Neuroscience and Biobehavioral Reviews 77 (2017) 74–86

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

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

Review article

The behavioural, cognitive, and neural corollaries of blunted cardiovascular and cortisol reactions to acute psychological stress

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ARTICLE INFO

Article history:
Received 5 August 2016
Received in revised form 6 February 2017
Accepted 26 February 2017
Available online 27 February 2017

Keywords:
Behaviour
Cognitive ability
Cardiovascular activity
Cortisol
Blunted stress reactivity
Health
Neural hypoactivation

ABSTRACT

Recent research shows that blunted cardiovascular and cortisol reactions to acute psychological stress are associated with adverse behavioural and health outcomes: depression, obesity, bulimia, and addictions. These outcomes may reflect suboptimal functioning of the brain's fronto-limbic systems that are needed to regulate motivated behaviour in the face of challenge. In support of this, brain imaging data demonstrate fronto-limbic hypoactivation during acute stress exposure. Those demonstrating blunted reactions also show impairments of motivation, including lower cognitive ability, more rapid cognitive decline, and poorer performance on motivation-dependent tests of lung function. Persons exhibiting blunted stress reactivity display well established temperament characteristics, including neuroticism and impulsivity, characteristic of various behavioural disorders. Notably, the outcomes related to blunted stress reactivity are similar to those that define Reward Deficiency Syndrome. Accordingly, some individuals may be characterised by a broad failure in cardiovascular and cortisol responding to both stress and reward, reflecting fronto-limbic dysregulation. Finally, we proffer a model of blunted stress reactivity, its antecedents and *sequelae*, and identify future research priorities.

Contents

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http://dx.doi.org/10.1016/j.neubiorev.2017.02.025
0149-7634/

1. Introduction

Most stressful encounters are short-lived, lasting seconds, minutes, hours at most, and what is now clear is that such acute stress exposures activate the sympatho-medullary (SAM) system, perturbing cardiovascular activity, and the hypothalamic pituitary-adrenal (HPA) axis, altering cortisol levels. Under optimal conditions, specific brain structures assess the threat and develop an adaptive pattern of autonomic and endocrine outflow to support the physiological and behavioural responses needed to meet the present homeostatic challenge (Lovallo, 2016; McEwen and Gianaros, 2011). Sympathetic nervous system responses to stress engage the cardiovascular system and adrenal medulla to produce a coordinated preparation for fight-or-flight efforts. Along with this, the hypothalamic-pituitary-adrenocortical system produces a significant rise in circulating cortisol which aids in liberation of stored energy and also helps to regulate the stress response in both the periphery and in the central nervous system. Stress responses resolve when the challenge has been eliminated. Autonomic, and endocrine control over stress responses may be altered in persons with particular genotypes or exposed to early-life adversity (Lovallo, 2016; McEwen and Gianaros, 2011). In such persons, physiological, behavioural, and cognitive processes may be altered, health behaviours biased toward maladaptive practices, and health outcomes worsened. More recent research has shown that, along with autonomic and endocrine modifications, early stress exposure may change the regulation of inflammatory processes with consequences for brain function, peripheral regulation of the stress response, and the ability of the system to manage immune system responses and tissue inflammation (Gianaros et al., 2014, 2013). The normal regulation of these stress responsive systems has been described in detail elsewhere (Lovallo, 2016; McEwen and Stellar, 1993). The goal of the present paper is to discuss the health and behavioural consequences of blunted cardiovascular and cortisol reactivity to stress.

As indicated, it is clear that individuals differ markedly in their cardiovascular and cortisol reactions to acute psychological stress. This biological variability has proved fertile territory for researchers over the years. For the most part, research, at least in the realm of cardiovascular stress reactions, has been guided by the reactivity hypothesis (Obrist, 1976) which argues compellingly that those who consistently show exaggerated cardiovascular reactions to acute stress will be at increased risk of subsequent cardiovascular disease. There is now substantial evidence in favour of this, with population studies attesting to a link between heightened cardiovascular reactions to laboratory stress exposures and hypertension (e.g., Carroll et al., 2011, 2003), atherosclerosis (e.g., Barnett et al., 1997), increased left ventricular mass (e.g., Kapuku et al., 1999), and even increased cardiovascular disease mortality (Carroll et al., 2012a). Qualitative reviews (e.g., Treiber et al., 2003) confirm the contention that exaggerated cardiovascular reactivity presages poorer cardiovascular health, although it should be conceded that effect sizes from the latter are generally small. By implication, low or blunted cardiovascular reactivity has long been presumed to be benign or even protective. However, recent evidence strongly indicates that this is far from the case. Blunted cardiovascular, as well as cortisol, stress reactivity is implicated in a range of adverse behavioural and health outcomes.

The perspective we are advancing is based on the critical role that normal stress responsivity plays in adaptive responses to the constant demands the environment places on survival. In this view, stress responses represent a systems-level response to threats to homeostasis. By extension, optimal responses to those threats require appropriate integration of the system at multiple levels including peripheral physiology, the brainstem and hypothalamus, and the cortex and limbic system. From this perspective, physiological responses to stress should occur within the normal range for a given homeostatic threat. By definition, deviations from a normative response may signal poor systems integration and therefore diminished homeostatic control. We consider that exaggerated stress responses have negative consequences for health, as described above. However, similarly blunted stress responses may also signal poor homeostatic regulation, with a different set of consequences for health and behaviour.

In this review, we also move toward a tentative model of blunted stress reactivity, by: first, enumerating its seemingly diverse behavioural and health corollaries, such as addiction, obesity, and poor self-reported health; second, by discussing the possible origins of blunted stress reactivity in genetic polymorphic variation and early life adversity; third, by suggesting possible pathways that link genetic inheritance and early adversity through sub-optimal functioning of brain areas central to both motivation and autonomic regulation to other more proximal corollaries of blunted stress reactions, such as relative poor cognitive ability and adverse behavioural phenotypes including depression, neuroticism, and poor impulse control; fourth, by considering these as increasing the risk of the more distal outcomes such as addiction, obesity, behaviour disorders, and poor self-reported health. This model regards blunted cardiovascular and cortisol stress reactivity, not as a cause, but rather as a marker of motivational dysregulation which, we propose, is the ultimate psychological determinant of the adverse health and behavioural outcome. This model is outlined in Fig. 1.

1.1. A tale of two cohort studies

Two large scale community studies have contributed much to our understanding of the corollaries of blunted stress reactivity and, accordingly, merit more than a cursory description.

1.1.1. The West of Scotland Twenty-07 Study

The first is the West of Scotland Twenty-07 Study (Ford et al., 1994). Established in 1987, its principal mission was to investigate the processes that generate and maintain socio-demographic differences in health. Participants, all from the Glasgow area, were chosen randomly with the probability proportional to the overall population of the same age within a postcode area. Thus, the sample was selected as a clustered, random, stratified sample of three narrow age cohorts who were 15, 35, and 55 years old at entry to the study; the achieved sample sizes in 1987 were 1009, 985, and 1042 for the three age cohorts, respectively. There was a fairly even gender split and an approximately equal number of manual and non-manual occupational households. However, the sample was predominantly Caucasian, reflecting the demographics of the West of Scotland population from which it was drawn. In all, there were five waves of data collection. Stress testing was undertaken at wave 3, in 1995/6, and consisted of exposure to the paced auditory serial addition test (PASAT), a test that requires attention and memory as well as simple arithmetic. Blood pressure and heart rate were recorded prior to and during the PASAT. The sample at this time