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# Investigating The Effects Of Obesity On Cardiovascular Reactivity And Recovery From Acute Physical And Psychological Stress

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# INVESTIGATING THE EFFECTS OF OBESITY ON CARDIOVASCULAR REACTIVITY AND RECOVERY FROM ACUTE PHYSICAL AND PSYCHOLOGICAL STRESS

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ABSTRACT

The role that excess adipose tissue plays in chronic inflammation gives rise to its importance as an independent risk factor in cardiovascular dysfunction. By operationalizing chronic stress as obesity, we sought to explore the relationship between obesity and cardiovascular responses to laboratory stressors, including recovery from stress. Further, we examined five adiposity measures to determine which were most related to cardiovascular dysfunction. Degree of obesity was able to predict dysfunction in both reactivity and recovery. Body mass index and waist circumference were found to be the best predictors of cardiovascular dysfunction. Blunted reactivity and slowed recovery were found in participants with greater levels of adiposity.

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# I. INTRODUCTION/BACKGROUND

## Investigating the Effects of Obesity on Cardiovascular Reactivity and Recovery from Acute Physical and Psychological Stress

### Obesity

Obesity is a rapidly spreading epidemic threatening the health of those it affects. For over a decade, Mississippi has consistently had one of the highest concentrations of obesity in the United States. According to the Centers for Disease Control and Prevention in 2009, 34.4 percent of the population in Mississippi was considered obese (defined as having a body mass index ≥ 30). Obesity is associated with devastating health complications including an increased risk for premature death (Katzmarzyk, Janssen, & Ardern, 2003). The pathology of obesity has been shown to increase the likelihood of cardiovascular complications including the following: congestive heart failure, stroke, myocardial infarction, hypertension, diabetes, dyslipidemia, atherosclerosis and peripheral vascular disease (Abel, Litwin, & Sweeney, 2008; Kenchaiah et al., 2002; Lakka, Lakka, Salonen, Kaplan, & Salonen, 2001; Mensah, 2004; Stapleton, James, Goodwil, & Frisbee, 2008; Wang & Nakayama, 2010; Wilson, D'Agostino, Sullivan, & Parise, 2002).

### Obesity Induced Inflammation

Obesity is characterized by an excess of adipose tissue. Adipose tissue was once thought to be a passive organ, used as a storage device for triglycerides that could be

reabsorbed into the blood stream at times of energy depletion. Adipose tissue is composed mostly of adipocytes. Adipocytes secrete adipokines, a form of cytokines that promote inflammation (Berg & Scherer, 2005; Van Gaal et al., 2006). Adipokines are often produced in parallel with other proteins that also cause inflammation. Normal cardiovascular functioning is hindered by the inflammation resulting from excess adipose tissue mediated by adipokines.

To understand how obesity affects the cardiovascular system, it is necessary to have a basic understanding of the inflammatory process associated with obesity. Comprehension of the pathways through which these chemicals inflict inflammation is complicated by the interactions of many of the chemicals on each other as well as tissues in the body. The chemicals involved in this process are many. For this reason only a few of the key players involved in the mediation between obesity and cardiovascular dysfunction will be described: leptin, tumor necrosis factor-alpha, interleukin-6, adiponectin, c-reactive protein, resistin and angiotensinogen (Singer & Granger, 2007).

Zhang et al. (1994) is often credited as opening the door for adipocyte endocrinology with the identification of adipose tissue as the source of the hormone leptin. In humans, leptin is produced proportionally to adipose tissue, thus obesity is associated with elevated leptin (Vachharajani & Granger, 2009; Van Gaal et al., 2006). Leptin, while causing some damage on its own, also stimulates the releases of tumor necrosis factor-alpha (TNF-$\alpha$) (Vachharajani & Granger, 2009). Due to leptin elevation in obesity, TNF-$\alpha$ is concurrently elevated. A review of the literature by Berg and Scherer (2005) found the effects of TNF-$\alpha$ on vasculopathy to be controversial. A more

recent review of the vascular dysfunction literature (Stapleton et al., 2008) found TNF-α to have direct effects on endothelial dysfunction.

Elevated levels of interleukin-6 (IL-6) also found in correlation with obesity have demonstrated instigation of endothelial function (Stapelton et al., 2008). This instigation disrupts the normal protective functioning of endothelial cells. IL-6 also stimulates the release of other inflammatory adipokines (Singer & Granger, 2007). Although a number of adipokines are elevated in relation to obesity, levels of the adipokine adiponectin are decreased (Berg & Scherer, 2005; Wang & Nakayama, 2010). A recent review of the literature by Wang and Nakayama (2010) reveals adiponectin works through a number of mechanisms to reduce inflammation; an example is the suppression of TNF-α and IL-6. Elevated levels of c-reactive protein (CRP) are undoubtedly linked to the prevalence of adipose tissue (Berg & Scherer, 2005). CRP is a specific marker of chronic inflammation (Wang & Nakayama, 2010). CRP elevation appears in response to circulating levels of TNF-α and IL-6; CRP then amplifies the inflammatory effects of other adipokines (Vachharajani & Granger, 2009).

Although many of the adipokines have a clear relationship with adipose tissue, this is not true for all. A literature review of a recently identified adipokine, resistin, shows no consensus regarding obesity and resistin levels in humans (Singer & Granger, 2007). Resistin should not be underestimated for its role in inflammation, stimulating increased production of TNF-α and IL-6 (Fu, Luo, L., Luo, N., & Garvey, 2006). Inhibition of anti-inflammatory cytokines by resistin also increases inflammation (Vachharajani & Granger, 2009).

The level of the protein angiotensinogen (AGT) is related to the pervasiveness of adipose tissue, yet this relationship seems to apply only to the prevalence of visceral adipose tissue (Singer & Granger, 2007). AGT is a known precursor to angiotensin II, which has direct links to vasculopathy and blood pressure. The elevated level of AGT in concurrence with visceral adipose tissue is further evidence of the importance of central adipose accumulation over that of total body or peripheral accumulation in the development of cardiovascular dysfunction (Singer & Granger, 2007). Decreases in IL-6, CRP and TNF-α have been reported in clinical trials of angiotensin II blockade (Berg & Scherer, 2005). This suggests one mechanism for AGT's inflammatory effect is stimulation of TNF-α, IL-6 and CRP production.

In obese subjects the excess adipose tissue, leading to elevated adipocyte circulation, is hypothesized to cause a state of chronic inflammation (Stapleton et al., 2008; Strohacker & McFarlin, 2010; Vachharajani & Granger, 2009). A substantial amount of research has demonstrated the deleterious effects of chronic stress on cardiovascular functioning. Few studies have assessed the effects of obesity as a chronic stressor on the cardiovascular system. The role that adipose tissue plays in chronic inflammation gives rise to its importance as an independent risk factor in cardiovascular dysfunction.

## Stress Response

Chronic stress is a poorly defined construct in cardiovascular physiology research that typically involves psychosocial factors. Chronic stress is commonly defined in the literature on cardiovascular reactivity as being social or environmental. However, chronic

stress of physical origin may provide new insight into impaired cardiovascular functioning. This study proposes to operationalize chronic stress as obesity.

When presented with a stressor the autonomic nervous system (ANS) and hypothalamic-pituitary-adrenal (HPA) axis are often activated simultaneously (Chida & Hamer, 2008). Bjorntorp (2001) gives a thorough description of the process of HPA activation. HPA activation in humans stimulates the production of cortisol from the adrenal glands, which then binds to glucocorticoid receptors. Stimulation of lipoprotein lipase (LPL) activity is seen with increases in cortisol (Ottosson, Vikman-Adolfsson, Enerback, & Olivecrona, 1994). LPL, when found in adipose tissue, acts as a catalyst for fat storage. Higher concentrations of glucocorticoid receptors in visceral rather than peripheral adipose tissue leads to a rise in cortisol binding in the visceral tissue. This binding leads to an increase in LPL activity, resulting in accumulation of visceral adipose tissue, the site of higher glucocorticoid receptor expression.

One consequence of cortisol production leading to visceral adipose tissue expansion is the complication of interpretation. It may be speculated that glucocorticoid receptor prevalence in existing adipose tissue leads to a cycle of continued increase in adiposity. A state of chronic stress lends evidence to a preexisting condition of impaired stress response or recovery predating that of obesity. This conjecture is outside the scope of the current study, yet both ideas have been noted in the literature. This study will focus on cardiovascular effects once obesity has been established; how obesity came about will not be addressed directly.

While often acting in tandem with the HPA axis following stress, study of the ANS has entertained a greater body of research. The role of the ANS in regard to

vascular function will be the focus of the next section. The ANS can be subdivided into the sympathetic nervous system (SNS) and parasympathetic nervous system (PNS). Each of these subdivisions play a vital role in stress response and recovery. Quick and sufficient response to stress is an integral part of survival and imperative to human health. Complications in this continuum of activation and recovery can lead to devastating consequences.

**Reactivity Hypothesis**

When presented with a stressful situation, generalized arousal of the SNS is often helpful in managing the stressor. In some people, the arousal or reactivity of the SNS is greater than what is thought to be normal. Cardiovascular reactivity is classically defined as the change in cardiovascular reactivity from a resting or baseline state to one of psychological or physical challenge. The reactivity hypotheses as conceptualized by Obrist (1981) suggest exaggerated physiological response to acute stressors could lead to cardiovascular dysfunction such as hypertension or coronary heart disease. Several recent studies confirm large cardiovascular response to acute stress as a risk factor for cardiovascular disease (Lovallo & Gerin, 2003; Matthews, 2004; Schwartz, 2003). The reactivity hypothesis has found support in obese populations (Silva, 2009; Steptoe, 2005; Voudoukis, 1970) with some evidence pointing to central obesity as the mediating factor (Davis & Twamley, 1999; Davy & Hall, 2004).

**Blunted Reactivity**

Research suggests that it is not only an exaggerated response that may be harmful, as suggested by the reactivity hypotheses, but blunted reactivity to stress may also be considered dysfunctional. Blunted reactivity is characterized by a diminished

cardiovascular response from baseline to change produced by an acute stressor. Blunted reactivity has been found in obese subjects, such that as BMI increased, heart rate response to acute stress decreased (Carroll & Phillips, 2008; Phillips, 2010).

Finding both an increase and a decrease in physiological response to acute stressors in obese subjects may seem to create a confound. However, a recent comprehensive review of the literature by Lovallo (2010) clarifies why this dichotomy may exist. Lovallo points out a common theme in the literature where, until recently, a large physiologic response was thought to lead to complications whereas a smaller response is healthier. The review indicates the importance of exaggerated as well as diminished physiological reactions to stress as indicators of poor health outcomes. That is, an intermediate degree of cardiovascular response to stressors may be most adaptive in dealing with environmental events. Too much or too little response may be maladaptive and associated with negative health outcomes. Thus, future studies should examine stress response on a continuum where response on either end of the normative distribution should be evaluated as possibly problematic.

**Recovery from Stress**

A person's reactivity to stress is undoubtedly important in creating a picture of cardiovascular health; nevertheless, it may not be telling the whole story. Although working together the HPA axis and ANS can produce helpful responses to stress that allow for quick, efficient resolve of the stressor, problems occur when these systems work in sustaining the stress response. A review of the literature suggests recovery from stress may help illuminate complications in cardiovascular functioning, even when reactivity does not appear to yield impairments. Recovery is measured as the time it takes

to return to baseline levels of cardiovascular functioning once a psychological or physical challenge has ended.

A meta-analysis by Chida and Hamer (2008) demonstrated general life stress resulted in slowed recovery from laboratory stressors. Exposure to chronic stress has also been associated with poor recovery (Gump & Matthews, 1999; Lepore, Miles, & Levy, 1997). Impaired recovery has been positively correlated with obesity, when reactivity showed no relationship (Brydon, 2011). Further, results suggest more obese subjects or those with greater abdominal obesity are at an increased risk of recovery impairment (Steptoe, 2005). A return to individual baseline parameters will eventually occur. However, the ineffective recovery results in an overexposure to stress hormones, leading to adipose tissue accumulation and excessive demands on the cardiovascular system.

**Gender**

The existence of individual differences are known with respect to reactivity and recovery; however, there may be a gender component that adds to these differences. Thus, gender differences can complicate findings. For example, it has been demonstrated that women tend to have greater cardiovascular reactivity to some laboratory stressors than men (Matthews, 2004; Schmaus 2008). Others have shown that women but not men have a blunted response to some laboratory stressors (Dishman & Nakamura, 2003). A gender dimorphism in cardiovascular response may also result in differences with females exhibiting a "cardiac" response and males a more "vascular" response to stress (Allen, Stoney, Owens, & Matthews, 1993). The causes of these differences are not fully understood. Further, previous recruitment in our lab has demonstrated a significant lack

of male participation. This makes gender comparisons difficult; for these reasons only females were used for the current study.

**Appraisal of the event**

In 1984, Krantz and Manuck highlighted the importance of an individual's appraisal of an event on cardiovascular functioning. It is important to gauge the participants' appraisal of the laboratory stressor to help ensure an adequate amount of stress has been evoked to elicit a response. Others have also acknowledged the importance of appraisal when evaluating response to laboratory stress (Gump & Matthews, 1999; McEwen, 2007). There are variables that can affect how an individual appraises laboratory stressors. For instance, chronic stress could lead to differences in acute stress appraisal (Flemming, Baum, Davidson, Rectanus, & McArdle, 1987). A Likert scale of appraisal will be administered in the current study to evaluate the feeling of stress elicited in response to the laboratory stressors.

**Differences in reaction to mental vs physical stress**

There are many dimensions used to classify different types of stressors: sensory intake versus sensory avoidance, active versus passive, or psychological versus physical. The proposed study will examine the latter two dimensions. The differences in response and recovery to different types of laboratory stressors uncover differences in the cardiovascular pathways activated, as manifested in different hemodynamic patterns being elicited by different types of stressors. The current study used two laboratory stressors that, according to the literature, elicit different hemodynamic patterns of response.

Sharpley and Gordon (1999) demonstrated a difference in the cardiovascular reaction patterns initiated by the mental arithmetic (MA) versus the cold pressor (CP) task. The results showed the MA task elicited a sharp heart rate increase in the first thirty seconds of the task followed by a gradual decrease. The CP task showed a smaller heart rate increase that was maintained over the course of the task. These tasks have also been found to elicit different responses in blood pressure that will be described in more detail below. There is some consensus in the literature that the CP and MA tasks activate different pathways (Allen, Obrist, Sherwood, & Crowell, 1987; Andrén & Hannson, 1981, Gianaros & Sheu, 2009, Obrist, 1981; Willemsen, Ring, Carroll, Evans, Clow, & Hucklebridge, 1998). The attempts made to describe the mechanisms responsible for differences in pathways of response have not reached a consensus.

In a review of the literature, Gianaros and Sheu (2009) examined possible pathway differences that would explain the dichotomy. The CP task is a physical stressor that requires passive coping. The vascular resistance or vasoconstriction that occurs in response to temperature results in stimulation of α-adrenergic receptors. There appears to be agreement in the literature that α-adrenergically mediated vasoconstriction leads to an increase in total peripheral resistance (Allen et al., 1987; Andrén & Hannson, 1981; Willemsen et al., 1998).

The increase in total peripheral resistance resulting from α-adrenergic receptor activation is speculated to lead to a rise in diastolic but not systolic blood pressure (Willemsen et al., 1998). Others have found a rise in both systolic and diastolic blood pressure following the CP task (Allen et al., 1987; Andrén & Hannson, 1981). The rise in systolic blood pressure could also be the result of an increase in heart rate (Allen, et al.,

1987) although some suggest there is actually a decrease in heart rate following the CP task (Willemsen et al., 1998). The possible increase in heart rate may be mediated by activation of β-adrenergic receptors (Allen et al., 1987; Sherwood, Allen, Obrist, & Langer, 1986). However, others suggest the CP task does not elicit a β-adrenergic response (Matthews, 2004). Speculation has also risen about possible desensitization of β-adrenergic receptors in participants under chronic stress resulting in higher baseline levels of SNS activity (Benschop, 1994).

It is speculated that participants engage in active coping when presented with the MA task, which is considered a psychological stressor. During this task, increases in vascular resistance are seen in parallel with increases in heart rate. There is not complete agreement in the literature, but several studies have suggested these responses originate from both α and β-adrenergic receptor stimulation (Allen et al., 1987; Gianaros & Sheu, 2009; Willemsen et al., 1998).

Alpha-adrenergic receptors activated in response to the MA task are thought to result in vasoconstriction of peripheral vessels. This may contribute to increases seen in both systolic and diastolic blood pressure following the MA task (Phillips, 2010; Willemsen et al., 1998). However, myocardial β-adrenergic receptor activation may have a larger role in increasing heart rate due to the negligible changes in total peripheral resistance (Allen et al., 1987, Andrén & Hannson, 1981).

The adrenergic receptors are more commonly cited in the cardiovascular literature. This is not to say they are the only ones with an important role in cardiovascular functioning. Cholinergic receptors are primarily associated with the PNS. Willemsen et al. (1998) detailed their implications in CP and MA tasks. The CP task

elicited α-adrenergic stimulation as previously demonstrated, but the task was also associated with cholinergic activation. The MA task was found to be associated with cholinergic withdrawal as well as previously established mixed α and β-adrenergic stimulation. Additional evidence has been found for cholinergic activity in the MA task (Allen et al., 1987). Although this study is concerned primarily with SNS activation, it is imperative that a complete picture of activity is understood to correctly interpret results.

Relatively few studies have examined the early warning signs of impaired cardiovascular reactivity and recovery in obese populations. Results from many of the studies are mixed regarding cardiovascular measures and obesity, yet there seems to be some consistent effects. Baseline levels of blood pressure have been found to be higher in obese participants. Thus, controlling for baseline levels of cardiovascular functioning is a critical component for comparisons between tasks (Brydon, 2011; Carroll & Phillips, 2008, Phillips, 2010; Steptoe, 2005).

Heart rate reactivity appears to be negatively correlated with BMI, suggesting blunted cardiac reactivity in obese participants (Carroll & Phillips, 2008; Phillips, 2010). However, obesity seems to have an exaggerated effect on blood pressure reactivity. This heightened reactivity seems to be attributed to an increase in diastolic blood pressure. More specifically, central adiposity may be a greater risk factor in exaggerated reactivity than peripheral adiposity (Carroll & Phillips, 2008; Davis & Twamley, 1999; Steptoe, 2005). There is evidence to suggest systolic blood pressure reactivity may be blunted in obese participants (Carroll & Phillips, 2008).

Measures of recovery have been neglected in many studies investigating obesity and cardiovascular reactivity. In studies where this information was considered, a delay

in diastolic blood pressure recovery was demonstrated in obese subjects, specifically those with greater abdominal obesity (Brydon, 2011; Steptoe, 2005). The reasons for these differences in obese subjects are not fully understood.

## Need For More Research

As mentioned previously, chronic stress in cardiovascular physiology literature is often characterized as psychosocial factors. The present study seeks to define chronic stress as obesity. Circulating levels of adipokines and other proteins resulting in inflammation have an impact on vascular functioning, and this influence can impair α and β-adrenergic receptor action. The specific effects adipokines have on the vascular system are often mediated through the excitation or inhibition of other chemicals.

Altered vessel functioning resulting from adipokines is often caused by their effect on nitric oxide (NO). The endothelial cells that line the interior of blood vessels produce NO, which leads to vasoprotective dilation of blood vessels (Förstermann & Münzel, 2006). In a review of the literature Lau (2005) found TNF-α, CRP and AGT impair vasodilatation by different mechanisms that inhibit NO. The presence of IL-6 and resistin has also been found to result in decreased NO (Eringa, Bakker, Smulders, Serné, Yudkin, & Stehouwer, 2007; Schrader, Kinzenbaw, Johnson, 2007; Singer & Granger, 2007).

Leptin seems to increase NO production, which in healthy individuals would lead to vasodilation (Lau, 2005). Hyperleptinemia resulting from increased levels of leptin seen in obese subjects would be expected to enhance vasoprotection (Davy & Hall, 2004). However, the expected influence of leptin leading to vasodilation is not seen. This is because of "leptin resistance," a recent finding that is coming to be known as a

hallmark of obesity (Beltowski, Wojcicka, & Jamroz, 2003; Rahmouni, Haynes, & Morgan, 2005). In a state of leptin resistance, the body is able to manufacture leptin, but its actions are impaired. The only adipokine discussed in this paper that has a negative relationship to obesity is adiponectin. Adiponectin, along with inhibiting the production of harmful adipokines, has also been shown to enhance vasodilation by increasing NO production (Goldstein, Barry, & Scalia, 2004). Unfortunately, the benefits of adiponectin are not realized in obese subjects due to decreased availability of this adipokine.

The increased production of endothelin 1 (ET-1) is a second mechanism shared by leptin, CRP and resistin that leads to impaired vascular functioning (Lau, 2005; Verma & Shu-Hong Li, 2003). Produced by the cardiovascular system, endothelin is a powerful vasoconstrictor leading to increased vascular resistance and elevated blood pressure (Cardillo, Kilcoyne, Waclawiw, Cannon, & Panza, 1999). These mechanisms of action represent only a few of the links between obesity and impaired vascular functioning. The relationship between obesity and its inflammatory assault on the vascular system is well documented (Berg & Scherer, 2005; Davy & Hall, 2004; Stapleton et al., 2008; Vachharajani & Granger, 2009; Van Gaal et al., 2006). The relative infancy of research on obesity and clinical hemodynamic functioning however creates a need to further define patterns of acute stress reactivity and recovery in obese subjects (Brydon, 2011; Carroll & Phillips, 2008; Phillips, 2010; Steptoe, 2005).

Finding a way to stop or drastically reduce the obesity epidemic would be the paramount answer to the negative implications excess adipose tissue has on the cardiovascular system. Obesity has been on the rise for years and shows no signs of slowing. For this reason it is imperative that research efforts be made to understand the

implications of obesity on vital body systems. The cardiovascular system is arguably the most critical. Understanding how this system is affected will lead to immediate but short-term solutions buying much needed time for the larger issue of obesity to be resolved.

## Purpose of the Current Study

The current study seeks to further explore the relationship between obesity and cardiovascular responses to laboratory stressors, including recovery from stress. However, there are a number of ways to index excess adiposity, so identifying which of five easily obtained measures of adiposity is most related to cardiovascular responses will be the purpose of the current study. Thus, we seek to not only examine cardiovascular reactivity and recovery in relation to excess adiposity, but to see which of the five adiposity measures are most related to cardiovascular dysfunction.

The ability to classify people correctly and efficiently as obese or overweight is important in the early diagnosis and prevention of obesity co-morbidities. There are a number of empirically accurate measures of adiposity: densitometry, dual-energy absorptiometry (DEXA), computed tomography (CT) and magnetic resonance imaging (MRI) (Rothman, 2008; Goran & Gower, 1999). However, these techniques are expensive and often require extensive training to operate properly (Goran & Gower, 1999). Thus, precise equipment is often not available, creating the need for less expensive and less demanding alternatives for diagnosis. We wish to investigate five anthropometric measures of obesity (body mass index, triceps skin fold, suprailiac skin fold, waist circumference and waist to hip ratio), and determine which, if any, are related to cardiovascular dysfunctions. Each of these measures has shown importance in creating an accurate and complete picture of obesity. A description of each of these follows.

**Body Mass Index**

Obesity as defined by body mass index has been used for decades as a convenient measure of obesity and related health complications. For instance, BMI has been shown to be a powerful predictor of high blood pressure (Bertsias & Mammas, 2003). However, using BMI to define obesity comes with a number of issues including inadequate specificity and sensitivity leading to misclassification (Rothman, 2008). Deurenberg et al. (2001) compared BMI measures of obesity to the more accurate DEXA. They found BMI incorrectly classified 8% of men and 7% of women as obese when they were not. More concerning, 41% of men and 32% of women were not classified as obese using BMI when DEXA found they were. Another major complaint of utilizing BMI as a measure of obesity is that is does not take into account the distribution or localization of excess adipose tissue. Numerous studies have demonstrated the importance of establishing where the surplus of adipose tissue resides (Davis & Twamley, 1999; Dobbelsteyn & Joffres, 2001; Kalkhoff, Harz, Kissebah, & Kelber, 1983; Pouliot, 1994; Vague, 1956). Thus, determining where the excess adipose tissue is located is an important part of clinical risk assessment for cardiovascular dysfunction, and BMI is insufficient for providing this information.

**Skin Folds**

There are a number of measures supposed to give insight into excess adipose deposition. Skinfold measurement can be taken from different standardized sites on the body to indicate different patterns of adipose accumulation. Two skinfold sites common in the literature are the triceps and suprailiac. In 1956 while researching fat distribution, Vague coined the terms “gynoid” and “android,” to describe peripheral and central

adiposity, respectively. Vague's conclusions regarding the significance of central adiposity over that of peripheral adiposity in the development of cardiovascular complications has been replicated in recent studies (Davy, 2009; Davy & Hall, 2004; Van Gaal, Mertens, & Block, 2006). The suprailiac skinfold measure, taken on the side of the body near the waist, has been shown to be a strong correlate of central obesity (Goran & Gower, 1999). Triceps skinfold, taken on the back of the arm approximately between the shoulder and elbow, is used as a measure of peripheral obesity (Shear, Freedman, Burke, Harsha, & Berenson, 1987). Both sites will be utilized in this study to gain additional information on the reliability of skinfold as a measure of adipose distribution and relation to cardiovascular dysfunction.

**Waist Circumference**

Measurement of waist circumference as a predictor of cardiovascular risk factors has been used in a number of studies. In 2001, Dobbelsteyn and Joffres reported that measurements taken over a seven-year period indicated WC as the best predictor of cardiovascular disease risk factors. However, WC was found to be a superior predictor over BMI and WHR for different reasons. Although WC was in general a stronger predictor of cardiovascular risk factors than BMI, WC was deemed superior to WHR due to the increased possibility of measurement error associated with WHR. This can occur as a result of the formula used to calculate WHR, where two measurements are required whereas WC requires a single measure. Referring back to the importance of central adiposity over peripheral in cardiovascular dysfunction, measurement of WC is specifically related to abdominal (central) obesity (Pouliot, 1994).

**Waist to Hip Ratio**

Dalton and Cameron (2003) acknowledge the ease of obtaining BMI and the correlation found in the literature between WC and abdominal obesity. Findings from their laboratory indicated WHR was more accurate than BMI or WC in identifying participants with vascular dysfunction. It should also be noted that obesity defined as WC captured the greatest number of participants, whereas WHR captured the smallest number with BMI falling in the middle. Thus, WHR according to this study is a better predictor of cardiovascular risk factors although a less robust measure of obesity.

Identifying a readily available anthropometric measure that accurately identifies obesity is critical. Determining what impact obesity, as defined by the above measures, has on cardiovascular functioning is the primary focus of this study. Based on past research investigating cardiovascular reactivity, exaggerated or blunted reactivity could be plausible. In contrast, the few studies that have examined cardiovascular reactivity as it relates to obesity would suggest a combination of effects. Blunted reactivity may be expressed by a decrease in heart rate, whereas blood pressure illustrates exaggerated reactivity. Recovery and appraisal of stressors will also add insight into the hemodynamic response elicited from laboratory stressors in obese participants.

# II. METHODOLOGY

## Participants

Seventy-Eight female participants were recruited from the University of Mississippi. Participants were recruited through introductory psychology courses requiring research participation. Mean age was 19.2 (SD = 1.3). All participants were non-smokers with no history of cardiovascular dysfunction or currently taking medication for hypertension (as self reported). Mean body mass index was 24.63 (SD = 6.18) $kg/m^2$. Of the 78 participants recruited for this study 16% were obese (BMI of 30 or greater), 22% were overweight (BMI between 25 and 29.9), 54% were normal weight (BMI between 18.5 and 24.9) and 9% were underweight (BMI of 18.4 or less).

## Measures

### Anthropometric measures.

***Body mass index.*** – BMI was calculated for each participant by the experimenter using the standard formula of body weight in kilograms divided by height in meters squared. Weight was measured using a manual balance beam scale in pounds then converted to kilograms. Height was measured using a wall-mounted stadiometer in inches then converted to meters squared.

***Waist circumference.*** – Waist circumference was obtained using measuring tape placed horizontally across the abdomen approximately at the navel. In subjects where the rib cage could be seen or palpated without discomfort, the tape was more specifically

placed horizontally between the twelfth rib and the ileac crest. Waist circumference was recorded in inches.

***Waist to hip ratio.*** – Each participant's measurement obtained from the waist circumference was divided by their hip circumference in order to calculated their waist to hip ratio. Hip circumference was obtained using measuring tape placed horizontally around the body at the widest point of the gluteus maximus. The hip ratio was recorded in inches.

***Skin folds.*** – Two sites were used to assess skin fold measurements. The right side of the body was used for both measurements. The first site was the triceps. The participant was asked to stand up straight with arms relaxed and hanging at the sides with palms turned forward. If palpable, the experimenter found the acromion (highest point of the shoulder) of the right arm; if this landmark could not be found the most superior point of the right arm was used. The experimenter then located the olecranon (tip of the elbow). Using a marker a small mark was placed on the participant midway between these two points. At this point, a vertical pinch was made parallel to the body along the posterior area of the arm. A Lange skin caliper was applied for measurement distal to the pinch; after four seconds had passed from the time of application, the measurement was taken. The caliper was removed and reapplied in the same fashion at least three times, until results consecutively reach consensus between one to two millimeters.

The second skin fold site was the iliac crest. The participant was asked to raise any clothing obstructing the site of measurement with the left hand. They were then asked to stand up straight with the right arm held in front of the body to allow access to the site. The iliac crest was palpated. Using a marker a small mark was placed on the

participant just above the crest. A horizontal pinch at a 45-degree angle was made at the mark. The skin caliper was applied for measurement ventral to the pinch, after four seconds had passed from the time of application the measurement was taken. The caliper was removed and reapplied in the same fashion at least three times, until results consecutively reach consensus between one to two millimeters.

**Physiological measures.**

***Blood pressure and heart rate.*** – A Suntech Tango automated blood pressure monitor was used to measure systolic and diastolic blood pressure. A blood pressure cuff was placed on the non-dominant upper arm of the participant. During cuff inflation, heart rate was also recorded.

***Heart rate variability***. – A Coulbourn Instruments bioamplifer attached to a Dataq Instruments analog-to-digital converter board was used to acquire a continuous measure of heart rate variability. The lead placement was as follows: Right Apex (RA) electrode was placed right below the clavicle in the intercostal space along the right midclavicular line, Left Apex (LA) electrode was placed right below the clavicle in the intercostal space along the left midclavicular line, Right Leg (RL) electrode was placed on the right foot/lower leg avoiding bony prominences for grounding purposes.

Dataq Instruments WinDaq data acquisition software was used to display the electrocardiogram waveform on a computer screen. This allowed viewing in real time; in addition, a copy was stored on a Dell personal computer for later analysis. Heart rate variability was calculated for baseline, tasks and recovery periods. This was done by calculating the time periods between adjacent R-waves using the peak-to-peak algorithms in the advanced CODAS programs supplied with Windaq. The mean successive

difference statistic was computed for baseline, tasks and recovery periods. This method has been used in a number of studies (Allen, Matthews, & Kenyon, 2000; Christie & Friedman, 2004). This statistic represents the average of the difference between successive heartbeats for a given period of time and is used as a measure of heart rate variability.

**Questionnaires.**

***General health questionnaire -12 (GHQ).*** – The GHQ12 consists of 12 items with responses on a four point Likert scale. The GHQ is a self-report measure used to assess the general mental health of participants while appearing to measure general health. The GHQ is a common and widely used tool in assessing mental health. It has demonstrated validity and reliability with an alpha coefficients ranging from 0.78 to 0.95 in various studies (Jackson, 2007).

***Perceived stress scale – 10 (PSS).*** – The PSS-10 consists of 10 items with responses on a five-point Likert scale. The PSS is a self-report measure used to obtain how stressful participants perceive events in their life to be. The PSS has demonstrated reliability with an alpha coefficient ranging from .84 to .86 depending on the sample population (Cohen, Kamarck, & Mermelstein, 1983).

***Likert scale of perceived stress specifically from laboratory stressors.*** – A 10 point Likert scale ranging from 0 (Not Stressful) to 9 (Extremely Stressful) was given to participants to assess the perceived stress produced by each laboratory stressor administered. As there were two stressors the Likert scale was administered twice, once following each laboratory stressor.

**Laboratory Stressors**

### Cold pressor.

The cold pressor task lasted for a total of ninety seconds. Water temperature was measured using a digital thermometer and recorded on the data sheet. Participants were asked to place their dominant hand in a container filled with ice and ice water. The participant was instructed to place their hand palm down, with fingers spread apart to allow adequate water coverage, in the container. Using the pisiform bone as a landmark, the participant was asked to keep the water at a horizontal level across the wrist just above this bone.

### Mental arithmetic.

The mental arithmetic task lasted for a total of three minutes during which the participant was asked to subtract by 13s from 1764 as quickly and accurately as possible. If the participant gave an incorrect answer, the experimenter corrected the participant, then asked them to continue from that point.

## Procedure

Upon entering the laboratory, all potential participants were given an informed consent form to read over and sign. After the consent form had been signed, the participant was assigned an identification number and asked a series of questions to determine any contraindications to participation in the study (e.g., age, cardiovascular risk factors). The participant was asked to complete the GHQ and PSS. Weight and height were then measured. The participant was asked to remove their shoes and any outerwear they had on that would affect their weight. Waist circumference was measured using the method described above. In the interest of protecting female participants' privacy, this measurement was taken by a female experimenter while any male

experimenter present stepped out of the room. The participant was asked to raise her shirt to the belly button to ensure accurate measurement. The participant was instructed to stand up straight and breathe normally being sure not to hold their breath. The hip circumference and skin fold measurements were then taken using the methods described above.

The participant was instructed to sit in a lounge chair located in the laboratory, which faces a television. A blood pressure cuff was placed on the non-dominant upper arm of the participant. Following a brief explanation of electrode placement electrodes were placed on the participant. A three-leadwire system was used. Immediately prior to placing the electrodes, the area was cleansed with an alcohol wipe. The wipe was also used to gently abrade the area to encourage conductivity of electrical signal.

Following placement of the blood pressure cuff and electrodes, the participant was instructed to rest quietly while watching an introductory yoga video for ten minutes. The introductory yoga video was used to encourage relaxation while giving participants some stimuli to prevent sleeping. Monitoring by the electrodes was continuous throughout the study. During this and all subsequent rest periods blood pressure was taken at the beginning of the third, fifth, seventh and ninth minute. The first two readings were used to acclimate the participant to the feeling of the blood pressure cuff inflating and give the participant time to relax. Thus, these readings were discarded. The final two readings were averaged and the single number was used as a baseline blood pressure reading.

Once the initial rest period was complete, the participant performed either a mental arithmetic task or a cold pressor task; the order was be counterbalanced. The

mental arithmetic task lasted for a total of three minutes. During the mental arithmetic task, blood pressure readings were recorded at ten seconds, seventy seconds and 130 seconds. Measurements at all three time points were averaged to determine a mean blood pressure score for the task. Following the mental arithmetic task, the participant was given a Likert Scale of Perceived Stress. They were asked to rate their perception of the degree of stress produced by the mental arithmetic task.

The cold pressor task lasted for a total of ninety seconds. During the cold pressor task, blood pressure readings were recorded at ten seconds and sixty seconds. Measurements at both time points were averaged to determine a mean blood pressure score for the task. Following the cold pressor task, the participant was given a Likert Scale of Perceived Stress. They were asked to rate their perception of the degree of stress produced by the cold pressor task.

Following the first task, be it the mental arithmetic task or the cold pressor task, the participant was asked to watch the yoga video for a second rest period of ten minutes. Blood pressure readings were recorded as previously stated. Subsequent to administration of the final laboratory stressor, the participant had a third and final rest period. The electrodes and blood pressure cuff were removed and the participant was debriefed.

# III. RESULTS

## Manipulation Check

We first wanted to establish that significant differences were present between baseline levels and task levels on the cardiovascular measures; that is, that our tasks did indeed produce significant levels of cardiovascular response compared to baseline. Four repeated measures ANOVAs were performed to compare baseline, CP, and MA levels of SBP, DBP, HR and HRV. Greenhouse-Geisser corrections were used for the first three ANOVAs (SBP, DBP and HR) due to violations of sphericity. Mean scores were significantly different between periods for SBP ($F(1.432, 72) = 76.400$, $p < 0.001$), DBP ($F(1.610, 72) = 147.317$, $p < 0.001$), HR ($F(1.826, 72) = 40.925$, $p < 0.001$) and HRV ($F(2, 72) = 11.324$, $p < 0.001$).

Post hoc analyses using paired t-tests were performed to confirm both stressors (CP and MA) produced effects that were significantly different from baseline for all cardiovascular measures (SBP, DBP, HR and HRV). Bonferroni corrections were used for all p-values. For SBP both the CP and MA task were significantly different from baseline, $t(74) = -12.248$, $p < 0.001$, $t(76) = -8.705$, $p < 0.001$ respectively. Further, Figure 1 illustrates SBP during the CP and MA task were significantly different from each other, $t(73) = 5.459$, $p < 0.001$.

The CP and MA task were significantly different from baseline as well as each other for DBP, $t(74) = -17.121$, $p < 0.001$; $t(76) = -6.612$, $p < 0.001$; $t(73) = 9.708$, $p <$

0.001, respectively (see Figure 2). The CP and MA task were significantly different from baseline, but not from each other for HR, $t(74) = -8.755$, $p < 0.001$; $t(76) = -8.487$, $p < 0.001$; $t(73) = 0.778$, $p = 0.439$, respectively (see Figure 3). The CP and MA task were significantly different from baseline, however, not from each other for HRV, $t(64) = 3.841$, $p < 0.001$; $t(62) = 4.247$, $p < 0.001$; $t(60) = 0.314$, $p = 0.755$, respectively (see Figure 4).

**Correlational analysis of questionnaires**

In order to evaluate the impact of mental health, perceived stress and task induced stress, four questionnaires were administered to participants (GHQ, PSS, Likert scale CP and Likert scale MA). A correlation analysis was used to determine relationships among questionnaires, cardiovascular measures and measures of obesity. The GHQ was significantly correlated with baseline DBP $r(78) = .322$, $p < .01$; WC $r(78) = .314$, $p < .01$; WHR $r(78) = .357$, $p < .01$; CP Likert scale $r(78) = .239$, $p = .035$ and the PSS $r(78) = .696$, $p < .01$. Significant correlations were found between the PSS and baseline DBP $r(78) = .284$, $p = .012$; CP average DBP $r(75) = .255$, $p = .027$; DBP reactivity during the MA task $r(77) = -.238$, $p = .037$; and CP Likert scale $r(78) = .337$, $p < .01$. The CP Likert scale was significantly correlated with CP average HR $r(75) = .257$, $p = .026$. The MA Likert scale was significantly correlated with SBP reactivity during the MA task $r(77) = .293$, $p = .010$; MA average DBP $r(77) = .325$, $p < .01$ and DBP reactivity during the MA task $r(77) = .366$, $p < .01$. (see Table 1).

**Correlational analysis of adiposity measures**

To better understand the relationship between adiposity measures a correlation analysis was performed. All five measures of obesity were included in the analysis (BMI,

triceps skin fold, suprailiac skin fold, WC and WHR). As shown in Table 2, all measures were highly correlated as predicted. Due to strong correlations, all adiposity measures could not be used as predictors in a regression analysis. Therefore, a second correlation matrix that included the cardiovascular measures as well as the obesity measures was used to choose two predictors for the regressions. Body mass index and waist circumference were chosen to use as change score predictors in the regressions due to high correlations with cardiovascular measures and support in the literature (Geiss, Parhofer, & Scheandt, 2001; Schneider et al., 2007).

Significant correlations were found between BMI and baseline SBP, CP average SBP and SBP change score from baseline to MA task. A significant correlation was found between WC and baseline SBP, CP average SBP, MA average SBP and SBP change score from baseline to MA task. A significant correlation was also found between WC and HR change score from baseline to MA average (see Table 3).

**Hierarchical regression predicting reactivity**

Assessing differences in reactivity due to degree of adiposity was one major aim of the current study. Reactivity, defined as a change score, was computed as the difference between baseline and task average. Sixteen individual hierarchical regressions were performed to evaluate the relationship between reactivity and adiposity. After controlling for baseline values, either BMI or WC were used as the predictor for change scores for each task. Table 4 illustrates two regressions that yielded significant results. For HR, both BMI and WC were significant predictors of change score for the MA task $\beta = -.23$, $t(75) = -2.08$, $p = .041$; $\beta = -.24$, $t(75) = -2.21$, $p = .030$ respectively. BMI was

also a significant predictor of SBP change score for the MA task β = -.37, t(77) = -2.94, p = .004 (table 5).

**Hierarchical regression predicting recovery**

Recovery is often overlooked in studies of cardiovascular physiology. Findings are emerging in the literature that obesity may impact recovery from stress (Steptoe, 2005); importantly, this can occur even in the absence of impaired reactivity (Brydon, 2011). For this reason, assessing differences in recovery due to degree of adiposity was the second major aim of this study. Two recovery scores were used to evaluate recovery over time. The first recovery score was defined as the difference between task average and minute three of recovery. The difference between minute three and minute nine of recovery was used as the second recovery score. BMI and WC were used as predictors of recovery in thirty-two individual hierarchical regressions. After controlling for baseline and reactivity values, six of the regressions yielded significant results.

Both BMI and WC significantly predicted impaired SBP recovery. For the CP task, differences in recovery were found from minute three to minute nine (BMI) β = -.24, t(77) = -2.03, p = .046; (WC) β = -.25, t(77) = -2.30, p = .024 (see Table 6). Task average to minute three of recovery revealed differences following the MA task (BMI) β = -.36, t(77) = -2.97, p = .004; (WC) β = .29, t(77) = -2.38, p = .020 (see Table 7). Impaired HR recovery was significantly predicted by BMI. The first recovery score showed impairment following both the CP (β = -.12, t(75) = -2.27, p = .026) (see Table 8) and MA task (β = -.17, t(77) = -3.03, p = .003) (see Table 9).

# IV. DISCUSSION

The present study was designed to examine cardiovascular reactivity and recovery in relation to degree of adiposity. Further, we sought to determine which of the five adiposity measures employed by the study are most related to cardiovascular dysfunction. Although there was a high degree of correlation between adiposity measures, BMI and WC had the strongest relationship with cardiovascular measures.

Participants with a larger BMI and WC had higher levels of baseline SBP. Elevated SBP was maintained during the CP task. Further, elevated SBP persisted in participants with a larger WC during the MA task. BMI and WC were also related to change scores for the MA task, illustrated by a higher obesity score being related to blunted SBP reactivity. WC was additionally related to HR such that a larger WC was associated with blunted reactivity for the MA task. The relationship between obesity and higher baseline blood pressure was predicted and supports findings in the current literature (Brydon, 2011; Carroll & Phillips, 2008; Steptoe, 2005). Elevated resting blood pressure results in continuous stress on the vascular system. In obese populations, this added stress is compounded by other health concerns brought on by obesity.

A primary objective of the current study was to examine the relationship between cardiovascular reactivity, defined as change from baseline to task, and level of adiposity. Past research examining cardiovascular reactivity as it relates to obesity would suggest blunted reactivity may be expressed by a decrease in HR, whereas blood pressure may

illustrate exaggerated reactivity (Carroll & Phillips, 2008). The current study provides additional evidence for blunted cardiac reactivity in obese participants during the MA task. This finding was strengthened when subsequent analysis showed the relationship remained significant, even after controlling for baseline HR values. Findings by Carroll and Philips (2008) initiated a reevaluation of the direction of the relationship between obesity and blunted cardiac reactivity. Their results suggest blunted HR reactivity is a predictor of future obesity over a five-year period. The short-term nature of our study did not allow us to assess this important finding. However, it is clear that more longitudinal studies are needed to evaluate this directional relationship.

Previous research indicating greater obesity resulted in exaggerated blood pressure reactivity was not supported by the current study (Davis &Twamley, 1999; Goldbacher, Matthews, & Salomon 2005). Conversely, we found greater obesity to be related to blunted SBP reactivity during the MA task. Moreover, this finding maintained significance once baseline SBP was entered as a covariate. Blunted SPB reactivity has been found in obese populations, yet significance did not remain after adjustment for confounders (Carroll & Phillips, 2008).

A second objective of the current study was to examine the relationship between cardiovascular recovery and level of adiposity. Two time points were used to assess early and later recovery. Impaired cardiovascular recovery is a predictor of poor health outcomes, including mortality (Cole, Foody, Blackstone, & Lauer, 2000; Jae et al., 2006). Many times HR is the cardiovascular variable measured when assessing recovery (Schuler & O'Brien, 1997). Poor HR recovery suggests impairment of the parasympathetic system. We found greater adiposity predicted delayed HR recovery at

the first time point following both stress tasks. Delayed HR recovery in addition to blunted HR reactivity may result from impaired parasympathetic and sympathetic activation.

SPB recovery was also impaired. The use of two tasks types (active vs. passive) allowed us to evaluate presumably different pathways of activation. Interestingly, task type may also elicit differences in recovery. Following the MA task, participants with greater adiposity had delayed SBP recovery at the first time point. Delayed recovery during the second time point was predicted by greater adiposity following the CP task.

A possible mediator of delayed SBP recovery is impaired vasodilation as a result of insulin resistance. Obesity is a single component in a series of coexisting disorders comprehensively referred to as the metabolic syndrome or syndrome X (Gami et al., 2007). Decreased sensitivity to insulin, or insulin resistance, is another factor in syndrome X. Further, insulin resistance is common in obese populations (Viner, Segal, Lichtarowicz-Krynska, & Hindmarsh, 2005). This resistance to insulin impairs normal vasodilation (Steinberg et al., 1996).

In addition to cardiovascular measures, questionnaires were used to uncover relationships between obesity and stress. The questionnaires used to assess mental health and perceived stress (GHQ and PSS, respectively) showed a high degree of correlation. Both questionnaires had a positive relationship with self-reported stress from the CP task. The GHQ was positively related to obesity measures (WC and WHR). This supports findings in the literature that demonstrate higher GHQ scores are associated with greater obesity (Kivimäki et al., 2009). The bidirectional nature of the relationship complicates further interpretation. Obesity is as likely a predictor of poor mental health, as poor

mental health is of obesity. Higher scores on the PSS were associated with blunted DBP reactivity during the MA task. This finding substantiates previous results from our lab indicating lower DBP reactivity during the MA task is related to higher perceived stress (Allen, Bocek & Burch, 2011).

The Likert scales of perceived stress from stressor tasks (CP and MA) failed to indicate a relationship with the obesity measures. An interesting and unexpected finding was the relationship between the Likert scales and cardiovascular measures. Self-reported stress from the CP task was positively related to average HR during the task, whereas self-reported stress from the MA task was positively related to blood pressure during the task. The CP task is usually thought of as promoting alpha-adrenergic mediated vasoconstriction, thereby increasing blood pressure. Increases in heart rate are typically seen in conjunction with elevated blood pressure during the MA task, suggesting activation of both alpha and beta-adrenergic stimulation.

The relationship found between the Likert scale rating of task and the cardiovascular measures may be due to the question asked. The Likert scale consisted of a single measure "Please circle the number that corresponds with the amount of stress you experienced during the task." "Stressful" can be interpreted in a number of ways. Participants may have rated the CP task as stressful due to the pain and anxiety experienced during the task, whereas stressful during the MA task may have been more related to frustration or embarrassment.

There are a number of limitations in the current study. First, this study excluded males. Gender may play an important role in the relationship between obesity and cardiovascular dysfunction; therefore larger studies should evaluate the possible

influence of gender. Anthropometric measurements were taken prior to taking cardiovascular measures. Because women can be especially sensitive about their body size, these measurements may have resulted in some sympathetic arousal. Taking anthropometric measures at the end of the study can easily eliminate this concern. To better understand participant rating of a task as stressful, more measures are needed. Recording performance during the MA task may be able to tell us something about stress perception of the task. If participants who perform poorly report higher levels of stress, this could be interpreted as frustration. Assessment of pain during the CP task may also be helpful.

In conclusion, we were able to use obesity to predict dysfunction in both reactivity and recovery. Both BMI and WC predicted blunted cardiac reactivity during the MA task. Blunted SBP reactivity to the MA task was also predicted by BMI. Further, BMI predicted impairment of early cardiac recovery following both tasks. Slowed SBP recovery was predicted by BMI and WC. Following the MA task impairment was seen during the first recovery period, whereas the second recovery period was impaired following the CP task. Findings from the current study warrant further investigation of obesity as a chronic stressor. The chronic stress brought on by obesity can be defined not only as elevated resting levels of blood pressure, but a prolonged stress response. Further, although task type is commonly recognized as eliciting different patterns of stress reactivity, the current study has demonstrated the possibility of task type to produce differences in recovery.

V. TABLES/FIGURES

| **General Health Questionnaire** | | **Perceived Stress Scale** | |
|---|---|---|---|
| Baseline DBP | .322** | Baseline DBP | .284* |
| Waist Circumference | .314** | CP Average DBP | .255* |
| Waist to Hip Ratio | .357** | Change Score (BL to Math) DBP | -.238* |
| Likert Scale CP | .239* | Likert Scale CP | .337** |
| PSS | .696** | GHQ | .696** |
| **Likert Scale CP** | | **Likert Scale MA** | |
| CP Average HR | .257* | Change Score (BL to Math) SBP | .293** |
| GHQ | .239* | Math Average DBP | .325** |
| PSS | .337** | Change Score (BL to Math) DBP | .366** |

* indicates p < .05
** indicates p < .01

*Table 1.* Significant correlations between questionnaires, cardiovascular measures and measures of obesity.

| | Waist Circumference | Waist to Hip Ratio | Triceps Skin Fold | Suprailiac Skin Fold |
|---|---|---|---|---|
| BMI | .877** | .327** | .482** | .735** |
| Waist Circumference | | 0.661** | .422** | .737** |
| Waist to Hip Ratio | | | 0.125 | .430** |
| Tricep Skin Fold | | | | .648** |

** indicates p < .01

*Table 2.* Correlation table of anthropometric measures.

| | | **BMI** | **Waist Circumference** |
|---|---|---|---|
| **SBP** | Baseline | .416** | .411** |
| | CP Average | .322** | .337** |
| | Math Average | | .231* |
| | Change Score (BL to Math) | -.334* | -.238* |
| **HR** | Change Score (BL to Math) | | -.236* |

* indicates p < .05
** indicates p < .01

*Table 3.* Significant correlations between BMI or WC and cardiovascular measures.

**Predicting Change Score for Math Task**

| | Variable | B | Standard Error | Beta | $R^2$ | Significance |
|---|---|---|---|---|---|---|
| Step 1 | Constant | 24.601 | 6.912 | | | 0.001 |
| | Baseline HR | -0.202 | 0.086 | -0.261 | 0.068 | 0.022 |
| Step 2 | Constant | 34.09 | 8.161 | | | 0.000 |
| | Baseline HR | -0.215 | 0.085 | -0.278 | | 0.013 |
| | **BMI** | **-0.347** | **0.167** | **-0.227** | **0.119** | **0.041** |
| | | | | | | |
| Step 1 | Constant | 24.601 | 6.912 | | | 0.001 |
| | Baseline HR | -0.202 | 0.086 | -0.261 | 0.068 | 0.022 |
| Step 2 | Constant | 38.55 | 9.226 | | | 0.000 |
| | Baseline HR | -0.205 | 0.084 | -0.265 | | 0.017 |
| | **WC** | **-0.41** | **0.185** | **-0.241** | **0.126** | **0.030** |

*Table 4.* Regression predicting HR reactivity for MA task from BMI or WC.

**Predicting Change Score for Math Task**

| | Variable | B | Standard Error | Beta | $R^2$ | Significance |
|---|---|---|---|---|---|---|
| Step 1 | Constant | 16.067 | 9.974 | | | 0.111 |
| | Baseline SBP | -0.084 | 0.086 | -0.112 | 0.012 | 0.334 |
| Step 2 | Constant | 10.042 | 9.721 | | | 0.305 |
| | Baseline SBP | 0.055 | 0.095 | 0.073 | | 0.564 |
| | **BMI** | **-0.41** | **0.139** | **-0.371** | **0.116** | **0.004** |

*Table 5.* Regression predicting SBP reactivity for MA task from BMI.

**Predicting Recovery (Minute 3 to 9) for Cold Pressor Task**

| | Variable | B | Standard Error | Beta | $R^2$ | Significance |
|---|---|---|---|---|---|---|
| Step 1 | Constant | -1.681 | 8.350 | | | .841 |
| | Baseline SBP | .021 | .072 | .034 | .001 | .770 |
| Step 2 | Constant | -1.347 | 7.190 | | | .852 |
| | Baseline SBP | .034 | .062 | .055 | | .582 |
| | Change SBP | -.324 | .062 | -.518 | .269 | .000 |
| Step 3 | Constant | -4.495 | 7.213 | | | .535 |
| | Baseline SBP | .109 | .071 | .174 | | .130 |
| | Change SBP | -.356 | .063 | -.570 | | .000 |
| | **BMI** | **-.216** | **.107** | **-.235** | **.308** | **.046** |
| | | | | | | |
| Step 1 | Constant | -1.681 | 8.350 | | | .841 |
| | Baseline SBP | .021 | .072 | .034 | .001 | .770 |
| Step 2 | Constant | -1.347 | 7.190 | | | .852 |
| | Baseline SBP | .034 | .062 | .055 | | .582 |
| | Change SBP | -.324 | .062 | -.518 | .269 | .000 |
| Step 3 | Constant | -.846 | 6.993 | | | .904 |
| | Baseline SBP | .107 | .068 | .171 | | .121 |
| | Change SBP | -.351 | .062 | -.562 | | .000 |
| | **WC** | **-.261** | **.114** | **-.254** | **.319** | **.024** |

*Table 6.* Regression predicting SBP recovery from CP task using BMI or WC.

**Predicting Recovery (Task Average to Minute 3) for Math Task**

| | Variable | B | Standard Error | Beta | $R^2$ | Significance |
|---|---|---|---|---|---|---|
| Step 1 | Constant | -.650 | 11.506 | | | .955 |
| | Baseline SBP | .062 | .099 | .072 | .005 | .536 |
| Step 2 | Constant | 1.147 | 11.140 | | | .918 |
| | Baseline SBP | .043 | .096 | .050 | | .657 |
| | Change SBP | -.269 | .107 | -.281 | .084 | .014 |
| Step 3 | Constant | -5.818 | 10.849 | | | .593 |
| | Baseline SBP | .201 | .106 | .233 | | .061 |
| | Change SBP | -.245 | .102 | -.256 | | .019 |
| | **BMI** | **-.463** | **.156** | **-.364** | **.183** | **.004** |
| | | | | | | |
| Step 1 | Constant | -.650 | 11.506 | | | .955 |
| | Baseline SBP | .062 | .099 | .072 | .005 | .536 |
| Step 2 | Constant | 1.147 | 11.140 | | | .918 |
| | Baseline SBP | .043 | .096 | .050 | | .657 |
| | Change SBP | -.269 | .107 | -.281 | .084 | .014 |
| Step 3 | Constant | 1.560 | 10.808 | | | .886 |
| | Baseline SBP | .161 | .106 | .186 | | .133 |
| | Change SBP | -.218 | .106 | -.227 | | .043 |
| | **WC** | **-.418** | **.176** | **-.294** | **.149** | **.020** |

*Table 7.* Regression predicting SBP recovery from MA task using BMI or WC.

**Predicting Recovery (Average to Minute 3) for Cold Pressor Task**

| | Variable | B | Standard Error | Beta | $R^2$ | Significance |
|---|---|---|---|---|---|---|
| Step 1 | Constant | 28.763 | 7.045 | | | .000 |
| | Baseline HR | -.188 | .089 | -.241 | .058 | .037 |
| Step 2 | Constant | -.738 | 3.654 | | | .840 |
| | Baseline HR | .078 | .043 | .100 | | .075 |
| | Change HR | .865 | .052 | .931 | .808 | .000 |
| Step 3 | Constant | 4.568 | 4.251 | | | .286 |
| | Baseline HR | .064 | .043 | .082 | | .136 |
| | Change HR | .862 | .050 | .927 | | .000 |
| | **BMI** | **-.168** | **.074** | **-.115** | **.821** | **.026** |

*Table 8.* Regression predicting HR recovery from CP task using BMI.

**Predicting Recovery (Average to Minute 3) for Math Task**

| | Variable | B | Standard Error | Beta | $R^2$ | Significance |
|---|---|---|---|---|---|---|
| Step 1 | Constant | 15.928 | 7.352 | | | .033 |
| | Baseline HR | -.061 | .092 | -.077 | .006 | .505 |
| Step 2 | Constant | -6.909 | 3.908 | | | .081 |
| | Baseline HR | .126 | .047 | .158 | | .009 |
| | Change HR | .928 | .060 | .901 | .763 | .000 |
| Step 3 | Constant | 1.354 | 4.601 | | | .769 |
| | Baseline HR | .108 | .045 | .135 | | .019 |
| | Change HR | .886 | .059 | .860 | | .000 |
| | **BMI** | **-.264** | **.087** | **-.168** | **.789** | **.003** |

*Table 9.* Regression predicting HR recovery from MA task using BMI.

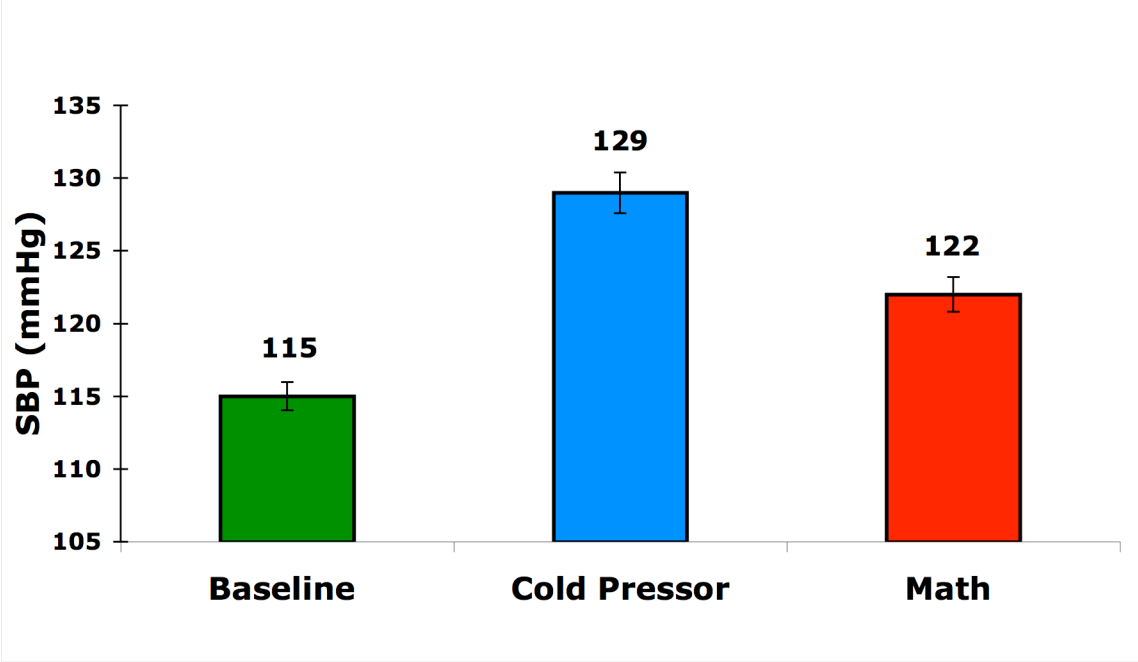


*Figure 1*. SBP during BL, CP and MA tasks.

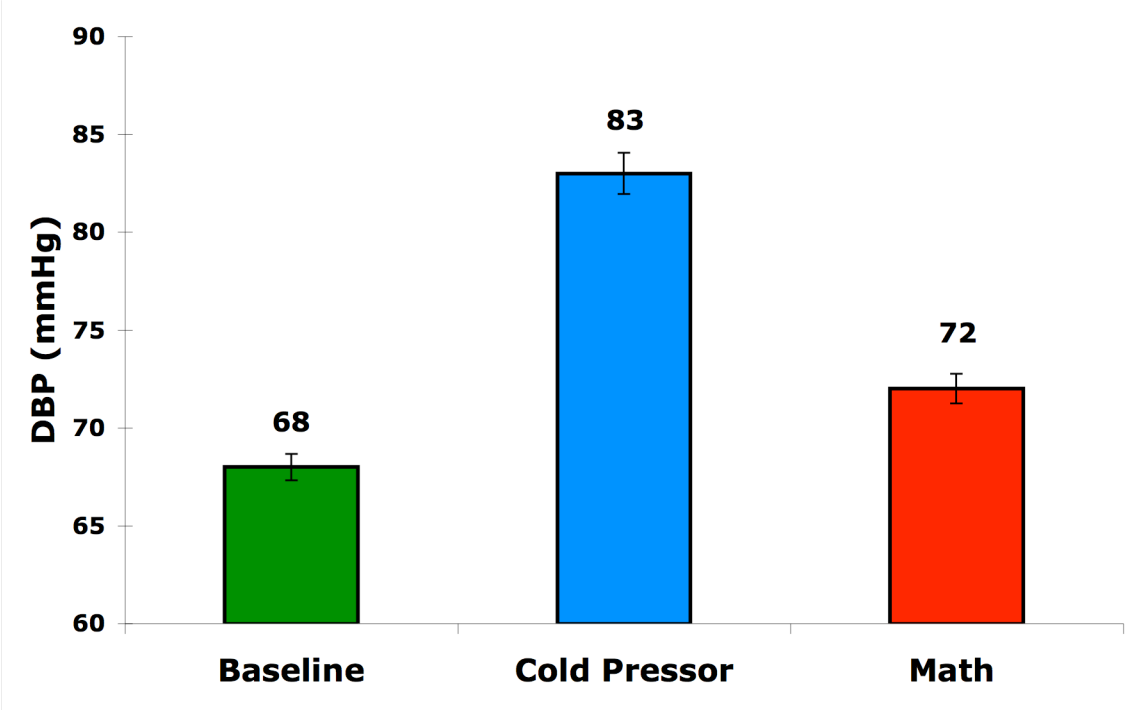


*Figure 2*. DBP during BL, CP and MA tasks.

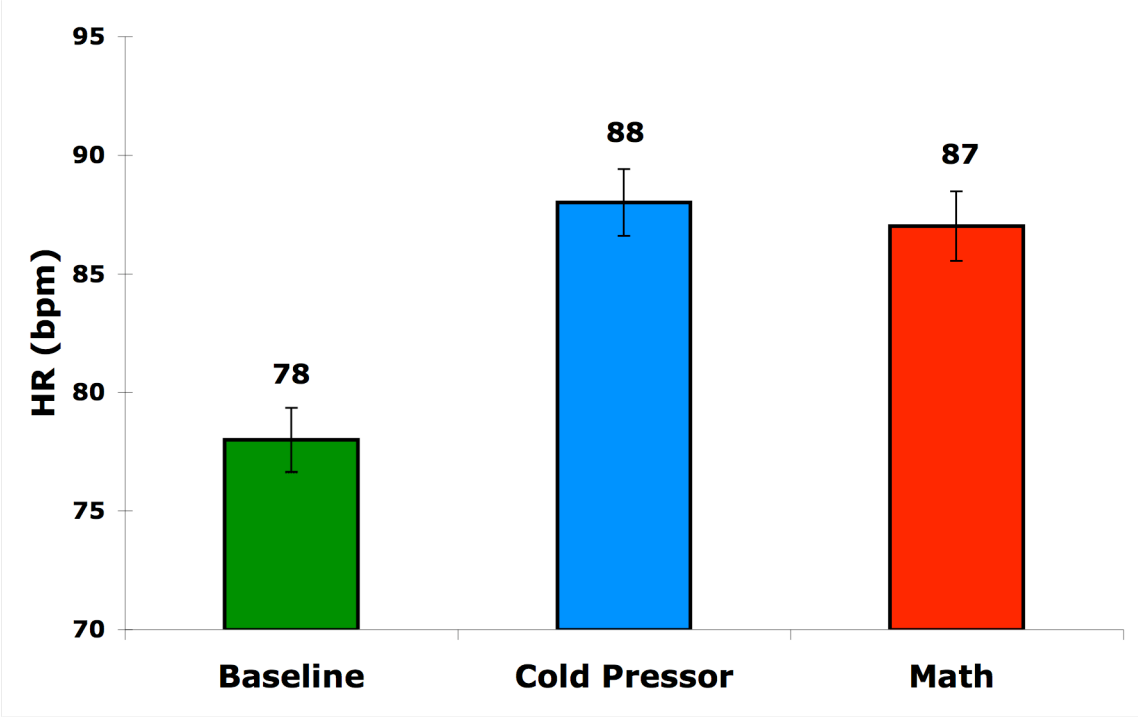


*Figure 3.* HR during BL, CP and MA tasks.

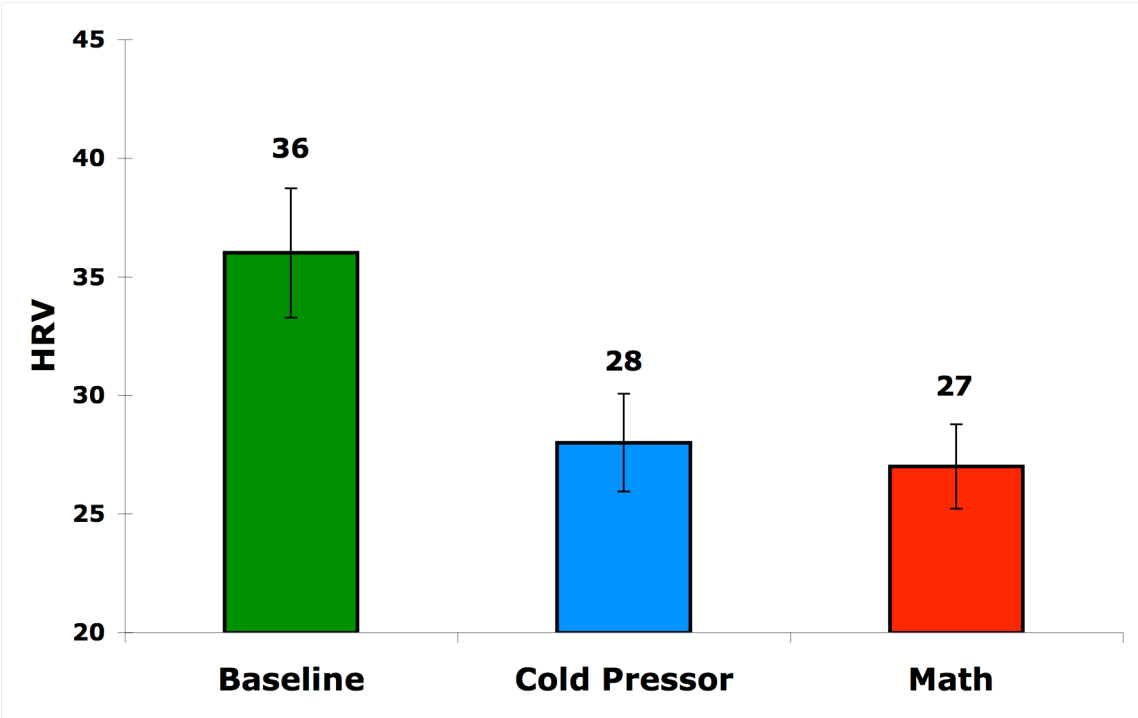


*Figure 4*. HRV during BL, CP and MA tasks.

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## VI. REFERENCES


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# VII. VITA

## EDUCATION

**Augusta State University**, Augusta, GA
December 2006
Overall GPA (for all degrees): 3.57, Cum Laude
- Bachelor of Arts in Psychology (B.A.)
- Bachelor of Social Work (B.S.W.)
- Associate of Applied Science: Criminal Justice (A.A.S.)

## HONORS/AWARDS

Graduate School Honors Fellowship
HOPE Scholarship
Dean's List

## WORK EXPERIENCE

**The University of Mississippi** November 2009 – Present
Office of Research and Sponsored Programs, University, MS
***Graduate Student Assistant***
Develop and maintain a database of protocols, process new protocols, protocol amendments, annual updates, progress reports, and protocol terminations, develop and edit policies and procedures, attend and participate in weekly IRB Executive Committee meetings, maintain the weekly and monthly activity reports

**The University of Mississippi** August 2009 – Present
Department of Psychology, University, MS
***Graduate Research Assistant***
Maintain ongoing studies, develop new protocols, maintain laboratory equipment, write and submit Institutional Review Board applications and amendments, train and supervise undergraduate research assistants, evaluate experimenter consistency, schedule participants, data entry and analysis

**The University of Utah** September 2007 – July 2009
Department of Neurology, Salt Lake City, UT
***Study Coordinator***

Co-investigator on a study measuring nerve regeneration capacity, assisted in protocol development and Institutional Review Board submission, participant recruitment, screening, data gathering and entry, meeting with site investigators, and performing physiological tests on study participants for multiple studies

**The Medical College of Georgia** October 2005 – May 2007
Department of Physiology, Augusta, GA
***Research Technician in Cardiovascular Research Lab, focusing on stroke and hypertension***
Developed Excel spreadsheets for analyzing blood pressure data, developed and wrote a protocol for using non-invasive blood pressure system, calculated dosages and administered experimental treatments to rats, performed enzyme immunoassays, western blots and wire and pressure myography, editing manuscripts

## TEACHING EXPERIENCE

General Psychology Honors Course, Guest Lecturer 2011
History and Systems of Psychology, Guest Lecturer 2011

## PUBLICATIONS

### Peer-Reviewed Journal Publications

Allen, M. T., Bocek, C. M., & **Burch, A. E.** (2011). Gender differences and the relationships of perceived background stress and psychological distress with cardiovascular responses to laboratory stressors. *International Journal of Psychophysiology*, 81(3), 209-217.

Rigsby CS, **Burch AE**, Ogbi S, Pollock DM, and Dorrance AM. Intact female stroke-prone hypertensive rats lack responsiveness to mineralocorticoid receptor antagonists. *Am J Physiol Regul Integr Comp Physiol* 293: R1754-1763, 2007.

### Conference Abstracts

Singleton JR, Marcus RL, Smith SB, Arsenault C, **Burch A**, Smith AG. (April, 2008). *Structured exercise improves small fiber function in diabetic subjects without overt neuropathy*. Annual American Academy of Neurology (AAN) Meeting, Chicago, IL.

Smith AG, Bixby B, **Burch A**, Arsenault CJ, Singleton JR. (April, 2008). *Diagnosis of early diabetic neuropathy.* Annual American Academy of Neurology (AAN) Meeting, Chicago, IL.

## PROFESSIONAL SERVICES

Institutional Review Board (IRB) Member 2011 – Present
Regional Science Fair Judge 2011, 2012
Psychology Colloquium, The University of Mississippi 2009 – Present
Neurology Weekly Journal Club Member, The University of Utah 2007 – 2009

Physiology Weekly Journal Club Member, The Medical College of Georgia 2005 – 2007

## MEMBERSHIPS AND AFFILATIONS

Society for Psychophysiological Research
Psi Chi