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Blunted cardiovascular reactivity relates to depression, obesity, and self-reported health

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ABSTRACT

The reactivity hypothesis implicates exaggerated cardiovascular reactions to acute psychological stress in the development of hypertension and other cardiovascular disease outcomes. However, cardiovascular reactivity has also been suggested as a mediator between a variety of psychosocial and behavioural risk factors and cardiovascular disease. Data from various analyses of the West of Scotland Twenty-07 study are discussed together, and caution against over-stretching the original reactivity hypothesis. Blood pressure and heart rate were assessed at rest and during an acute mental arithmetic stress task. First, depression, though a putative risk factor for cardiovascular disease, does not appear to confer this risk via exaggerated reactivity, as it was negatively related to reactivity. Second, obesity, another risk factor, was also associated with blunted rather than heightened reactivity. Finally, lower reactivity was related to poorer self-reported health. Similar associations emerged from both cross-sectional and prospective analyses. These seemingly paradoxical results are discussed in terms of implications for the reactivity hypothesis.

1. Introduction

The reactivity hypothesis, as originally conceptualised, proposed that large magnitude cardiovascular reactions to acute psychological stress exposures increase the risk of the development of hypertension (Obrist, 1981). Studies testing the hypothesis have shown that high reactivity is associated with higher blood pressure; the most compelling evidence emerges from prospective studies, where an initial assessment of cardiovascular reactivity is followed by measurement of blood pressure status in the future. For example, in the Twenty-07 study in the West of Scotland, the magnitude of cardiovascular reactions to stress predicted 2–3% of the variance in 5-year upward drift in blood pressure, when controlling for initial resting blood pressure as well as body mass index (Carroll et al., 2003). A number of large-scale studies in a range of populations also attest to a reliable positive association between the magnitude of cardiovascular reactions to acute psychological stress tasks and future blood pressure status (Carroll et al., 2001; Markovitz et al., 1998; Treiber et al., 1997). The additional risk associated with high cardiovascular reactivity has also been shown to extend to other cardiovascular outcomes including carotid atherosclerosis, carotid intima thickness, and increased left ventricular mass/cardiac hypertrophy (e.g. Everson et al., 1997; Georgiades et al., 1997; Kamarck et al., 1997; Matthews et al., 2006; Treiber et al., 2003).

Recently, high cardiovascular reactivity has also been suggested as a mediator between a wide variety of psychosocial and behavioural risk factors and cardiovascular disease (Chida and Hamer, 2008), such as life events stress (Lepore et al., 1997; Matthews et al., 1997), social support (O'Donovan and Hughes, 2008; Roy et al., 1998), hostility (Suls and Wan, 1993; Vella and Friedman, 2009), and smoking (Davis and Matthews, 1990; Tersman et al., 1991). An obvious corollary of the reactivity hypothesis is that low reactivity during acute stress is an adaptive response and protective against cardiovascular disease. By extension, then, low reactivity would be expected to characterise more positive psychological and behavioural factors implicated in mitigating cardiovascular disease risk. However, it is from research examining aspects of this expanded role for reactivity that a number of seemingly paradoxical findings are beginning to emerge. Analyses from the West of Scotland Twenty-07 study will be used to illustrate that caution is warranted when attempting to stretch the reactivity hypothesis beyond its original postulates. First indications that supposedly negative characteristics or behaviours might not always be associated with higher cardiovascular reactivity emerged from analyses of the associations between life events stress and reactivity in the Twenty-07 study data. For example, socio-economic status was positively associated with acute stress reactivity such that those from manual occupational households had lower cardiovascular responses to acute stress than those from non-manual

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households (Carroll et al., 2000). Similarly, given that acute laboratory stress responses are considered to be indicative of how one responds to stressful events in real life, it was expected that those with higher stress ratings for events which had occurred over a 2-year period, would have greater cardiovascular responses to laboratory stress. However, data from the Twenty-07 study showed that middle-aged and older adults who rated their stressful experiences as more highly disruptive, at the time of occurrence and at the time of recall, exhibited blunted systolic blood pressure reactions to acute mental stress (Carroll et al., 2005). Likewise, among the younger adults in the Twenty-07 study, the total number of events and the number of personal events were negatively associated with systolic blood pressure and heart rate reactions to acute stress, whereas the number of work-related events was negatively associated with diastolic blood pressure and pulse rate reactivity (Phillips et al., 2005b).

The present article will address the scope and the limitations of the reactivity hypothesis through discussion of both cross-sectional and prospective associations between cardiovascular reactivity to acute stress and three key psychosocial/behavioural risk factors; depression, obesity, and self-reported health, in a large community-based sample.

1.1. Depression

Depression has been linked prospectively with mortality from cardiovascular disease (Hemingway and Marmot, 1999; Wulsin et al., 1999). The mediators and mechanisms underlying this association have yet to be established, but might include factors such as socio-economic position; ill-health and disability; unhealthy behaviours (Wulsin et al., 1999); increased platelet aggregation (Mikuni et al., 1992); and exaggerated cardiovascular reactions to psychological stress exposure (Kibler and Ma, 2004). With regard to this latter possibility, depression has been associated with a variety of physiological adaptations that suggest altered autonomic function. For example, enhancement of cardiac sympathetic activity relative to vagal tone has been reported in those with depression and sub-clinical depressive symptoms (Carney et al., 1988; Light et al., 1998), as have increased plasma noradrenalin concentrations in patients with major depression (Rudorfer et al., 1985). Thus, the hypothesis that such autonomic dysregulation in depression may also be manifest as exaggerated cardiovascular reactivity, which in turn increases the risk of cardiovascular pathology, is intuitively appealing. There would appear to be at least some provisional evidence that symptoms of depression may be associated with heightened reactivity. For example, in a study of 91 healthy participants, those with high amounts of depressive symptoms manifested significantly greater systemic vascular resistance in response to a stressor task than did those with low amounts of depressive symptoms (Matthews et al., 2005). A meta-analysis of 11 relevant studies found small to moderate effect sizes indicative of a positive relationship between depressive symptomatology and cardiovascular reactions to acute psychological stress (Kibler and Ma, 2004). Unfortunately, none of the aggregate effects were statistically significant at conventional levels. Previous studies generally tested fairly small samples and few adjusted for potential confounding variables such as demographic factors and medication status. In contrast, in a larger sample of over 100 coronary artery disease patients, higher depressive symptom scores were associated with lower, not higher, cardiovascular reactions to acute psychological stress, even after controlling for a number of likely confounders (York et al., 2007). The West of Scotland Twenty-07 dataset was used to revisit this issue in a substantial and demographically diverse sample of participants (Carroll et al., 2007). Statistical adjustment for a range of possible confounders was possible. Uniquely, it was also possible to examine prospectively the association between cardiovascular reactivity and symptoms of depression 5 years later.

1.2. Obesity

Obesity is a fast growing epidemic in Western countries (Hughes et al., 2002; WHO, 1997). The adverse health consequences of this increase in adiposity are starting to become apparent. Obesity, defined in terms of a body mass index of 30 kg/m^2 or more, has been consistently linked to all-cause and especially cardiovascular disease mortality (Adams et al., 2006; Allison et al., 1999; Calle et al., 1999; Stevens et al., 1998). It is also associated with a range of cardiovascular and metabolic disease outcomes, such as type 2 diabetes (Ford et al., 1997; Resnick et al., 2000) and hypertension (Hirani et al., 2007; Mokdad et al., 2003), as well as overall cardiovascular disease morbidity (Bogers et al., 2007; Wilson et al., 2002). Abdominal adiposity has also been linked with psychological distress, and it has been argued that an increased vulnerability to stress in the abdominally obese may be manifest as physiological hyper-reactivity (Bjorntorp, 1991). The impact of stress on the neuroendocrine system is thought to promote abdominal fat deposition (Bjorntorp, 1996), and it has been suggested that obesity, and especially central adiposity, will be associated with exaggerated cardiovascular reactions to stress (Davis et al., 1999; Waldstein et al., 1999). The question arises as to whether obesity and exaggerated cardiovascular reactivity to acute stress are positively related or whether they are independent risk factors for cardiovascular pathology. A few mainly small-scale studies have attempted to address this issue. Systemic vascular resistance levels during mental stress were negatively correlated with body mass index in 20 young men, but positively associated with waist-hip ratio; no significant associations emerged for blood pressure or cardiac activity during stress (Jern et al., 1992). From a study of 95 adolescents, the peak systolic blood pressure (SBP) reaction to mental stress was larger for participants in the upper tertile of waist-hip ratios, although neither cardiac nor resistance reactions were associated with abdominal adiposity (Barnes et al., 1998). Waist circumference has been reported to be positively associated with heart rate (HR) and diastolic blood pressure (DBP) reactivity in a sample of 22 older African American men, but these associations did not withstand correction for basal blood pressure and insulin levels (Waldstein et al., 1999). In a contemporary study of 24 women with body mass indices $\geq$28 kg/m^2, those with abdominal obesity, i.e. high waist-hip ratios, had higher DBP and systemic resistance reactions, but lower HR reactions, to a speech task (Davis et al., 1999). In the largest study to date, body mass index was not significantly related to cardiovascular reactivity in 225 middle-aged public servants, although waist-hip ratio, a measure of abdominal adiposity, was positively associated with diastolic reactivity; the greater the abdominal adiposity, the higher the reactivity (Steptoe and Wardle, 2005). In addition, it was expected that disturbances in cardiovascular reactivity may reflect autonomic dysfunction which in turn may contribute to the development of obesity and adiposity, yet the upward drift in body mass index and waist-hip ratio over a 3-year follow-up period was not associated with the earlier measures of cardiovascular reactivity (Steptoe and Wardle, 2005). In contrast, greater fatness was related to a blunted vasodilatation response to mental stress in 48 healthy young men (Hamer et al., 2007).

It is difficult to draw firm confident conclusions from the results of these studies, particularly given that most samples were small and poorly representative of the general population, and few adjusted for potential confounding variables, including baseline cardiovascular levels. The most consistent result appears to be a positive association between systemic resistance reactivity, as reflected by DBP and/or total peripheral resistance, and abdominal adiposity, although not all studies report this. The West of