to those who are not active (Crews & Landers, 1987, as cited in Georgiades et al., 2000).

Stress will generally increase heart rate and tense muscles. It is therefore a logical extension that exercise would reduce stress because those who are aerobically fit will sustain a lower heart rate, less muscular intensity, slower respiration and less accumulation of lactate (Ledwidge, 1980). Because those that exercise tend to incur a lower rate for these physical responses to stress, the physical symptoms of stress will not be as destructive in those with a high level of fitness.

Exercise can be very effective in reducing hypertension. Several studies have shown that exercise and physical activity have been found to be inversely related to blood pressure (Criqui et al., 1982; Hickey et al., 1975; Miall, 1958; deVries, 1980; Jennings et al., 1986, as cited in Blumenthal et al., 2008). Similarly, other studies have shown that those who are more active tend to have a lower cardiovascular response to psychosocial stressors compared to those who are not active (Crews & Landers, 1987, as cited in Georgiades et al., 2000). Therefore, exercise could be of particular importance when considering individual differences in cardiovascular stress responses For example, Georgiades et al. (2000) were able to show that in mild to moderately obese individuals with high blood pressure, exercise training is effective in lowering blood pressure, both during rest and during mental stress.

A number of studies have shown that those who are not physically fit will tend to take longer to recover from a stressful event, especially with regard to heart rate (Blumenthal et al., 1988; Cox, Evans, & Jamieson, 1979; Hollander & Seraganian, 1984; Sherwood, Light, & Blumenthal, 1989; Sinyor et al., 1983, as cited in Jamieson, Flood, & Lavoie, 1994). In a study assessing the relationship between stress recovery and fitness level, Jamieson et al. (1994) found that heart rate recovery during the first three minutes after the task was significantly quicker in individuals with a higher self-reported fitness level, an increased activity at sports, and a lower BMI, among other fitness-related measures. Additionally, after familiar treadmill running, norepinephrine levels were relatively protected during a novel stressor in rats, indicating that routine exercise may have a protective effect of both physical and psychological stressors (Dishman et al., 2000). Given delayed cardiovascular recovery is a risk factor for coronary heart disease (Linden et al., 1997, as cited in Suchday et al., 2004), introducing exercise to those with a prolonged cardiovascular recovery to stress or hostility may allow for lessened impact to the cardiovascular system.

The buffering effects of exercise have been shown to be effective in reducing the risk for coronary heart disease, diabetes, and hypertension. While exercise has been shown to reduce hypertension and coronary heart disease with contributions of weight loss and improved insulin sensitivity (Blumenthal et al., 2000), exercise also positively contributes to other sources of cardiovascular diseases. The influence of exercise on improved cardiovascular recovery and a decreased cardiovascular response to major stressors contributes to the efforts to relieve cardiovascular disease. Exercise has been shown to effectively reduce both the physical and psychological responses to stress

(Broman-Fulks et al., 2003; Criqui et al., 1982; Hickey et al., 1975; Miall, 1958; deVries, 1980; Jennings et al., 1986, as cited in Blumenthal et al., 2008). Because most research done regarding how exercise moderates the response to stress is done on major stressors, it is clear that further research needs to be performed on how exercise may affect the response to daily hassles.

Study Rationale & Hypothesis

Elevated levels of cardiovascular reactivity and recovery to stress are risk factors for the future development of cardiovascular disease. Exercise has been established as a way to decrease the effects of major stressors on cardiovascular reactivity and recovery. Literature on how exercise contributes to the effects of daily hassles on cardiovascular reactivity and recovery is limited, however. Because the literature suggests that daily hassles are a better predictor of psychological and physical symptoms than are major stressors, it is important to research the effects of exercise on cardiovascular reactivity and recovery to daily hassles. The present study attempts to do such by measuring the differences in cardiovascular reactivity and recovery to daily hassles between those who engage in regular exercise and those who lead a more sedentary lifestyle. To do this, heart rate and blood pressure were continuously measured during an interview used to describe a pet peeve. Heart rate and blood pressure were also be measured during a 10 minute period to evaluate how close to baseline these variables return.

The interview, an adaptation of the Prkachin interview (Prkachin, Mills, Zwaal, & Husted, 2001), required that the participants to describe a pet peeve or stress that is incurred on a daily basis. Once the participant finished describing the daily hassle, the interviewer probed the participant to further describe how that specific minor stressor makes them feel and what emotions are elicited because of the stressor. This task was designed to bring forth similar feelings that are experienced by the patient when the minor stressor is occurring, thereby simulating the cardiovascular response of the participant during the time of the stressor.

The first trend expected is an increase in blood pressure and heart rate during the daily hassles interview. Those who engage in regular exercise are expected to show a lesser increase in BP and HR than those who do not regularly exercise, because those who regularly exercise generally begin with lower blood pressure and heart rate levels and have shown a smaller increase in cardiovascular levels to major stressors than those who do not regularly exercise. It is also expected that those who exercise will show a greater decrease in BP and HR during the recovery time following the interview. These results are expected because of the literature indicating a quicker return to baseline cardiovascular activity in response to major stressors in those who engage in regular exercise compared to those who do not (see Figure 1).

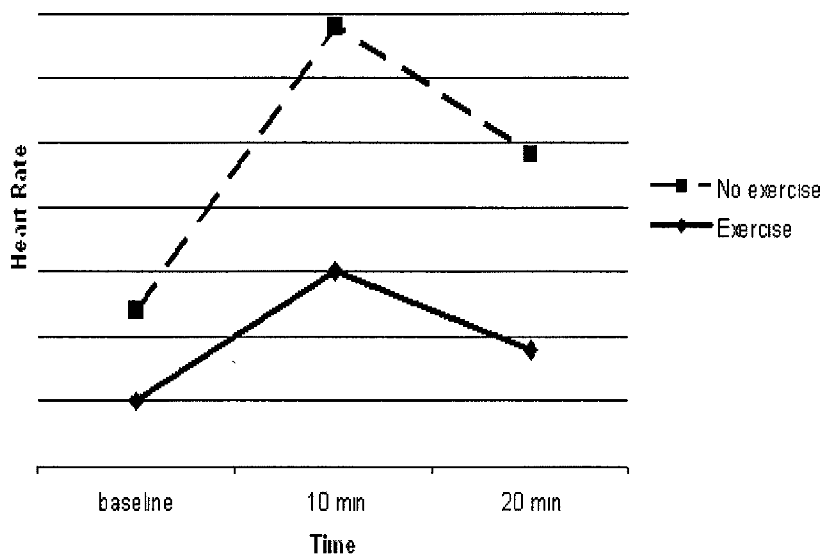

Figure 1. Expected trend for heart rate reactivity and recovery in this experiment

Although the data on how gender influences cardiovascular reactivity and recovery are inconsistent, it is likely that women will show higher reactivity and slower recovery to daily hassles. This trend is expected for two reasons. Kopper and Epperson (1991, as cited in Kopper, 1993) found that those with a more masculine role tended to have higher levels of anger and were less likely to control their anger. In a follow up study, Kopper (1993) found that women were more prone to indirect hostility, whereas men tended to display higher levels of aggressiveness. While men tend to experience more anger, which would tend to lead to a higher cardiovascular reactivity, this study will covary for anger, thereby eliminating any differences due to anger. Additionally, women tend to experience more daily hassles than men. This will probably create a greater cardiovascular reactivity and prolonged response when discussing a pet peeve or daily hassle due to the cumulative effects.

CHAPTER II

METHOD

Participants

The sample included 59 students (24 men, 35 women), 17-41 years of age (*M* = 21.44 years, *SD* = 4.74), from Psychology classes at Texas State University-San Marcos. Participants were asked if they were taking any medication that may interfere with cardiovascular readings, such as antihistamines and decongestants (*N* = 0). In addition, those participants with a history of cardiovascular disease or psychological disorders were excluded (*N* = 0). Extra credit was offered to the students in exchange for participation in the study. If students chose to not participate in the study, other means of obtaining extra credit were offered.

Informed consent was obtained before participation in the procedure and an opportunity to obtain the study results were provided upon completion of the study. The Texas State Institutional Review Board approved the procedures for this study. Participants were asked to not participate in strenuous exercise for two hours before participating in this study, as it has been found that post-exercise hypertension can persist several hours after exercise, which can lead to a reduction in blood pressure even in the presence of stress (Chafin, Christenfeld, & Gerin, 2008, as cited in Brownley et al., 1996). For female participants, information about the date of their last menstrual period and birth control were also obtained.

Apparatus

The BIOPAC MP150 workstation was used to gather electrophysiological data, running Acknowledge V.3.8.1 (BIOPAC, 2006). All data were recorded at a sampling rate of 200 Hz and was notch-filtered for line noise at 60 Hz. Heart rate was evaluated with the PPG100C pulse photoplethysmogram amplifier set at a gain of 100 and a finger cuff with a small photocell. The photocell was placed on the middle finger of the nondominant hand. Heart rate was calculated online from the PPG 110C signal into beats per minute.

Blood pressure was monitored continuously with the NIBP100A continuous blood pressure meter connected to a DA100C amplifier and a pneumatic wrist cuff containing the BP sensor. The sensor was placed at the distal end of the radius on the participant's non-dominant wrist over the radial artery. Readings were taken every 14 seconds. Systolic and diastolic blood pressure were then calculated online separately for analysis.

Questionnaires

The Habitual Physical Activity Level Scale: This is a 16 item scale with questions rated on a five point scale ranging from never to always. This scale assesses physical activity for physical activity at work, for sport and during leisure time not including sport. Test-retest reliability over three months has been rated at .88, .81 and .74 (Jamieson & Flood, 1994, as cited in Baecke, Burema, & Frijters, 1982).

The State-Trait Anger Expression Inventory (STAXI): This is a 10 item inventory that evaluates a subject's tendency to convey and feel anger without a specific cause by using a four point scale, with 1 being not at all and 4 being very much so. The STAXI is separated into three anger expression trait scales, a State-Anger scale, and a Trait-Anger scale. Internal consistency measures yielded alpha levels of at least .84 or higher for all subscales (Spielberger, 1988). For this particular study, internal consistency measures yielded a Chronbach's alpha of .64.

Interview

A variation of the Prkachin Anger Interview was used (Prkachin et al., 2001). The Prkachin Anger Interview showed significant differences between the interview and baseline periods as well as between the interview and recovery periods, indicating this is a valid measure by which to evaluate change in cardiovascular measures (all $p < .001$). This variation of the interview was revised to assess pet peeves/daily hassles instead of anger. During this interview, participants were asked to speak about a daily hassle generally experienced or a pet peeve of the participant. If 10 minutes was filled by the participant's description, the interviewer prompted the participant for further details and feelings elicited by this daily hassle. With prompting, all participants were able to fill the entire 10 minute period with discussion of a pet peeve. (See appendix)

Procedure

Upon arrival, informed consent was obtained and the participants were weighed and their height was measured. Female participants were also asked for the date of their last period, to analyze possible effects due to menstrual phase. Participants then completed the questionnaires, which lasted approximately 15 minutes. The questionnaires were always completed before the interview to allow the participants to familiarize themselves with the lab environment and to allow any elevated cardiovascular variables to return to baseline values. Participants were then fit with the blood pressure and heart rate recording apparatus and an initial baseline reading of heart rate and blood pressure was recorded for a total of 10 minutes. During the interview portion of the study, the participants were asked to describe a pet peeve or a daily hassle and the way it made them feel for 10 minutes. If the participant was not able complete 10 minutes of discussion, the interviewer then probed the participant using a standard format set by the Prkachin Anger Interview (see appendix). After the interview was complete, a 10 minute period of silence was utilized to record the participant's cardiovascular recovery time.

Data Analysis

A participant's data was omitted from analyses if, due to recording problems, at least 30% of the reading was not complete in baseline, interview, or recovery sessions (females removed, $N = 7$, males removed, $N = 3$). Total number of participants after removal was 49 (21 males, 28 females).

Means for HR, DBP and SBP were taken initially over two 5 minute periods, twice during the baseline period, during the interview, and for recovery, to allow a direct comparison between those who exercise and those who do not on average cardiovascular measures at each stage. Initial inspection of these 5 minute periods indicated that the data tended to show elevated cardiovascular levels at the beginning of the experiment. Therefore, the first five minutes of the baseline period was dropped for all participants and only the last five minutes of that 10 minute period were used as a baseline estimate. Means for the baseline (T1) were subtracted from means for the interview (T2) to achieve reactivity scores for heart rate, and systolic and diastolic blood pressure. Similarly, means for the recovery period (T3) were subtracted from the interview (T2) to achieve the recovery scores.

A median split (Mdn = 2.5) was used to divide the participants into two groups, an exercise group ($M = 3.3304$, $N = 25$) and a non-exercise group ($M = 1.7732$, $N = 24$). Cell means for the two exercise groups and other summary statistics are shown in Table 1.

Table 1: Descriptive information by exercise and gender

Data	**High Exercise Males**	**High Exercise Females**	**Low Exercise Males**	**Low Exercise Females**
Mean	3.31	3.35	1.76	1.78
N	13	12	9	16
Median	2.94	3.09	1.75	1.85
Std. Deviation	1.09	1.06	1.53	1.18
Mean BMI	23.3	24.1	26.4	26.8

To determine anger expression, the AX/Out (anger expression out) subscale of the STAXI was used. AX/Out measures "how often angry feelings are expressed with verbally or physically aggressive behavior" (Spielberger, 1999, pg. 2). AX/Out was found to highly correlate with SBP, DBP and HR reactivity for college males who were provoked during a subtraction task (Spielberger, 1999). Additionally, among middle-aged Finnish men, AX/Out was found to be significantly correlated with carotid artery atherosclerosis; those who scored above a median split showed an increased level of this disorder (Julkunen et al., 1994, as cited in Spielberger, 1999). Because of the past use of anger expression to measure cardiovascular reactivity and the correlation with cardiovascular disease, anger expression was used in this study to establish the level of anger for participants. Because there is a reliable amount of evidence suggesting anger expression to be related to cardiovascular reactivity and cardiovascular disease, it was not the intention within this study to replicate this finding. Instead, anger expression was covaried for within the analysis so that any effects found will be due to differences in exercise levels rather than anger expression.

Table 2: Descriptive information by anger level and gender

Data	High Anger Males	High Anger Females	Low Anger Males	Low Anger Females
Mean	19	19.15	13.6	12.88
N	7	14	14	14
Median	19	19	14	13
Std. Deviation	1.51	1.63	1.825	1.13

CHAPTER III

RESULTS

Preliminary analyses

Preliminary analyses were conducted to determine if there were baseline differences for the exercise/no exercise groups or genders. To this end, three univariate analyses of covariance (ANCOVA) were conducted with HR, SBP, and DBP as the dependent variables, exercise (low vs. high levels) and gender (male vs. female) as between subjects variables, and anger expression and BMI as covariates. Separate univariate ANCOVAs were used to compare any differences between the cardiovascular variables because of previous findings that SBP may be a more sensitive variable with certain groups. Separate analyses were used because of the evidence that each of these variables manifest differently relative to cardiovascular disease. A higher risk for hypertension and other cardiovascular diseases such as myocardial infarction and heart failure is related to an increased systolic blood pressure but is actually inversely related to diastolic blood pressure (Izzo, Levy, & Black, 2000). Differences in diastolic blood pressure and systolic blood pressure may be due to ventricular ejection interacting with the viscoelastic properties of the large arteries as well as possible differences in wave reflection (Nichols & O'Rourke, 1992; Nichols & O'Rourke, 1998, as cited in Franklin, Shehzad, Wong, Larson, & Levy, 1999; O'Rourke, 1999). No differences in anger or exercise level were found in the first five minutes of the baseline period (all *p*'s > .05).

In order to determine whether the interview elicited cardiovascular reactivity and recovery, three repeated measures analyses of variance (ANOVAs) for each of the cardiovascular variables (SBP, DBP and HR), with time (baseline, interview, recovery) as a within-subjects factor were conducted. There was a main effect of time, indicating that SBP was significantly different across all measures of time, $F(2,48) = 72.92, p < .001$, as was DBP, $F(2,48) = 60.74, p < .001$, as well as HR, $F(2,48) = 26.84, p < .001$.

Paired samples t-tests were used to examine the differences between SBP at T1 (baseline) and T2 (interview) and at T2 and T3 (recovery). SBP at T1 ($M = 120.02, SD = 13.74$) was significantly less than SBP at T2 ($M = 133.53, SD = 14.97$), $t(48) = 61.12, p < .001$. SBP at T2 was significantly higher than SBP at T3 ($M = 125.79, SD = 14.83$).

Paired samples t-tests were used to examine the differences between DBP at T1 and T2 and at T2 and T3. DBP at T1 ($M = 65.25, SD = 9.92$) was significantly less than DBP at T2 ($M = 75.50, SD = 11.67$), $t(48) = 46.03, p < .001$. DBP at T2 was significantly higher than DBP at T3 ($M = 69.53, SD = 10.09$), $t(48) = 45.30, p < .001$.

Paired samples t-tests were used to examine the differences between HR at T1 and T2 and at T2 and T3. HR at T1 ($M = 74.51, SD = 10.71$) was significantly less than HR at T2 ($M = 80.59, SD = 10.07$), $t(48) = 48.69, p < .001$. HR at T2 was significantly higher than HR at T3 ($M = 76.88, SD = 9.84$), $t(48) = 55.99, p < .001$.

Women were split into 2 groups depending on the phase of their menstrual cycle. They were categorized into either the follicular phase ($N = 18$) or the luteal phase ($N = 8$).

One woman was not assigned to either category as she was taking birth control that restricted the ovulation cycle. Eight ANOVAs (one for each of the three cardiovascular variables during both reactivity and recovery, and 2 additional analyses for raw exercise and anger expression scale scores) were conducted on female data to determine if menstrual phase was a factor in this experiment. Cardiovascular variables (HR, SBP and DBP), exercise and anger expression (raw scale scores) served as the dependent variables, with menstrual phase (follicular vs. luteal) as a between subjects variable. No significant results were found (all *p*'s > .05); therefore, the two female groups were combined across all subsequent analyses.

Cardiovascular reactivity

Univariate (3 x 2) ANCOVAs were used to examine the relationship between exercise (exercise vs. no exercise), gender (male vs. female) and the change in SBP, DBP and HR from baseline to the interview, while controlling for AXO (anger expression) and the Body Mass Index (BMI) of the participant.

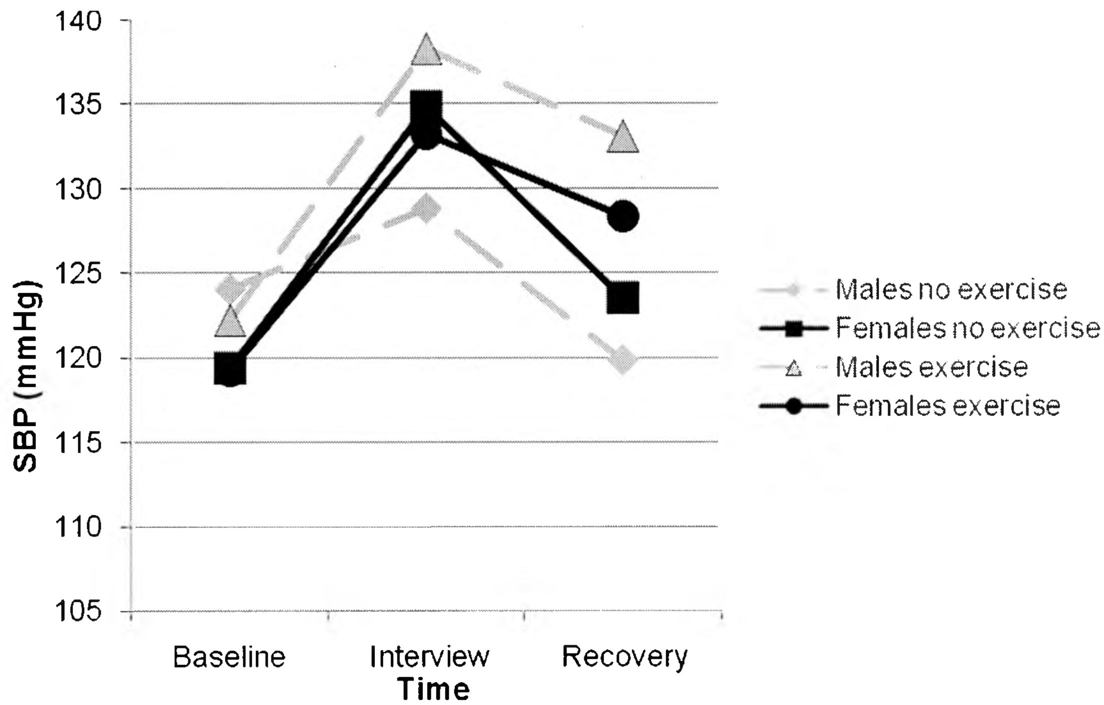

Figure 2. Mean systolic blood pressure across the three phases of the experiment

An ANCOVA was conducted on SBP reactivity values to examine the relationship between exercise, gender, and systolic reactivity while controlling for AXO and BMI. There was no significant difference by exercise on SBP reactivity, $F(1,48) = .13, p = .72$. There was no significant difference by gender on SBP reactivity, $F(1,48) = .20, p = .66$. A marginal interaction was found between gender and exercise for systolic blood pressure reactivity, $F(1,48) = 2.9, p = .10$

In addition, planned comparisons were conducted to determine whether gender and exercise had differential effects on SBP. An independent-samples t-test was used to compare males who did not exercise to females who did not exercise to SBP reactivity. Non-exercising males ($M = 8.80$, $SD = 11.022$) showed a marginally smaller systolic reactivity than did females who did not exercise (*M* = 15.79, *SD* = 11.27), $t(22) = 2.36$, $p = .165$. (see figure 3).

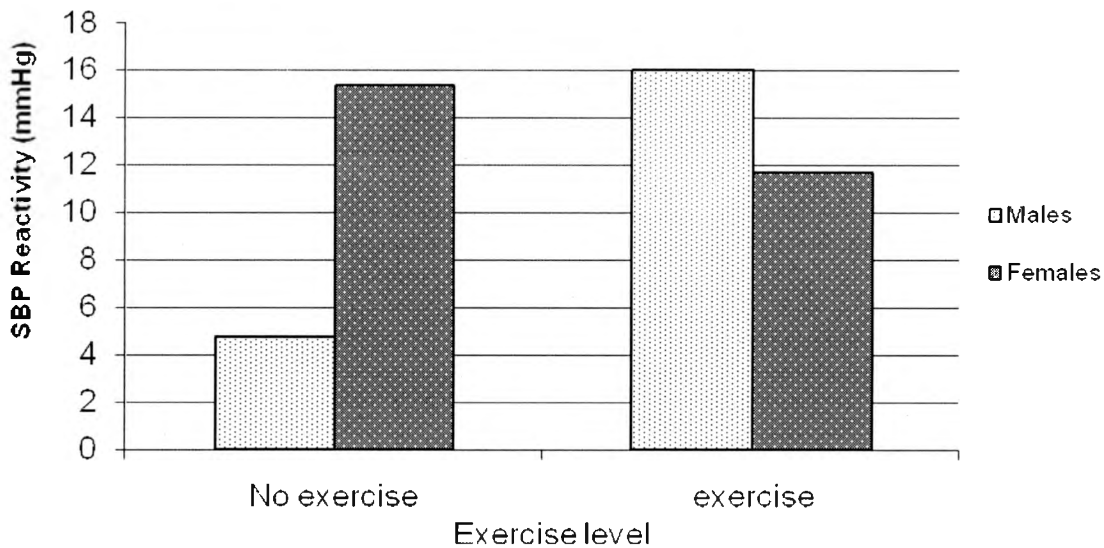

Figure 3. Systolic blood pressure reactivity (mmHg) by exercise level for males and females. Error bars represent standard error of the mean.

An ANCOVA with DBP as the dependent variable was conducted to examine the relationship between exercise, gender, and diastolic reactivity while controlling for AXO and BMI. There was no significant difference by exercise on DBP reactivity, $F(1,48) = .32$, $p = .58$. There was no significant difference by gender on DBP reactivity, $F(1,48) = .030$, $p = .86$. A marginally significant interaction was found between gender and exercise for diastolic blood pressure reactivity, $F(1,48) = 3.74$, $p = .06$.

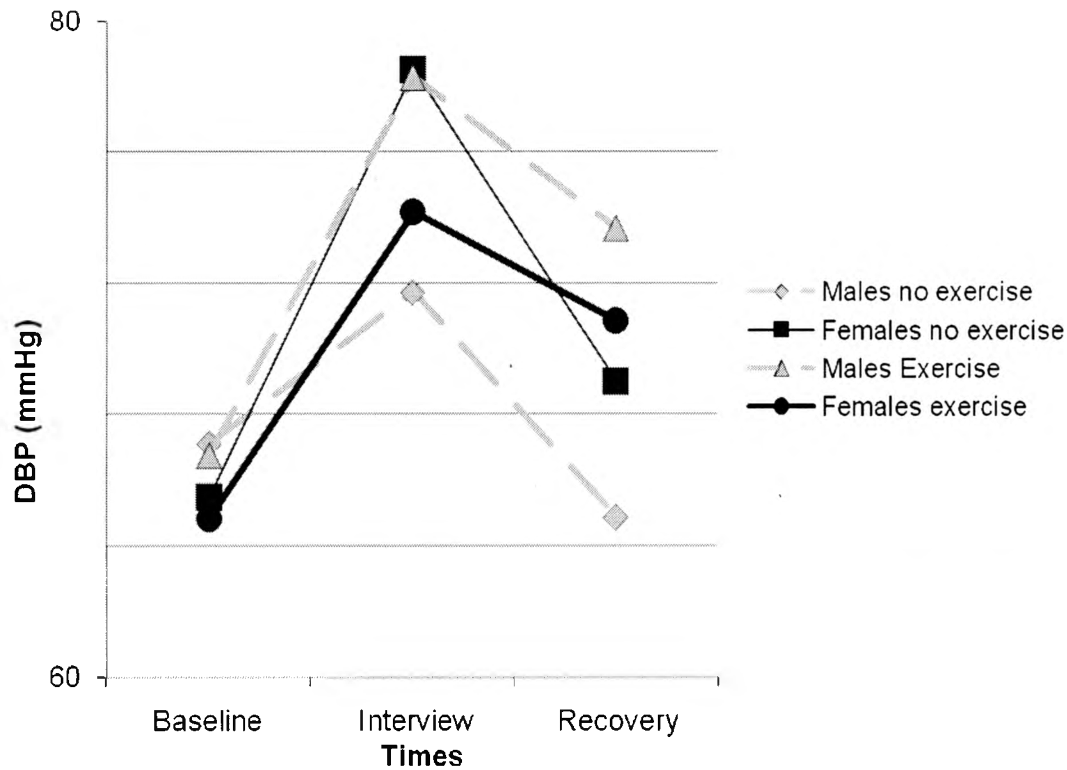

Figure 4. Mean diastolic blood pressure across the three phases of the experiment.

Planned comparisons were conducted to determine whether gender and exercise had differential effects on DBP. An independent-samples t-test was used to compare the DBP reactivity of males who did not exercise to females who did not exercise. Males who did not exercise ($M = 7.6$, $SD = 7.8$) showed a trend for smaller diastolic reactivity than did females who did not exercise ($M = 13.0$, $SD = 5.3$), $t(22) = 2.01$, $p = .057$.

An independent-samples t-test was also used to analyze the difference between reactivity DBPs for females who exercised and females who did not exercise. The results suggest that females who do not exercise ($M = 13.15$, $SD = 8.77$) have a marginally higher diastolic reactivity than do females who regularly exercise ($M = 6.9$, $SD = 8.48$), $t(26) = 2.23$, $p = .069$.

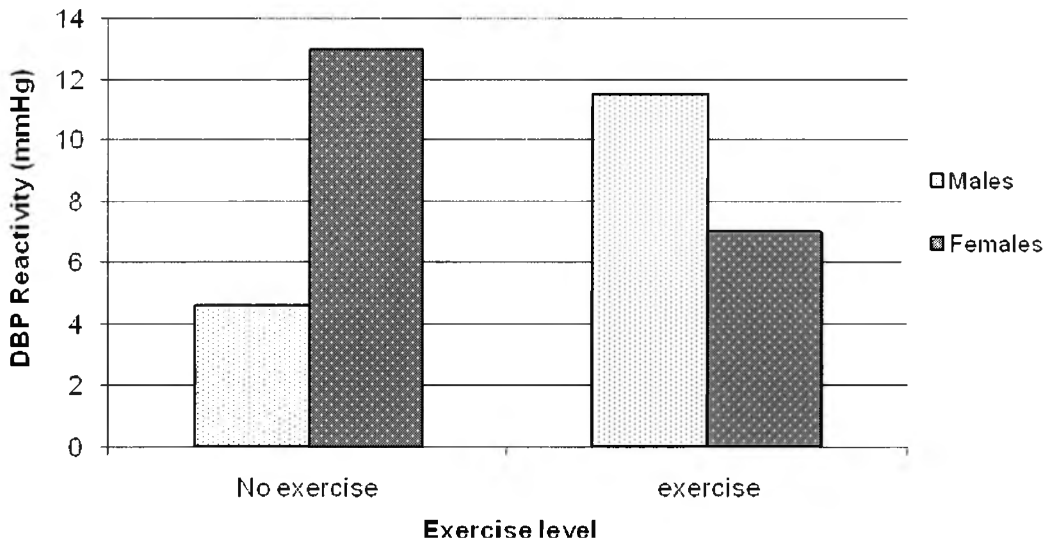

Figure 5. Diastolic blood pressure reactivity (mmHg) by exercise level for males and females. Error bars represent standard error of the mean.

An ANCOVA with heart rate reactivity as the dependent variable was used to examine the relationship between exercise, gender, and heart rate reactivity while controlling for AXO and BMI. There was no significant difference by exercise on HR reactivity, $F(1,48) = .837, p = .365$. There was no significant difference by gender on HR reactivity, $F(1,48) = .467, p = .498$. A marginal interaction was found between gender and exercise for heart rate reactivity, $F(1,48) = 2.750, p = .105$.

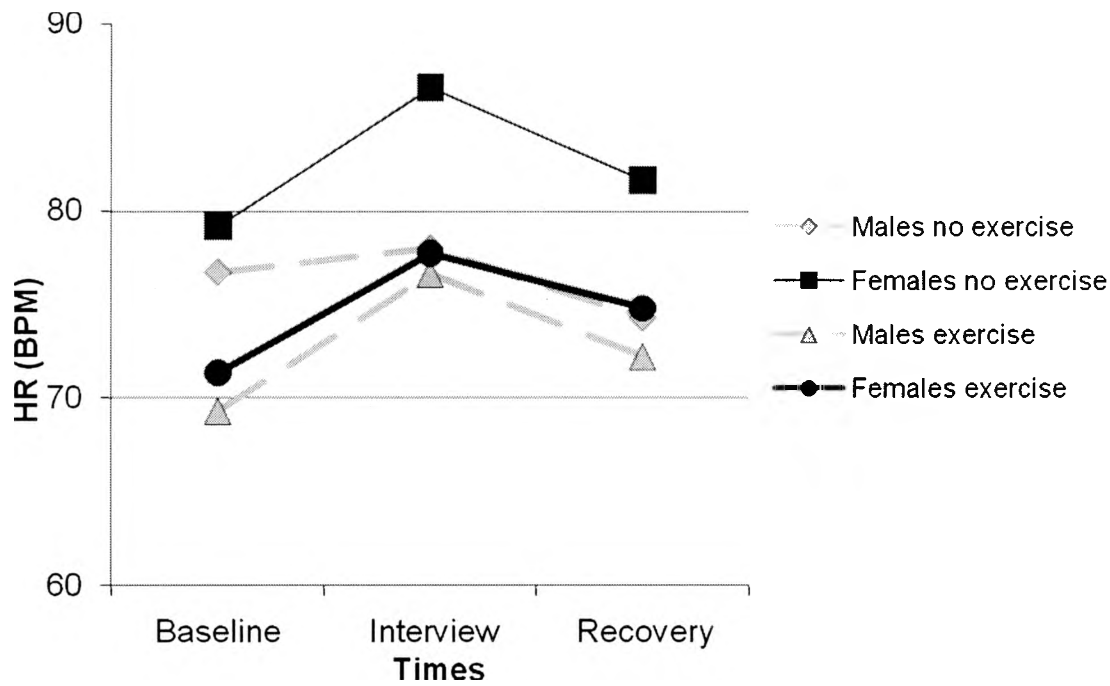

Figure 6. Mean heart rate across the three phases of the experiment

Planned independent-samples t-tests were used to compare males who did not exercise to females who did not exercise, males who exercised to males who did not exercise, females who exercised to females who did not exercise, and males who exercised to females who exercised to HR reactivity. All p's > .05.

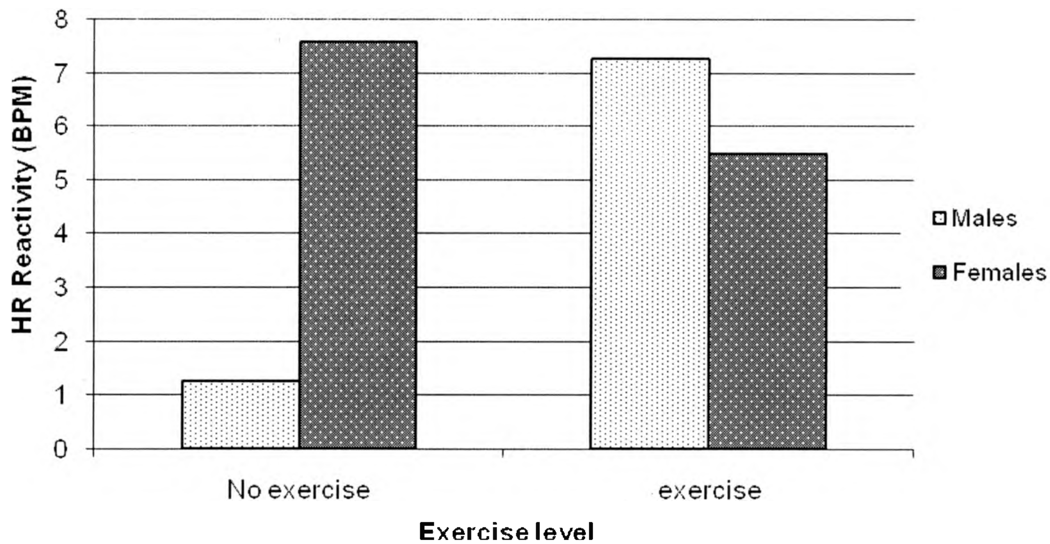

Figure 7. Heart rate pressure reactivity (BPM) by exercise level for males and females. Error bars represent standard error of the mean.

Cardiovascular recovery

Univariate (3 x 2) ANCOVAs were used to examine the relationship between exercise, gender and the differences over time between three measures of the cardiovascular response to stress; namely SBP, DBP, HR, while controlling for AXO (anger expression) and the Body Mass Index (BMI) of the participant. Differences in heart rate (HR), systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured for recovery periods.

An ANCOVA was conducted on SBP recovery values to examine the relationship between exercise, gender, and systolic recovery while controlling for AXO and BMI. A significant effect of exercise on SBP recovery was found showing that those who exercise ($M = 4.87$, $SD = 8.19$) had a smaller SBP recovery than those who did not exercise ($M = 10.74$, $SD = 9.63$), $F(1,48) = 4.13$, $p < .05$. There were no significant differences due to gender on SBP recovery, $F(1,48) = .01$, $p = .92$.

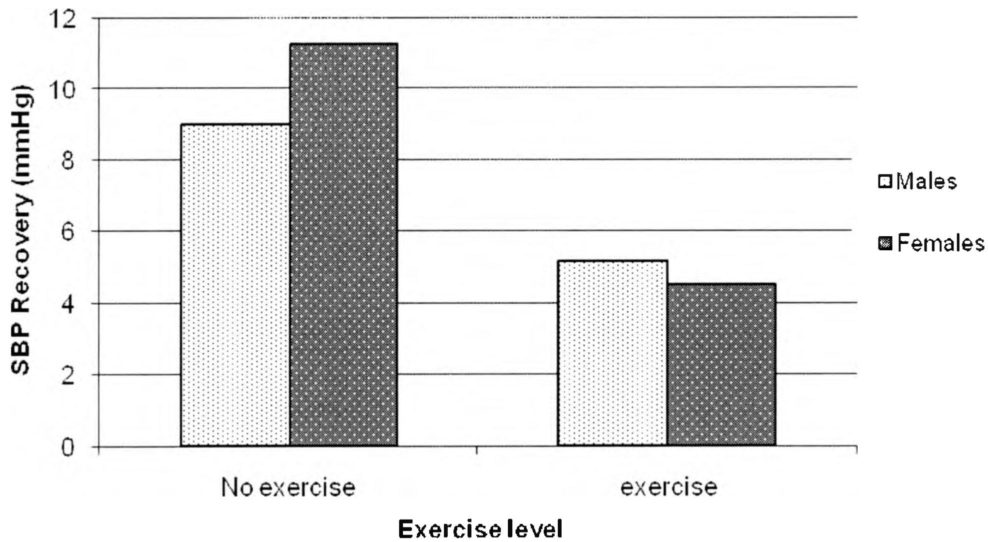

Figure 8. Systolic blood pressure recovery (mmHg) by exercise level for males and females. Error bars represent standard error of the mean.

A planned independent-samples t-test was used to compare females who did not exercise to females who did exercise. Females who exercised (*M* = 4.24, *SD* = 9.29) showed a marginally smaller systolic blood pressure recovery than females who did not exercise (*M* = 10.94, *SD* = 4.24), $t(26) = 1.87, p = .076$.

An ANCOVA was used to examine the relationship between exercise, gender, and diastolic reactivity while controlling for AXO and BMI. A significant effect of exercise on DBP recovery was found showing that those who exercise (*M* = 3.32, *SD* = 6.99) had less DBP recovery than those who do not exercise (*M*= 8.74, *SD* = 8.01), $F(1,48) = 4.800$, $p < .05$. There was no significant difference of gender on DBP recovery, $F(1,48) = .097, p = .757$.

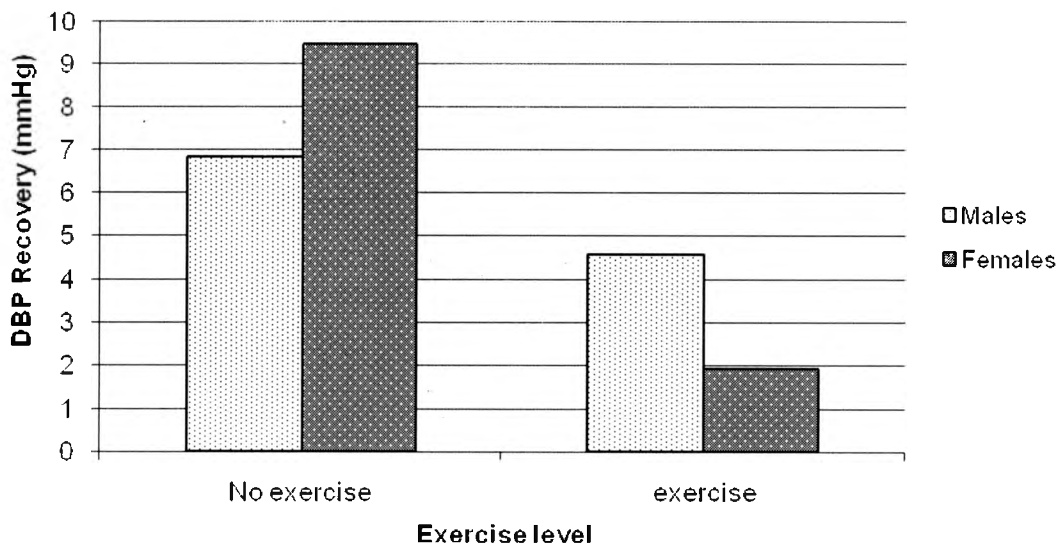

Figure 9. Diastolic blood pressure recovery (mmHg) by exercise level for males and females. Error bars represent standard error of the mean

A planned independent-samples t-test was used to compare females who did exercise to females who did not exercise to DBP recovery. Females who exercised (M = 1.75, SD = 7.76) were found to have a smaller recovery than females who did not exercise (M = 9.22, SD = 8.03), $t(26) = 2.50, p < .05$.

An ANCOVA was used to examine the relationship between exercise, gender, and heart rate recovery while controlling for AXO and BMI. There was no significant difference in exercise for HR recovery, $F(1,48) = .430, p = .516$. There was no significant difference in gender for HR recovery, $F(1,48) = .246, p = .622$.

A planned independent-samples t-test was used to compare females who exercised to females who did not exercise to HR recovery. Females who did not exercise showed a larger HR recovery (M = 4.83, SD = 1.86) than females who exercised (M = 1.27, SD = 2.08), $p < .0001$.

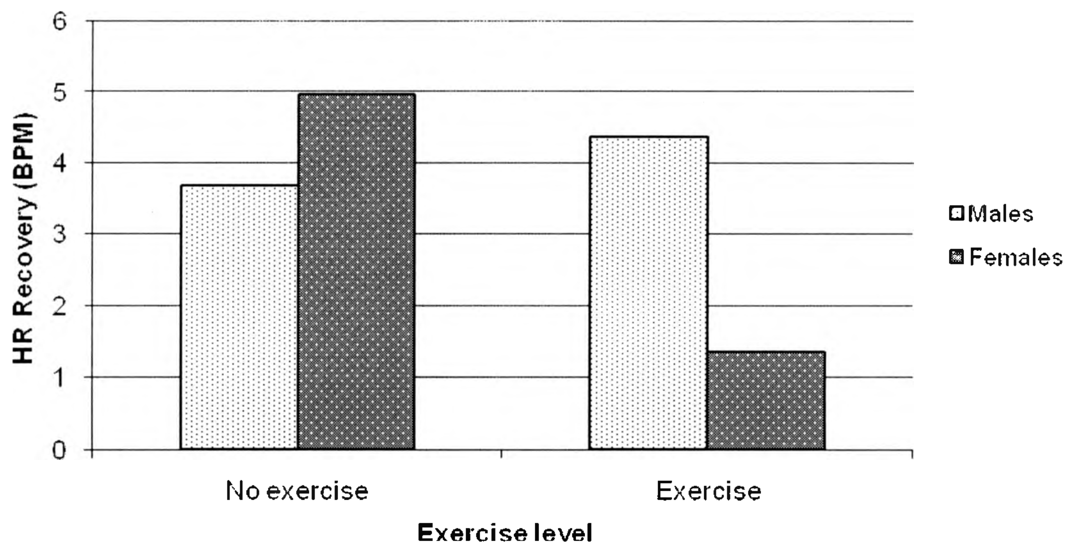

Figure 10. Heart rate recovery (BPM) by exercise level for males and females. Error bars represent standard error of the mean.

CHAPTER IV

DISCUSSION

Cardiovascular disease and hypertension are major problems in America today. Because stress is a possible cause of development of these diseases, there is a drastic need for a defense against this cause. As a heightened cardiovascular response to stress and a lengthened recovery from stress can be detrimental to health by increasing the risk for cardiovascular disease, it is important to examine the mechanisms by which this heightened response and lengthened recovery may be moderated. The present study was conducted to examine the effects of exercise on the cardiovascular reactivity and response to daily hassles. Because past studies have shown exercise to have a moderating effect on cardiovascular reactivity and response to major stressors and because daily hassles have been found to be highly correlated with major stressors, it was presumed that exercise would have a similar effect on daily hassles as it did on major stressors. This study, however, was not able to completely confirm that presumption.

Stress and cardiovascular reactivity

Exaggerated cardiovascular reactivity to stress has been shown to increase the risk for the development of hypertension, myocardial ischemia and coronary heart disease (Krantz & Manuk, 1984, 1986, as cited in Chafin, Christenfeld & Gerin, 2008; Lovallo & Wilson, 1992); therefore, it is important to understand the mechanisms by which this reactivity may be moderated. As previously mentioned, multiple studies have shown that exercise can help moderate the cardiovascular response to major stressors (e.g., Broman-Fulks et al., 2003; Criqui et al., 1982; Dishman et al., 2000). In contrast, the results of the current study did not show that exercise was a moderating factor to the cardiovascular reactivity to daily hassles. In fact, the only significant findings with regard to reactivity were the interaction between gender and exercise for systolic blood pressure and the marginal interaction between gender and exercise for diastolic blood pressure, and heart rate reactivity. These results are discussed in further detail below.

Consistent with the hypothesis and with previous literature, females who did not exercise were found to show a higher diastolic reactivity than females who exercised. Beyond the physical contributions added by exercise, such as a sustained lower heart rate and an overall reduction to the biological response to stress (Crews & Landers, 1987, as cited in Georgiades et al., 2000), exercise may also offer a therapeutic effect to stress. Exercise can be a way to escape from daily stressors, which may eliminate some of the accumulative effects shown for daily stressors. Larger ACTH and cortisol responses to stress (Traustadottir, Bosch, & Matt, 2003) might be particularly true for women who do not exercise, possibly explaining the finding that who did not exercise had a higher diastolic reactivity than females who exercised. Long (1991) demonstrated that after a mental arithmetic task and an "electrocardiogram quiz", women who were physically fit demonstrated a decrease in heart rate and norepinephrine levels while women who were less fit showed an increase in these levels during the 5 min to 30 min recovery period. That women who did not exercise exhibited a larger DBP reactivity than women who exercised while men did not show that same trend could be explained if women who do not exercise do, in fact, have an even larger ACTH and cortisol responses to stress.

A significant gender by exercise interaction was observed for SBP reactivity, such that males who did not exercise tended to have a smaller overall smaller cardiovascular reactivity (i.e., systolic, diastolic blood pressure, and heart rate reactivity) than did females who did not exercise. Because exercise levels in these two groups were the same and for the influence of anger expression was statistically controlled, it can be inferred that the differences in scores were due solely to gender. Previous research has shown women to have larger ACTH and cortisol responses to stress (Traustadottir, Bosch, & Matt, 2003), which can directly influence the blood pressure response to a stressful event, including the discussion of a daily hassle. Future studies should measure cortisol levels throughout the study to confirm if HPA axis activation also varies as a function of exercise and gender.

That there were no significant main effects of gender or exercise with regard to cardiovascular reactivity is noteworthy. Because exercise has been found in numerous studies to offer a protective effect on cardiovascular reactivity, it was surprising to not find any significant given the correlations between major stressors and minor stressors (eg. Hickey et al., 1975; Miall, 1958; deVries, 1980). One possibility for the lack of significant main effects in this study is that, while there are several similarities and high correlations between major stressors and daily hassles and although the interview did elicit cardiovascular change, it is possible that discussion of a daily hassle was not enough to cause subjective stress. In fact, it is possible that participants felt a therapeutic release while discussing daily hassles. Had the interview itself been more stressful (i.e., experiencing the daily hassle itself), reactivity might have been more dramatic, allowing for dissociations between exercise and gender to emerge. These are certainly important considerations for future studies.

Stress and cardiovascular recovery

Those who exercised tended to show a smaller recovery than the non-exercise group, which was contrary to the hypothesis. It is possible that the smaller recovery levels for those who exercise is related to a smaller reactivity. If reactivity levels do not increase a substantial amount, there is not as much from which to recover. It is worth considering that, while major stressors tend to show a smaller reactivity and larger recovery in those who exercise because daily hassles are not as dramatic and do not elicit as large of a response, a smaller reactivity may actually mean a smaller recovery merely because there is not as great of a difference from which to recover.

While males did not show any significant differences dependent on exercise level, females who exercised showed an opposite trend than the hypothesis. Females who exercised actually had a smaller recovery than females who did not exercise, similar to the overall results. Seemingly, those who consistently exercise may tend to take on more in their life that would add to their daily hassles. Since women tend to ruminate, or excessively wallow, more so than men (Tamres et al., 2002), a larger amount of daily hassles may given women more about which they can ruminate, contributing to an extended cardiovascular recovery. This hypothesis will be discussed further in the following section.

Limitations and future directions

Exercise was found to offer a protective effect the cardiovascular reactivity of women. Although this was the only result consistent with the hypothesis, all of the results offer contributions to the body of knowledge because of the lack of prior studies on this subject. Nevertheless, it is important to recognize certain limitations of this study. First, recording problems with the psychophysiological apparatus led to the removal of several subjects. It is possible that with the inclusion of these subjects or a larger sample in general, a more representative sample would have been obtained, increasing the generalizability of the results. In addition, a larger sample size would have provided more statistical power, allowing for the detection of smaller effects. The current study did not control for variables such as ethnicity, which may also influence cardiovascular reactivity and recovery. Ethnicity was not included in this study because this has generally not been included in previous literature. Additionally, to find any differences due to ethnicity would require a large number of subjects and a population outside the scope of the current study. While Salomon and Jagusztyn (2008) found some differences in SBP and HR among white and Latina/o participants, this was specifically related to ethnic discrimination. Even with the lack of previous findings regarding ethnicity and cardiovascular reactivity or recovery, this variable is still important to evaluate to compare possible differences due to ethnicity.

It is certainly important to note the representative sample of exercise across participants. Groups did not differ significantly across gender or anger in exercise. Additionally, there was a broad range of exercise scores for all participants. This indicates that, while there were several limitations to the study, the lack of statistically significant results is in itself significant. However, that a median split of exercise groups was performed may have contributed to the lack of findings. Splitting exercise into two groups could possibly conceal differences between exercise groups that a more extreme split may have found. A possible quartile split may have given results more consistent with the hypothesis. This is an important consideration for future studies as participants tend to fall on a range of exercise levels and are not divided into two groups in real life situations. This being said, the complete range of scores obtained in this study allows for more substantial generalizability and should be considered a major asset of this study.

Limitations in the exercise scale itself should be considered. A self-report measure, such as that which was used in this study, can allow for personal interpretation and possible exaggeration. As each person differs in their assessment of exercise, especially the intensity measure, an objective measure of fitness should be used, rather than relying on a subjective interpretation of one's own exercise habits. Fitness measures such as VO_2Max, a measure relaying of the body's ability to use oxygen, or the Astrand-Rhyming nomogram test, which shows an estimate of the maximal oxygen uptake based upon heart rate, could provide a more accurate measure of participant exercise rates and fitness level (Jamieson, Flood, & Lavoie, 1994). These alternative measures should be considered for future studies.

Additionally, hostility is an important factor to consider in regards to personality differences that may contribute to the cardiovascular response to stress. Originally, the Cook-Medley Hostility Scale (Hathaway & McKinley, 1943, as cited in Vella, Kamarck & Shiffman, 2008) was planned to be used as a covariate in this study; however, the full scale was not given to the subjects so the data was not used. Hostility as an independent variable has been related to cardiovascular disease and to cardiovascular reactivity (Smith, 1992, as cited in Powch & Houston, 1996), making this an important variable to study relative to daily hassles.

While the variation of the Prkachin Interview was statistically successful in eliciting a cardiovascular response to daily hassles, there was no measure of just how much each minor stressor bothered the individual. There may be personality differences in how one measures a daily hassle or minor stressor; to some a daily hassle may be as simple as over cooked toast, to others a daily hassle may be a traffic jam that causes a missed appointment. While there was an explanation of what a pet peeve or daily hassle is to those who asked, a subjective sale of 1-10 may be informative to ensure each participant is bothered experiences a similar level of subjective stress.

It may also be beneficial for future studies to find alternative methods to bring about emotions and reactions caused by daily hassles. The current study used a self-disclosure method, intended to bring about an emotional response associated with the daily hassle being discussed. However, some participants may have found the self-disclosure therapeutic, thereby skewing the results. It has been suggested that writing or talking about emotional experiences is related to an increase in immune functioning and a decrease in heart rate and muscular activity (Greenberg & Stone, 1992; Pennebaker & Beall, 1986; Richards et al., 1995; as cited in Pennebaker1997). Additionally, long term improvements in grades and a quicker rate of finding a new job has been associated with writing about an emotional topic (Cameron & Nicholls, 1996; Krantz & Pennebaker, 1996; Pennebaker et al., 1990; Pennebaker, 1994; Spera, Buhrfeind, and Bennebaker, 1994, as cited in Pennebaker, 1997), suggesting that self-disclosure can be a beneficial and therapeutic mechanism by which to release stress. This may account for some discrepancies in the expected cardiovascular reactivity and recovery findings of this study. Future studies using this interview protocol should also assess reported mood states before and after the interview in order to examine this issue. It is recommended, however, to continue this type of measure to extract a response to stress as using a self-disclosure method seems to be a more realistic measure to elicit emotions of stress, than using laboratory stressors such as a math task, be it a minor stressor or a major stressor.

Because cortisol is highly correlated with stress, it would be also beneficial for future studies to measure cortisol levels in patients. It would be expected that women would exhibit higher levels of cortisol in response to daily hassles because of findings that females tend to show higher levels in response to major stressors compared to males. Without confirmation; however, it is not clear whether cortisol levels would show similar results to this study or tend to respond more similarly to the cortisol level changes to major stressors. Distinguishing cortisol levels by gender as well as by exercise level would add to the growing body of knowledge that can suggests mechanisms by which patients may lower major stressors and daily hassles, by which they may lessen their risk for cardiovascular disease.

An important implication from these results can be inferred from the finding that females who exercised showed a smaller cardiovascular recovery than females who did not exercise, should be studied further. Cerin et al. (2008) have found leisure-time physical activity (LTPA) to be significantly related to self-efficacy in English speaking adults. Bandura and Locke (2003) mention that self-efficacy is related to how well a person motivates him or herself during difficulties, therefore it is possible that those with a high self-efficacy may tend to take on more challenges as they tend to believe that they are able to accomplish these challenges more than those with a lower self-efficacy. This can lead to the assumption that those who exercise may be systematically different from those who do not exercise and tend to take on more tasks because of a higher self-efficacy. This would therefore lead to a higher accumulation of daily hassles. This assumption could help explain why females tended to show less recovery to daily hassles if they were exercisers, which is contrary to the expected results.

Differences in coping mechanisms may also help explain why the cardiovascular recovery results for females did not mimic the cardiovascular results that males who exercised did not show a difference in SBP, DBP, or HR recovery compared to males who did not exercise. One possible reason for this is that women have been found to ruminate more than men (Tamres et al., 2002). Given this assumption, women who exercise would expect to have a slower recovery than those who do not exercise because they would have more to ruminate on during the recovery period. Future studies may want to investigate whether exercise as a moderator of the cardiovascular response to daily hassles is influenced by coping styles, such as rumination. If people who exercise are naturally inclined to assume more responsibilities than those who do not exercise, it may be even more important for them to continue their exercise regimen.

The effects of exercise and gender on reactivity and recovery could be studied more directly with completely within-subjects designs that include an exercise intervention. Future studies may want to assign participants to exercise and non-exercise groups to attempt to isolate the impact made on the cardiovascular response to stress by exercise, while controlling for other factors such as individual differences in rumination and hostility. An added benefit of this design is that it also allows for the simultaneous evaluation of individual differences, such as coping styles relative to the cardiovascular reactivity and recovery to daily hassles and the moderating effects of exercise.

This study represents one attempt to characterize cardiovascular changes to minor daily hassles and the moderating effects of gender and exercise. Comparisons of several studies with the current study will help add to the body of knowledge of how exercise may affect the cardiovascular response to daily hassles. Unfortunately, at this time, there is limited information on this subject against which to compare the current results. Given that daily hassles occur much more frequently than major stressors and may be a more important contributing factor to cardiovascular health, a better understanding of how the cardiovascular system is challenged by daily hassles and buffered by behaviors such as exercise and individual differences is necessary.

Applications

As previously suggested, the finding that those who exercised showed a slower cardiovascular recovery than those who do not exercise may indicate a systematic difference about those who exercise such that those who exercise may tend to take on more tasks which would lead to a larger accumulation of daily hassles. If this assumption is correct, it may be even more important for those who are naturally inclined to exercise to continue with their exercise. If an individual with this type of personality suddenly stops exercising while continuing to maintain the larger than average accumulation of daily hassles, an even more exaggerated cardiovascular recovery may appear. This would extend the amount of time that the body is in an aroused state, further contributing to a risk of cardiovascular disease.

Even though the results did not suggest that exercise offers a protective effect to the cardiovascular response to daily hassles, it is still important for the population to become informed of the benefits of exercise. First of all, the lack of results do not necessarily mean that exercise does not have a protective effect to the cardiovascular reactivity or recovery to daily hassles; rather there may be other intervening variables such as coping mechanisms that are concealing these effects in this study. Additionally, even without a cardiovascular benefit, exercise offers a way to escape from daily hassles, which is a safe and effective coping mechanism within itself. Exercise also offers other benefits, such as weight loss and improved overall cardiovascular function, both of which reduce the risk for cardiovascular disease and hypertension. Exercise has also been shown to reduce anxiety, which can build up during period of high stress. For these reasons, stressing the value of regular exercise is important for virtually everyone.

Conclusion

This study did not find cardiovascular reactivity to daily hassles to be moderated by exercise. As there is overwhelming evidence linking the moderation of exercise to the cardiovascular reactivity to major stressors, this lack of significance could be interpreted in one of two ways. Certainly, it could be that daily hassles do not cause as significant of a reaction as do major stressors, leading to the lack of findings. The other interpretation could be that a limitation to the study, be it a small number of participants, an ineffective method of simulating a daily hassle, or an inconsistent amount of daily hassles per patient, led to a lack of significant results. As this was a pioneering effort to study cardiovascular reactivity to daily hassles, more studies in this area can lend results to help answer this question. Although contrary to the hypothesis, the findings in this study are an important first step in recognizing the effects of exercise on the cardiovascular reactivity and recovery to daily hassles as well as any gender differences that may occur.

Given the limited amount of research on the use of exercise to influence the cardiovascular reactivity to and recovery from daily hassles, it is important that other research be done to continue the growth in this body of knowledge. Although there is much research that has been done showing the impact that exercise has on the cardiovascular response to major stressors, there has been little to no research on this relationship to minor stressors. As there is evidence showing that daily hassles, because of the cumulative effects, may have more impact on cardiovascular health than major stressors, it is important that the literature on the cardiovascular response to daily stress catches up to the literature on the cardiovascular response to major stress. While certainly not the most recognized cause of cardiovascular disease, cardiovascular reactivity to stress and cardiovascular recovery to stress have been shown to be critical sources for the development of hypertension and coronary heart disease. With the growing number of citizens affected by cardiovascular disease, such as hypertension and coronary heart disease, identifying mechanisms by which these diseases may be minimized or prevented altogether has becoming increasingly important. Cardiovascular disease can be debilitating and expensive. While weight loss and overall improved cardiovascular health has become a generally recognizable means of reducing risk factors for cardiovascular disease, continuing research on using exercise as a protective factor to minor stressors can establish additional benefits of exercise, which may potentially influence those at risk to seek the benefits of exercise.

APPENDIX

Interview Script:

1. Please describe a pet peeve of yours
2. When does this incident generally occur/when do you notice this?
3. Why is this important to you?
4. What are any physical responses you have to this experience?
5. What would you like to change about this experience/how would you like to change this experience?
6. How do you cope with this pet peeve?

REFERENCES

Anderson, S.F. & Lawler, K.A. (1995). The Anger Recall Interview and Cardiovascular Reactivity in Women: An Examination of Context and Experience. *Journal of Psychosomatic Research, 39,* 335-343.

Bandura, A. & Locke, E.A. (2003). Negative Self-Efficacy and Goal Effects Revisited. *Journal of Applied Psychology, 88*, 87-99.

Barefoot, J.C., Dodge, K.A., Peterson, B.L., Dahlstrom, G., & Williams, R.B. (1989). The Cook-Medley Hostility Scale: Item Content and Ability to Predict Survival. *Psychosomatic Medicine, 51,* 46-57.

Barefoot, J.C., Dahlstrom, G., & Williams, R.B. (1989). Hostility, CHD Incidence, and Total Mortality: A 25-Year Follow-Up Study of 255 Physicians. *Psychosomatic Medicine, 45,* 59-63.

Baecke, J.A., Burema, J., Frijters, J.E. (1982). A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *The American Journal of Clinical Nutrition, 36,* 936-942.

Bellarosa, C., & Chen, P. (1997). The Effectiveness and Practicality of Occupational Stress Management Interventions: A Survey of Subject Matter Experts. *Journal of Occupational Health Psychology,2*, 247-262.

Blankstein, K.R. & Flett, G.L. (1992). Specificity in the Assessment of Daily Hassles: Hassles, Locus of Control, and Adjustment in College Students. *Canadian of Behavioural Science, 24,* 382-398.

Blumenthal, J.A., Sherwood, A., Gullette, E.C.D., Babyak, M., Waugh, R., Georgiades, A., et al. (2000). Exercise and Weight Loss Reduce Blood Pressure in Men and Women With Mild Hypertension. *Arch Intern Med , 160,* 1947-1958.

Broman-Fulks, J., Berman, M., Rabian, B., & Webster, M. (2004). Effects of Aerobic Exercise on Anxiety Sensitivity. *Behaviour Research and Therapy,42*, 125-136.

Cerin, E., Vandelanotte, C., Leslie, E., & Merom, D. (2008). Recreational Facilities and Leisure-Time Physical Activity: An Analysis of Moderators and Self-Efficacy as a Mediator. *Health Psychology, 27,* S126-S135.

Chafin, S., Christenfeld, N., & Gerin, W. (2008). Improving Cardiovascular Recovery From Stress with Brief Poststress Exercise. *Health Psychology, 27,* 864-872.

Chida, Y. & Hamer, M. (2008). Chronic Psychosocial Factors and Acute Physiological Response to Laboratory-Induced Stress in Healthy Populations: A Quantitative Review of 30 Years of Investigation. *Psychological Bulletin, 134,* 829-885.

Christensen, A.J. & Smith, T.W. (1993). Cynical Hostility and Cardiovascular Reactivity During Self-Disclosure. *Psychosomatic Medicine, 55,* 193-202.

Cotman, C., & Berchtold, N. (2002). Exercise: a behavioral intervention to enhance brain health and plasticity. *TRENDS in Neurosciences (25)*, 295-301.

Davis, M.C. & Matthews, K.A. (1996). Do Gender-Relevant Characteristics Determine Cardiovascular Reactivity? Match Versus Mismatch of Traits and Situations. *Journal of Personality and Social Psychology, 71,* 527-535.

Dishman, R.K., Renner, K.J., White-Welkley, J.E., Burke, K.A., & Bunnell, B.N. (2000). Treadmill exercise training augments brain norepinephrine response to familiar and novel stress. *Brain Research Bulletin, 52,* 337-342.

Fitness, J. (2000). Anger in the workplace: an emotion script approach to anger episodes between workers and their superiors, co-workers and subordinates. *Journal of Organizational Behavior, 21,* 147-162.

Forcier, K., Stroud, L.R., Papandonatos, G.D., Hitsman, B., Reiches, M., Krishnamoorthy, J. et al. (2006). Links Between Physical Fitness and Cardiovascular Reactivity and Recovery to Psychological Stressors: A Meta-Analysis. *Health Psychology, 25,* 723-739.

Franklin, S.S., Khan, S.A., Wong, N.D., Larson, M.G. & Levy, D. (1999). Is Pulse Pressure Useful in Predicting Risk for Coronary Heart Disease? : The Framingham Heart Study. *Circulation, 100,* 354-360.

Georgiades, A., Sherwood, A., Gullette, E.C.D., Babyak, M.A., Hinderliter, A., Waugh, R., et al. (2008). Effects of Exercise and Weight Loss on Mental Stress-Induced Cardiovascular Responses in Individuals With High Blood Pressure. *Journal of the American Heart Association, 36,* 171-176

Girdler, S.S., Pedersen, C.A., Stern, R.A., & Light, K.C. (1993). Menstrual Cycle and Premenstrual Syndrome: Modifiers of Cardiovascular Reactivity in Women. *Health Psychology, 12,* 180-192.

Gollwitzer, M., Eid, M., & Jurgensen, R. (2005). Response Styles in the Assessment of Anger Expression. *Psychological Assessment, 17,* 56-69.

Ironson, G. et al. (1992). Effects of Anger on Left Ventricular Ejection Fraction in Coronary Artery Disease. *American Journal of Cardiology, 70,* 281-285.

Iwasaki, Y. (2001). Contributions of Leisure to Coping with Daily Hassles in University Students' Lives. *Canadian Journal of Behavioural Science, 33,* 128-141.

Izzo, J.L., Levy, D. & Black, H.R. (2000). Importance of Systolic Blood Pressure in Older Americans. *Hypertension, 35,* 1021-1024.

Jamieson, J., Flood, K., & Lavoie, N. (1994). Physical Fitness and Heart Rate Recovery from Stress. *Canadian Journal of Behavioural Science, 26*, 566-577.

Kamarck, T.W., Manuck, S. B, Jennings, J.R. (1990). Social Support Reduces Cardiovascular Reactivity to Psychological Challenge: A Laboratory Model. *Psychosomatic Medicine, 52,* 42-58.

Kanner, A.D., Coyne, J.C., Schaefer, C., & Lazarus, R.S. (1981). Comparison of Two Modes of Stress Measurement: Daily Hassles and Uplifts Versus Major Life Events. *Journal of Behavioral Medicine, 4,* 1-37.

Kolb, B. & Whishaw, I. Q. (2005). *An Introduction to Brain and Behavior.* New York: Worth Publishers.

Kopper, B.A. (1993). Role of Gender, Sex Role Identity, and Type A Behavior in Anger Expression and Mental Health Functioning. *Journal of Counseling Psychology, 40,* 232-237.

Lamensdorf, A.M. & Linden, W. (1992). Faily History of Hypertension and Cardiovascular Changes During High and Low Affect Provocation. *Psychophysiology, 29,* 558-564.

Ledwidge, B. (1980). Run for your mind: Aerobic exercise as a means of alleviating anxiety and depression. *Canad. J. Behav. Sci. (12)*, 126-140.

Long, B.C. (1991). Physiological and Psychological Stress Recovery of Physically Fit and Unfit Women. *Canadian Journal of Behavioural Science, 23,* 53-65.

Matthews, K.A., Davis, M.C., Stoney, C.M., Owens, J.F. & Caggiula, A.R. (1991). Does the Gender Relevance of the Stressor Influence Sex Differences in Psychophysiological Responses? *Health Psychology, 10,* 112-120.

Matthews, K.A., Gump, B.B., & Owens, J.F. (2001). Chronic Stress Influences Cardiovascular and Neuroendocrine Responses During Acute Stress and Recovery, Especially in Men. *Health Psychology, 20,* 403-410.

Neumann, S.A., Waldstein, S.R., Sollers, J.J., Thayer, J.F., & Sorkin, J.D. (2004). Hostility and Distraction Have Differential Influences on Cardiovascular Recovery From Anger Recall in Women. *Health Psychology, 23,* 631-640.

Ottaviani, C., Shapiro, D., Davydov, D.M., & Goldstein, I.B. (2008). Autonomic Stress Response Modes and Ambulatory Heart Rate Level and Variability. *Journal of Psychophysiology, 22,* 28-40.

Paffenbarger, R.S., Wing, W.L., & Hyde, R. T. (1995). Physical Activity as an Index of Heart Attack Risk in College Alumni. *American Journal of Epidemiology, 108,* 161-175.

Pennebaker, J.W. (1997). Writing About Emotional Experiences as a Therapeutic Process. *Psychological Science, 8,* 162-166.

Powch, I.G. & Houston, B.K. (1996). Hostility, Anger-In, and Cardiovascular Reactivity in White Women. *Health Psychology, 15,* 200-208.

Prkachin, K.M., Mills, D.E., Zwaal, C., & Husted, J. (2001). Comparison of hemodynamic responses to social and nonsocial stress: Evaluation of an anger interview. *Psychophysiology, 38,* 879-885.

Prkachin, K.M. & Silverman, B.E. (2002). Hostility and Facial Expression in Young Men and Women: Is Social Regulation More Important Than Negative Affect? *Health Psychology, 21,* 33-39.

Richardson, K., & Rothstein, H. R. (2008). Effects of Occupational Stress Management Intervention Programs: A Meta-Analysis. *Journal of Occupational Health Psychology, 13*, 69-93.

Roth, D.L., Wiebe, D.J., Fillingim, R.B., & Shay, K.A. (1989). Life Events, Fitness, Hardiness, and Health: A Simultaneous Analysis of Proposed Stress-Resistance Effects. *Journal of Personality and Social Psychology, 57,* 136-142.

Rozanski, A., Blumenthal, J.A., & Kaplan, J. (1999). Impact of Psychological Factors on the Pathogenesis of Cardiovascular Disease and Implications for Therapy. *Journal of the American Heart Association, 99,* 2192-2217.

Salomon, K. & Jagusztyn, N.E. (2008). Resting Cardiovascular Levels and Reactivity to Interpersonal Incivility Among Black, Latina/o, and White Individuals: The Moderating Role of Ethnic Discrimination. *Health Psychology, 27,* 473-481.

Schwerdtfeger, A., Konermann, L., & Schonhofen, K. (2008). Self-Efficacy As a Health-Protective Resource in Teachers? A Biopsychology Approach. *Health Psychology, 27,* 358-368.

Seigman, A.W. (1994). From Type A to Hostility and Anger: Reflections on the History of Coronary- Prone Behavior. In A. W. Seigman & T. W. Smith (Eds) *Anger, Hostility, and the Heart* (pp. 1-22). Hillsdale, NJ: Lawrence Erlbaum.

Siegman, A.W., Smith, T.W. (1993). Anger, Hostility, and the Heart (pp. 25-26). Hillsdale, NJ: Lawrence Erlbaum.

Smith, T.W., Glazer, K., Ruiz, J.M., & Gallo, L.C. (2004). Hostility, Anger, Aggressiveness, and Coronary Heart Disease: An Interpersonal Perspective on Personality, Emption, and Health. *Journal of Personality, 72,* 1217-1265.

Spielberger, C.D. (1988). *STAXI. State-Trait Anger Expression Inventory.* Tampa, FL: Psychological Assessment Resources.

Stewart, J.C., Janicki, D.L., & Kamarck, T.W. (2006). Cardiovascular Reactivity to and Recovery From Psychological Challenge as Predictors of 3-Year Change in Blood Pressure. *Health Psychology, 25,* 111-118.

Suchday, S., Carter, M.M., Ewart, C.K., Larkin, K.T., & Desiderato, O. (2004). Anger Cognitions and Cardiovascular Recovery Following Provocation. *Journal of Behavioral Medicine, 27,* 319-341.

Tamres, L.K., Janicki, D., & Helgeson, V.S. (2002). Sex Differences in Coping Behavior: A Meta-Analytic Review and an Examination of Relative Coping. *Personality and Social Psychology Review, 6,* 2-30.

Taylor, A. & Katomeri, M. (2006). Effects of Brisk Walk on Blood Pressure Responses to the Stroop, a Speect Task and a Smoking Cue Among Temporarily Abstinent Smokers. *Psychopharmacology, 184,* 247-253.

Traustadottir, T., Bosch, P.R., & Matt, K.S. (2003). Gender Differences in Cardiovascular and Hypothalamic-Pituitary-Adrenal Axis Responses to Psychological Stress in Healthy Older Adult Men and Women. *Stress, 6,* 133-140.

Wagner, B.M., Compas, B.E., & Howell, D.C. (1988). Daily and Major Life Events: A Test of an Integrative Model of Psychosocial Stress. *American Journal of Community Psychology, 16,* 189-205.

Watson, D. (1988). Intraindividual and Interindividual Analysis of Positive and Negative Affect: Their Relation to Health Complaints, Perceived Stress, and Daily Activities. *Journal of Personality and Social Psychology, 54,* 1020-1030.

Wilson, P.M., Mack, D.E., & Grattan, K.P. (2008). Understanding Motivation for Exercise: A Self-Determination Theory Perspective. *Canadian Psychology, 2008,* 250-256.

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