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Review

The other side of the coin: Blunted cardiovascular and cortisol reactivity are associated with negative health outcomes

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ABSTRACT

A cornerstone for research into the link between stress and health has been the reactivity hypothesis; cardiovascular reactivity to psychological stressors, if prolonged or exaggerated, can promote the development of cardiovascular disease. However, it has recently been argued that low or blunted reactivity is also associated with negative health outcomes. As such, in this special issue we present further evidence implicating that cardiovascular and stress hormone responses to acute stress at the other end of the response spectrum can also be considered a pathway to ill health. In this introductory article, we explore and review the origins of and potential mechanisms underlying blunted responses to acute stress. In so doing, we aim to highlight: what is currently known regarding this new conceptualization of the reactivity hypothesis; the potential explanations for blunted reactivity; the pathways underlying associations with health outcomes; and where this field is headed in terms of developing our understanding of the link between reactivity and health.

1. Introduction

A key focus of research into the link between stress and health for several decades has been the reactivity hypothesis, which proposes that cardiovascular reactivity to psychological stressors, if prolonged or exaggerated, can promote the development of cardiovascular disease (Obrist, 1981). Heightened cardiovascular reactivity is posited to result in changes to the structure and functioning of the heart that potentially promote a number of adverse disease outcomes, including sustained hypertension (Obrist, 1981) cardiac and vascular hypertrophy (Lovallo and Gerin, 2003), oxidation of low-density lipoproteins (Raitakari et al., 1997), and increased serum concentrations of both pro-inflammatory cytokines (Georgiades, 2007) and blood insulin (Nazzaro et al., 2002). Theoretical considerations of how negative health results from reactivity-related adaptations of the cardiovascular system have been corroborated by an extensive empirical literature linking heightened reactivity to objective outcomes, including hypertension (Carroll et al., 2003, 1995, 2001; Everson et al., 1996; Markovitz et al., 1998; Matthews et al., 1993; Newman et al., 1999; Treiber et al., 1997), atherosclerosis (Barnett et al., 1997; Everson et al., 1997; Lynch et al., 1998; Matthews et al., 1998), increased left ventricular mass and/or hypertrophy of the heart (Georgiades et al., 1997; Kapuku et al., 1999; Murdison et al., 1998), and cardiovascular disease mortality (Carroll et al., 2013). Both qualitative reviews and meta-analyses of this evidence confirm the contention that exaggerated stress reactions signal poor future cardiovascular health (Gerin et al., 2000; Chida & Steptoe, 2010; Schwartz et al., 2003; Taylor, Kamarck, & Dianzumba, 2003; Treiber et al., 2003).

Given the emphasis placed on associations between exaggerated reactivity and disease pathogenesis, low or blunted reactivity to acute stress has, by implication, been assumed to be benign or even protective. However, recent evidence suggests that low cardiovascular reactivity to stress, as well as low cortisol reactivity, may actually have serious adverse consequences for health and behaviour. For example, comparatively low cardiovascular and cortisol reactions to acute psychological stress have been found to characterize smokers (al'Absi, 2006; al'Absi et al., 2005; Girdler et al., 1997; Phillips et al., 2009b; Roy et al., 1994; Sheffield et al., 1997) and those with alcohol and other substance addictions (Brenner and Beauchaine, 2011; Lovallo, 2005; Lovallo et al., 2000; Panknin et al., 2002), as well as being associated with obesity (Carroll et al., 2008), depression (Brinkmann et al., 2009; Carroll et al., 2007; de Rooij et al., 2010; Phillips et al., 2011; Rottenberg et al., 2007; Salomon et al., 2009a; Schwerdtfeger and Rosenkaimer, 2011; York et al., 2007), poor self-reported health (De Rooij and Roseboom, 2010; Phillips et al., 2009a), exercise addiction (Heaney et al., 2011), eating disorders (Ginty et al., 2012a), and poorer cognitive function (Ginty et al., 2011a, 2011b, 2012b). Comparatively reduced cardiovascular stress responses have also been associated with personality traits also indicative of future disease, such as Type D personality (Howard et al., 2011) and neuroticism (Hughes et al., 2011), and other studies have found low reactivity among people taking medications known to increase heart disease risk, such as oral contraceptives (Schallmayer & Hughes, 2010). In light of accumulating evidence supporting an association between blunted stress reactivity and

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addiction, and the emerging evidence relating blunted reactivity to other unhealthy behaviours and negative health outcomes, it would appear that both extremes, exaggerated and diminished reactivity are maladaptive responses to stress. This suggests that the most optimally healthy response to stress is a moderate reaction (Lovallo, 2013). This paper will examine the concept of blunted responses to stress, their possible origins and underlying mechanisms as an introduction to this special issue on blunted reactivity to acute stress.

2. What are the bases of 'blunted' reactivity?

We take the term 'blunted cardiovascular reactivity' to refer to an empirically demonstrable cardiovascular response pattern which is comparatively lower than that seen during typical states of homeostatic function during stress. As yet, the precise mechanism that determines the occurrence of blunting is unclear, and an important distinction needs to be made between reactivity that is blunted (which implies sub-normality) and reactivity that is simply low (which may be biologically normal within a given context). There are several possible bases of blunted reactivity, which we outline below. Overall, it can be noted that while there are a number of possibilities, several remain to be tested empirically.

2.1. Lower effort

One possibility is that lower reactions to acute stress tasks may reflect *lower effort* on the part of a participant; in other words, that blunted responses are primarily the result of behavioural factors rather than of cognitive or biological factors. Perceptions of how stressful a psychological stress task is might impact upon an individual's willingness to engage with the task, with reduced effort or motivation then underlying a negative health impact. It could be argued that symptoms of conditions such as depression, which are associated with blunted responses, might also relate to lower motivation levels. Certainly, depression is characterised by a reduced degree of motivation, and both depression and low motivation appear to be related to the same gene polymorphisms, such as the met variant of the Val158Met COMT gene (Aberg et al., 2011), which results in different levels of extracellular dopamine within key brain reward system areas. Yet, in neuropsychological tests requiring cognitive effort, depressed participants have not consistently performed worse (Hammar et al., 2011). Further, not all of the behaviours or conditions associated with blunted responses to stress relate to lower motivation or effort; indeed some such as exercise dependence (Heaney et al., 2011) require considerable motivation and effort. Finally, in studies produced by the Birmingham group, we have always found blunted responses to be independent of ratings of task stressfulness and engagement, as well as objective performance scores which are a proxy for task engagement (see e.g. Ginty et al., 2012a; Heaney et al., 2011). Taken together, this evidence suggests that individuals characterised by blunted responses are not necessarily demonstrating lower task performance or perceptions of motivation. However, as performance is merely a proxy for effort, and not the same construct, it remains unclear whether or not the tendency to invest less effort accounts for trait-like patterns of blunted responses.

2.2. Reduced awareness or perception of stress

An alternative theory of blunted reactivity is that it might reflect a *reduced awareness or perception of stress*; in other words, that blunted responses are primarily the result of cognitive factors rather than of behavioural or biological factors. An inability to detect stressors in the environment, or a tendency to view dangerous stimuli as innocuous, would logically serve to dampen physiological stress responses. It has long been established (a) that perceptual factors are important in determining whether an individual exhibits a physiological stress response (Speisman et al., 1964) and (b) that individuals differ in the extent to which they habitually regard otherwise innocuous stimuli as negatively valenced (Bishop, 2008). It has also been established that training participants to bias their attention away from negative stimuli serves to dampen their cardiovascular responses to subsequent stressors (Higgins and Hughes, 2012), implying that such perceptions are causally responsible for cardiovascular response profiles rather than the other way around. Moreover, such effects are most pronounced among persons with high levels of trait neuroticism (Connor-Smith and Flachsbart, 2007), a group also characterised by blunted responses to stress (Phillips et al., 2005). As such, blunted cardiovascular responses may reflect diminutions in stress vigilance and/or detection, although this remains to be tested directly.

2.3. The impact of task difficulty

One possibility underlying blunted reactivity is that of the impact of task difficulty. In stress tasks which employ active coping, it has been shown that the extent of reactivity is related to the difficulty or challenge levels of the task, such that both easy and over-challenging tasks can result in weaker cardiovascular responses (Richter and Gendolla, 2006). When task difficulty is manipulated, as it increases, so too do the magnitude of cardiovascular responses until difficulty levels where success is impossible (Carroll et al., 1986; Richter et al., 2008), although this has not been shown in all studies (Willemsen et al., 2000). This is considered to be due to effects on motivational intensity in active coping situations (Wright, 1996). Task difficulty is also thought to interact with mood resulting in different levels of perceived subjective demand, which in turn determine the amplitude of cardiovascular reactivity (Gendolla and Krusken, 2001). Indeed these situational manipulations of perceived task demand have been replicated multiple times to show the impact of effort on cardiovascular responses to stress. However, other studies have revealed that in situations of matched task difficulty (Phillips, 2011), and across different types of tasks (de Rooij, 2013, in this special issue), there appears to be a sub-group of individuals who consistently show trait-like blunted responses to acute stress, independent of perceptions of tasks difficulty, see e.g., (Ginty et al., 2012a; Heaney et al., 2011). This brings us to the next potential explanation for blunting.

2.4. Reduced physiological capacity to respond

A fourth explanation for blunted reactivity is that the blunted responses may reflect a reduced physiological capacity to respond to stress or a disengagement of the biological systems, rather than of behavioural or cognitive factors. For example, there is reasonably consistent evidence that the sympathetic nervous systems of individuals who have become obese, although characterised by high basal sympathetic nervous system activity (Carroll et al., 2008; Tentolouris et al., 2006), and higher basal cortisol levels (Bjorntorp, 1993; Bjorntorp and Rosmond, 2000), are less responsive to stimulation (Carroll et al., 2008; Tentolouris et al., 2006). There is evidence of a postprandial sympathetic nervous system response, as indicated by higher plasma norepinephrine concentrations and an increased low to high frequency ratio in the HR variability spectrum after ingestion of a meal (Tentolouris et al., 2003; Welle et al., 1981). Similarly, this is smaller among obese individuals than the non-obese (Tentolouris et al., 2003). In addition, the changes in HR and muscle sympathetic nerve stimulation following infusion of anti-hypertensive and anti-hypotensive drugs are significantly smaller in the obese than the non-obese (Grassi et al., 1995), consistent with the view that such blunted responses result from biologically-based attenuation. A further example is that of depression; where basal sympathetic nervous system activity, as indexed by a shift enhanced cardiac sympathetic activity relative to vagal tone (Carney et al., 1988), increased plasma noradrenaline concentrations (Rudorfer et al., 1985), and increased 24-hour urinary noradrenaline excretion (Hughes et al., 2004) has been shown to be higher in individuals with depression or