Abdominal obesity and chronic stress interact to predict blunted cardiovascular reactivity

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Abstract
Abdominal obesity and chronic stress have independent effects on cardiac autonomic regulation, and may also interact to influence cardiovascular reactivity. In addition to main effects, we hypothesized that abdominal obesity and chronic stress would interact and predict blunted cardiovascular reactivity. One hundred and twenty-two undergraduate students engaged in two stressful laboratory tasks while cardiovascular activity was assessed. Results indicated that higher abdominal obesity significantly predicted blunted systolic blood pressure (SBP) and mean arterial pressure (MAP) change, while chronic stress was not directly associated with any measure of cardiovascular reactivity. Furthermore, there was a significant interaction between abdominal obesity and chronic stress on SBP and MAP change such that among participants with higher chronic stress, higher abdominal obesity was significantly associated with reduced SBP and MAP reactivity. In addition, body-mass index (BMI), a measure of overall obesity, also had both main and interaction effects with chronic stress to predict blunted cardiovascular reactivity. These results suggest that abdominally obese individuals may incur difficulty in mounting appropriately-sized cardiovascular responses during acute stress, particularly when under high levels of chronic stress.

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1. Introduction

Rates of obesity have been increasing dramatically worldwide. Its prevalence has doubled in the United States (Stein and Colditz, 2004) and tripled in developing countries (Hossain et al., 2007) since the 1970s. As prevalence rates increase, the health and economic costs of obesity continue to mount. In the United States, the direct and indirect cost of obesity is estimated to be over 215 billion dollars per year (Hammond and Levine, 2010). Excessive weight gain is closely tied to the rising prevalence of a number of chronic diseases, including cardiovascular disease, type 2 diabetes, stroke, and cancer (Hossain et al., 2007). Whereas generalized obesity is characterized by an overall accumulation of adipose tissue spread throughout the body, abdominal obesity refers to high levels of fat accumulation centered primarily in the abdominal region. Although these two forms of adiposity have been demonstrated to be highly correlated (Goldbacher et al., 2005), they may also exist independently of each other. For example, some evidence suggests that being lean but having higher abdominal fat is especially associated with risk for earlier death (Larsson et al., 1984). Also, obese individuals may possess high levels of adipose tissue throughout the entire body in the absence of a relatively high accumulation of centralized abdominal fat. In addition, the measurement of both generalized and abdominal obesity also differ. Generalized obesity is typically measured by body-mass index (BMI), a measure based on an individual’s weight and height, with obesity defined as a BMI of 30 kg/m² or higher. Abdominal obesity can be measured using a variety of methods that are either invasive (e.g., DEXA scan) or non-invasive (e.g., measuring tape). Non-invasive assessment of abdominal obesity typically includes either the measurement of individuals’ waist-circumference (WC) alone, or the assessment of both WC and hip-circumference (HC) to calculate the ratio of the circumference of the waist to the hips, commonly known as waist-to-hip ratio (WHR).

Abdominal obesity confers additional health risks above and beyond those associated with generalized obesity, including an increased risk of hypertension, left ventricular dysfunction, coronary heart disease, diabetes, lipoprotein alterations and overall cardiovascular disease morbidity (Ammar et al., 2008; Despres et al., 1990; Lee et al., 2008). These associations are independent of other contributing risk factors such as high cholesterol, smoking, education, race, age, and obesity defined by BMI (Freedman et al., 1995). Although abdominal obesity has been recognized as a significant risk factor for chronic disease, the mechanisms by which it may lead to negative health outcomes remain poorly understood. Cardiovascular reactivity has been suggested as a potential mechanism linking abdominal...
obesity and pathophysiological processes. For example, some studies have demonstrated a positive association between abdominal obesity and cardiovascular responses to mental stress (Barnes et al., 1998; Davis et al., 1999; Goldbacher et al., 2005; Steptoe and Wardle, 2005; Waldstein et al., 1999). An exaggerated cardiovascular stress response has been found to be associated with future negative health outcomes, including clinical and sub-clinical cardiovascular disease (Treiber et al., 2003), hypertension (Light et al., 1992), increased left-ventricular mass (Allen et al., 1997), and carotid atherosclerosis (Barnett et al., 1997).

Thus, an exaggerated cardiovascular response to mental stress, which has been associated with abdominal obesity, may be a contributing factor linking abdominal obesity to pathophysiological processes. In contrast, although seemingly counter-intuitive, emerging evidence suggests that blunted cardiovascular reactivity may also be a risk factor for negative health outcomes, including immunological suppression, depression, and poor self-reported health (Phillips, 2011).

Abdominal obesity has been associated with both exaggerated (Barnes et al., 1998; Davis et al., 1999; Goldbacher et al., 2005; Steptoe and Wardle, 2005; Waldstein et al., 1999) and blunted (Carroll et al., 2008; Hamer et al., 2007; Laederach-Hoffmann et al., 2000; Phillips, 2011; Phillips et al., 2012) cardiovascular reactivity. There are a number of factors that may contribute to the mixed results. First, existing studies differ vastly in sample characteristics, selection criteria and statistical covariates included in their designs and analyses. Participant characteristics, such as age, gender, smoking status, medication, age, alcohol, smoking status, and baseline cardiovascular activity have been found to significantly impact cardiovascular reactivity (Lawler et al., 1995; Mills and Dimsdale, 1991; Straneva et al., 2000; Uchino et al., 1999) and are infrequently included as covariates across studies. Carroll et al. (2008) found that when only controlling for baseline cardiovascular measures, generalized obesity was positively associated with diastolic blood pressure (DBP) reactivity and negatively associated with heart rate (HR) reactivity. In addition, abdominal obesity, measured with WHR, was positively associated with SBP and DBP reactivity and negatively associated with HR reactivity. Interestingly, after controlling for the effects of age, cohort, sex, occupational group, and strenuous physical exercise 24 h before their scheduled appointment, the study was approved by the University Institutional Review Board. All participants provided informed consent before partaking in the study.

2.2. Measures

2.2.1. Anthropometric measures

Height and weight were measured using a scale. BMI was calculated as weight in kilograms (kg) divided by height in meters squared (kg/m²). In addition, WC, used as the primary index of abdominal obesity in the current study, is a noninvasive measure that is most predictive of cardiovascular events (Maffei et al., 2001) and strongly correlated with DEXA scan measures of abdominal obesity (Daniels et al., 2000; Pouliot et al., 1994). The WC was measured in centimeters (cm) at the level of the umbilicus with a flexible tape. In addition, HC was measured at the point of most intrusion on the hips, and WHR was calculated as WC divided with HC. To reduce measurement error, both WC and HC were measured twice, and the averaged values were used.

2.2.2. Chronic stress

The Inventory of College Students’ Recent Life Experiences Scale (ICSRLE) was used to measure chronic stress (Kohn et al., 1990). This 49-item measure was designed to measure stress in multiple domains most relevant to college life, including academic, social mistreatment, time pressure, friendship, and romantic stress. It has been validated in college students and demonstrated high internal consistency reliability (Cronbach’s α = .89) (Kohn et al., 1990).

Stress currency, duration, and intensity each may have unique effects on cardiovascular reactivity (Lepore et al., 1997; Matthews et al., 1997). Because the original ICSRLE does not measure currency and duration, it was modified to include these dimensions in addition to stress intensity. For stressors endorsed as being currently experienced, participants were asked to indicate the number of months they had been dealing with the event and the level of intensity of the stressor. Two subscales, stress duration and intensity, were produced for the chronic
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