International Journal of Psychophysiology 90 (2013) 73–79

Contents lists available at ScienceDirect

International Journal of Psychophysiology

journal homepage: www.elsevier.com/locate/ijpsycho

Abdominal obesity and chronic stress interact to predict blunted cardiovascular reactivity☆

Kulwinder Singh [a,*], Biing-Jiun Shen [b,1]

[a] Department of Psychology, Seeley G. Mudd Building Room 501, University of Southern California, 3620 South McClintock Ave., 90089-1061, Los Angeles, CA, USA
[b] Department of Psychology, Ohio University, 245 Porter Hall, 45701, Athens, OH, USA

ARTICLE INFO

Article history:
Received 10 November 2012
Received in revised form 7 March 2013
Accepted 18 March 2013
Available online 25 March 2013

Keywords:
Cardiovascular reactivity
Blunting
Abdominal obesity
Chronic stress
Body-mass index

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.

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 differs. 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/m^2 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

☆ We would like to thank the following students and colleagues for their assistance with data collection: Stacy Eisenburg, Uta Maeda, Emily Kamen, Jessica Matlock, Jon Arrington, Kelsey Rupp, Farva Jafri, and Danny Liu.

* Corresponding author. Tel.: +1 415 246 0394; fax: +1 213 746 9082.
E-mail addresses: Kulwinds@usc.edu (K. Singh), Shenb@ohio.edu (B.-J. Shen).
[1] Tel.: +1 740 593 1086; fax: +1 740 593 0579.

0167-8760/$ – see front matter © 2013 Elsevier B.V. All rights reserved.
http://dx.doi.org/10.1016/j.ijpsycho.2013.03.010

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 shown 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-Hofmann 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, medications, oral contraceptive use, 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, Paced Auditory Serial Addition Test performance scores (a test involving working memory and divided attention), medication, smoking status, and baseline cardiovascular levels, previously observed positive associations between cardiovascular reactivity, generalized obesity, and abdominal obesity became non-significant, while all negative associations remained. In general, studies with comprehensive statistical adjustment and stringent exclusion criteria mostly have reported negative associations between cardiovascular reactivity and abdominal obesity (Hamer et al., 2007; Laederach-Hofmann et al., 2000; Phillips, 2011), although some have continued to report positive associations (Goldbacher et al., 2005; Steptoe and Wardle, 2005). The mixed findings strongly suggest that additional factors may help explain the relationship between abdominal obesity and cardiovascular reactivity.

It has been suggested that chronic stress may be one of these factors, as it may have particularly strong physiological consequences among individuals who are abdominally obese (Björntorp, 2001; De Vriendt et al., 2009; Shen et al., 2010). Chronic stress has been consistently associated with impaired hypothalamic–pituitary–adrenal axis (HPA) and central sympathetic nervous system functioning (Miller et al., 2007; Pike et al., 1997). Excessive fat in tissues is also associated with elevations in glucocorticoids and catecholamines (Grassi et al., 2004; Marin et al., 1992). Individuals with high levels of chronic stress and excessive abdominal fat centralization demonstrate persistent elevations in glucocorticoid and catecholamine activity (Björntorp, 1996, 2001; McEwen, 1998), which may influence the body's ability to maintain allostasis. Under high allostatic load, the physiological "wear and tear" from repeated activation of the stress response (McEwen and Seeman, 1998), the individual's physiological stress systems may become "burned out" and less responsive to environmental demands (Björntorp, 2001; McEwen, 1998). This "burned out" state may be reflected by a diminished cardiovascular response to stress (McEwen and Seeman, 1998).

The primary goal of this study was to examine the relationship between abdominal obesity, chronic stress, and cardiovascular reactivity in a sample of healthy young adults. We first aimed to examine whether these characteristics influenced baseline cardiovascular activity. We also aimed to examine whether chronic stress and abdominal obesity would interact to predict blunted cardiovascular reactivity. We hypothesized that in addition to their main effects, abdominal obesity and chronic stress would interact to predict a profile of blunted cardiovascular reactivity across multiple cardiovascular measures. In addition, we examined patterns of cardiovascular responding across other measures of obesity, and whether chronic stress interacted with these measures to predict a blunted cardiovascular response.

2. Method

2.1. Participants

One hundred and twenty-two undergraduate students were recruited for this study. Participants were 35 male and 87 female students between the ages of 18 and 28 ($M = 19.9$, $SD = 1.68$) who met the study inclusion criteria. The exclusion criteria were selected on the basis of factors known to impact cardiovascular reactivity (Mills and Dimsdale, 1991; Straneva et al., 2000). These included: currently consuming medications that affect the cardiovascular system, currently smoking or using nicotine products, use of oral contraceptives, use of illicit substances, and current or past history of chronic illness. Eligible participants were instructed to abstain from caffeine, alcohol 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/m^2). 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 (Maffeis 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 $\alpha = .89$) (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