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Stress task specific impairments of cardiovascular functioning in obese participants

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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. Operationalizing chronic stress as obesity, we sought to explore the relationship between obesity, perceived stress and cardiovascular reactivity and recovery from laboratory stressors. Cardiovascular function was assessed using blood pressure and heart rate. Two stress tasks (mental arithmetic and cold pressor) were employed to examine potential differences between type of stress and cardiovascular response. Body mass index (BMI) was able to predict dysfunction in both cardiovascular reactivity and recovery. Participants with a higher BMI exhibited blunted systolic blood pressure and heart rate reactivity to the mental arithmetic task. In contrast, BMI has an incongruent effect on blood pressure reactivity to the cold pressor task that is dependent on the level of perceived stress. This suggests that in some instances the effect of BMI on cardiovascular response to acute stress may be moderated by perceived stress. Further, we found greater adiposity was related to delayed heart rate recovery following both stress tasks.

1. Introduction

Obesity is a rapidly spreading epidemic associated with devastating health outcomes including an increased risk for premature death (Katzmarzyk et al., 2003). The pathology of obesity has been shown to increase the likelihood of cardiovascular complications including congestive heart failure, stroke, myocardial infarction, hypertension, diabetes, dyslipidemia, atherosclerosis and peripheral vascular disease (Abel et al., 2008; Kenchaiah et al., 2002; Lakka et al., 2001; Mensah, 2004; Stapleton et al., 2008; Wang and Nakayama, 2010; Wilson et al., 2002). Obesity is characterized by an excess of adipose tissue, which promotes inflammation that can hinder normal cardiovascular functioning (Berg and Scherer, 2005). More specifically, adipokines, which are signaling molecules secreted by adipose tissue, can impair nitric oxide synthase resulting in inflammation and abnormal endothelial functioning (Van Gaal et al., 2006). The role that adipose tissue plays in chronic inflammation reinforces its importance as an independent risk factor in cardiovascular dysfunction.

When presented with a stressful situation, generalized arousal of the sympathetic nervous system is often helpful in managing the stressor. In some people, the arousal or reactivity of the sympathetic nervous system is greater than what is thought to be normal. The reactivity hypotheses as conceptualized by Obrist (1981) and others suggest that exaggerated physiological response to acute stressors could lead to adverse cardiovascular endpoints such as hypertension or coronary heart disease (Krantz and Manuck, 1984). A number of studies confirm that an exaggerated cardiovascular response to acute stress is a risk factor for cardiovascular disease (Lovallo and Gerin, 2003; Matthews, 2004; Schwartz, 2003).

Interestingly, more recent findings suggest 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 has been found in obese subjects, such that as body mass index (BMI) increased, heart rate (HR) response to acute stress decreased (Carroll et al., 2008; Phillips, 2011; de Rooij, 2013). A comprehensive review of the literature by Lovallo (2011) 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 was considered healthier. The review emphasizes the importance of diminished as well as exaggerated physiological reactions to stress as predictors of poor health outcomes. It is suggested that an intermediate degree of cardiovascular response to stressors may be most adaptive in dealing with challenges. A compilation of articles examining this previously overlooked continuum of reactivity was recently published in a special edition of the *International Journal of Psychophysiology* (Phillips et al., 2013), as well as a commentary on the articles in this special edition (Allen, 2013).

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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. Quick and sufficient response to stress is an integral part of survival and imperative to human health. However, the importance of the stress response should not overshadow the significance of effective recovery following the termination of a stressor (Geurts, and Sonnentag, 2006; Linden et al., 1997). Complications in this continuum of activation and recovery can lead to devastating consequences. Although recovery is often overlooked in studies of cardiovascular psychophysiology, a review of the literature suggests recovery from stress may illuminate disordered cardiovascular functioning, even when reactivity does not appear to yield impairments (Chida and Hamer, 2008). A meta-analysis by Chida and Hamer (2008) demonstrated that elevated general life stress resulted in slowed recovery from laboratory stressors. Exposure to chronic stress has also been associated with poor recovery from acute stressors (Gump and Matthews, 1999; Lepore et al., 1997). Obesity has also been found to have a relationship with impaired recovery (Brydon, 2011; Steptoe and Wardle, 2005). Although a return to individual baseline parameters eventually does occur, the delayed recovery instigates an overexposure to stress hormones, resulting in adipose tissue accumulation and excessive demands on the cardiovascular system (Benson et al., 2009; Bjorntorp, 2001; Steptoe and Wardle, 2005). Although relationships between obesity and cardiovascular reactivity and recovery from acute stress have been found, the precise mechanisms linking obesity to an impaired cardiovascular stress response remain unknown.

Circulating levels of adipokines and other proteins resulting in inflammation have an impact on vascular functioning; evidence suggests there may be an impairment of adrenergic receptor action (Hou and Luo, 2011; Larson et al., 2011). Although the relationship between obesity and its inflammatory assault on the vascular system is well documented (Berg and Scherer, 2005; Davy and Hall, 2004; Stapleton et al., 2008; Vachharajani and Granger, 2009; Van Gaal et al., 2006), the relative infancy of research on obesity and hemodynamic functioning creates a need to further define patterns of acute stress reactivity and recovery in obese subjects (Brydon, 2011; Carroll et al., 2008; Phillips, 2011; Steptoe and Wardle, 2005). The cardiovascular response to laboratory stressors designed to differentiate between pathways of adrenergic activation may uncover obesity specific impairments in reactivity and recovery. The current study seeks to explore the relationship between obesity and cardiovascular response to laboratory stressors, including recovery from stress.

2. Methods

2.1. Participants

Participants were 116 female undergraduate students at the University of Mississippi who were recruited from the subject pool for the course in general psychology; they received credit for the required research participation component of the course. Each participant read and signed an informed consent form; the Institutional Review Board of the University of Mississippi approved the form and the study. Smokers and anyone with a history of cardiovascular disorders or diagnosis of Reynaud's disease were excluded from the study. Equipment problems and failure to answer all items on questionnaires reduced the sample size on some of the measures. The self-reported ethnicity breakdown was 66 Caucasians, 38 African-Americans, 1 reported as "Mixed," 5 reported as "Other Ethnicity," and 6 whose ethnicity was not given. The means and standard deviations for age, height, weight and BMI are given in Table 1.

2.2. Physiological recording apparatus

Blood pressure (BP) was monitored using a model Tango automated BP monitor (SunTech Medical Instruments, Raleigh, NC, USA). An occluding cuff was placed on the left arm so that the sensor was placed over an area on the inner aspect of the upper arm where the brachial artery could be palpated. The monitor measured systolic (SBP) and diastolic blood pressure (DBP) using the oscillometric method. The monitor also determined HR during periods of cuff inflation.

Table 1
Means and standard deviations (SD): age, height, weight, Body Mass Index (BMI), Percieved Stress Scale (PSS) and task stressfulness ratings.

	Mean	SD	Range (Min-Max)
Age (years)	19	1.2	18 - 26
Height (inches)	63.9 (1.6 m)	2.3 (0.01 m)	58 - 70
Weight (lbs.)	140.7 (63.8 kg)	35.5 (16.1 kg)	81 - 290
BMI	24.2	5.9	14.6 - 43.7
PSS	16.5	6.0	2 - 29
CP Task Stressfulness	5.6	2.4	0 - 9
MA Task Stressfulness	4.6	2.1	0 - 9

Notes: CP = cold pressor, MA = mental arithmetic.

2.3. Experimental tasks

2.3.1. Cold pressor (CP)

Participants were asked to place their right hand in a container filled with ice and ice water. Water temperature was measured using a digital thermometer and maintained at approximately 0 degrees Celsius. The participants were 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 participants were asked to keep the water at a horizontal level across the wrist just above this bone. The task lasted for 90 seconds.

2.3.2. Mental arithmetic (MA)

Participants were given the number 1764 and asked to subtract by 13 s from that number as quickly as possible. The experimenter kept a neutral demeanor, corrected the participant when an incorrect response was given, and had minimal eye contact with the participant in order to not give encouragement to the participant. This was done to standardize the administration of the task across participants, and to presumably keep the stress level higher. The task lasted for 3 minutes.

2.4. Questionnaires

2.4.1. Perceived Stress Scale-10 (PSS)

Cohen et al. (1983) developed the 14-item PSS to index the degree to which respondents found their lives to be unpredictable, uncontrollable, and overloaded. The PSS-10 is a shortened, 10-item scale that was recommended by Cohen and Williamson (1988) due to its tighter factor structure and somewhat better internal reliability as compared to the 14-item version. Items on the PSS are scored on a 5-point scale ranging from 0 (never) to 4 (very often). Respondents are asked about their feelings and thoughts related to stress over the past month. The measure was designed for use with community samples. A Cronbach's alpha of .89 for the PSS was calculated for a previous study using a similar population, indicating good internal consistency (Allen et al., 2011).

2.4.2. Task Stressfulness Rating

A 10-point Likert-type scale ranging from 0 (not stressful) to 9 (extremely stressful) was given to participants to assess the perceived stress produced by each laboratory stressor. The stressfulness rating scale was administered immediately following each stress task.

2.5. Procedure

Participants reported to the testing site and were asked to read and sign the consent form. Anthropometric measures, which included height and weight were taken. BMI was then calculated by dividing