Neuroscience and Biobehavioral Reviews 75 (2017) 40–52

Contents lists available at ScienceDirect

Neuroscience and Biobehavioral Reviews

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

Review article

Costs and benefits of self-efficacy: Differences of the stress response and clinical implications

P. Schönfeld*, F. Preusser, J. Margraf

Mental Health Research and Treatment Center, Ruhr-University Bochum, Germany

ARTICLE INFO

Article history:
Received 2 May 2016
Received in revised form
22 December 2016
Accepted 23 January 2017
Available online 29 January 2017

Keywords:
Self-efficacy
Stress
Stress response
Autonomic arousal
Negative effects
Performance
Mental health

ABSTRACT

Encounters with stressors induce diverse idiosyncratic neuroendocrine, behavioral and psychological reactions across people. Perceived self-efficacy can alter autonomic responses and their effects on mental health. The beneficial effects of self-efficacy in buffering physiological arousal, enhancing performance, and diminishing psychopathological symptoms have been observed in diverse contexts. We show that the role of self-efficacy is not uniformly beneficial, and that higher levels of self-efficacy can sometimes lead to increases in neuroendocrine and psychological stress responses and decreases in performance, a phenomenon that has been widely neglected. We discuss specific conditions under which self-efficacy effects do not uniformly ameliorate or prevent the consequences of stress. These conditions suggest that therapeutic interventions need not always promote self-efficacy in principal. Simultaneously, they to do suggest that keeping self-efficacy high might be disadvantageous or detrimental.

Contents

* Corresponding author at: Mental Health Research and Treatment Center, Ruhr-Universität Bochum, 44787 Bochum, Massenbergstr. 9-13, Germany.
E-mail address: pia.schoenfeld@rub.de (P. Schönfeld).

http://dx.doi.org/10.1016/j.neubiorev.2017.01.031
0149-7634/

1. Introduction

The brain regulates physiological and behavioral responses to stressors. Although stress can negatively affect mental well-being, not everyone exposed to daily hassles or stressful life events is harmed or becomes impaired in their mental health. One explanation for this apparent immunity to harm from stress lies in the mechanisms by which stress impacts psychobiological functioning. Psychological factors also impact the pathology of stress-related disorders and can cause individual differences in the stress response (see Sebastian, 2013). A prominent psychological construct in relation to stress is self-efficacy, that is, people's perceived ability to perform well in challenging situations and the belief that they can manage environmental demands in various functional areas (Bandura, 1977). Associations of perceived self-efficacy with the reduction of stress and the consequences for mental health have even been suggested (e.g., Bandura et al., 1985; Bandura et al., 2003; Bisschop et al., 2004; O'Leary, 1985). Stress leads to alterations in brain functions and frequently results in health impairments (Chaby et al., 2015; Farajdokht et al., 2015; see McEwen, 2007). Yet, the elaboration of a clear and unified definition of stress is complex because the physiological response to aversive and appetitive stimuli can be similar in direction and magnitude (Bonilla-Jaime et al., 2006; Koolhaas et al., 1996; Koolhaas et al., 2011). Research also indicates that self-reported and physiological stress measures may be unrelated (e.g., Martinek et al., 2003).

Despite the frequently replicated finding that perceived self-efficacy plays a mentally protective role (e.g. see Bandura, 2012), literature of recent years on specific effects of self-efficacy on neuroendocrine reactivity, performance and psychological well-being has obtained distinct results. With respect to the neuroendocrine stress response and behavioral reactions to cognitive tasks, the role of perceived self-efficacy is not clearly established as uniformly positive. We explored the hypothesis that individual differences in the perception of one's self-efficacy are crucial in influencing peripheral physiological reactions, effective performance, and mental health.

The traditional view that a high confidence in one's abilities always diminishes the negative impact of challenges is no longer current and comprehensive. This leads to the assumption that the positive model of self-efficacy does not hold true from a general point of view. Divergent presumptions on self-regulation processes in motivational contexts and stress management situations generated empirical findings dissenting such overall protective effects. Some research provides evidence contradicting such uniformly positive effects and instead reports a nonlinear and not exclusively positive relationship between increased self-efficacy level and stress reduction and behavior (e.g., Vancouver, 2005, 2012). This position implies that higher perceived self-efficacy does not always induce lower neuroendocrine reactivity and better performance and psychological adjustment. As the effects of self-efficacy are furthermore dependent on a number of covariates, greater self-efficacy can also lead to increases in autonomic arousal (e.g., Sanz and Villamarín Cíd, 1997; Sanz and Villamarin, 2001; Sanz et al., 2006). As shown in Fig. 1, the physiological, behavioral and psychological responses to stressors can be positive and negative. The sign of the effects of self-efficacy is dependent on a number of covariates, the cognitive paradigm, the methodology and the study design.

The present review examines the effects of stress exposure on key biomarkers of the stress system, including dysregulations and consequences on mental health, and discusses theories on the association between stress, appraisal and coping mechanisms by emphasizing self-efficacy (Section 2). The controversial function of self-efficacy as a factor modulating neuroendocrine, behavioral and psychological relations is stated (Section 3). A critical overview of interventions to boost self-efficacy is then given (Section 4). Finally, important clinical and research implications are surveyed, followed by suggestions for future research (Section 5).

2. The stress system and mental health

2.1. Neuroendocrine (re)activity and dysregulation

2.1.1. The sympathetic-adrenomedullar (SAM) system

The concept of stress was originally delineated by Selye (1975) as a non-specific neuroendocrine response to detrimental stimuli. Afterwards it was extended through a clear distinction between stressor and stress response, which entailed the evaluation that stress is the reaction to environmental demands surpassing the regulatory potential. When faced with a potential stressor, which is normally perceived as a threat interfering with homeostatic control, numerous transactions release a cascade of hormones, peptides and neurotransmitters (e.g. Joëls and Baram, 2009; Schwabe et al., 2012; Wolf, 2012). The release of adrenaline and noradrenaline from the adrenal medulla is induced through the rapidly acting autonomic nervous system. This stimulates vagal afferents to the nucleus tractus solitarius and the locus coeruleus, which activate brainstem noradrenergic nuclei (McGaugh, 2004). Unlike the peak in cortisol arising with a delay of several minutes after stressor onset (see Section 2.1.2), heart rate is elevated from the beginning of stress exposure. The activation of the monoaminergic system has been shown to be mainly located in the hippocampus, the amygdala, the prefrontal cortex and the nucleus accumbens (see de Kloet et al., 2005). Hyperactivity of the SAM has been demonstrated to be associated with increased cardiovascular risk status, risk for hypertension and coronary artery diseases (Chida and Steptoe, 2010; Lovallo and Gerin, 2003). An elevated resting heart rate is correlated with cardiovascular mortality and recognized as an independent risk factor for cardiovascular diseases (Cook et al., 2006). In Short-term activation of interacting adrenocortical mediators associated with *allostasis,* the process of sustaining stability through changes in the hormonal system, facilitate adaption (see McEwen, 2004). Compared to this response to acute stressors, chronic stress and persistent overactivity may lead to damaging effects. In patients with manifest vascular diseases, resting heart rate is related with higher risk for mortality (Bemelmans, 2012). Another quantitative biomarker influenced by sympathetic activity and vagal parasympathetic activity is the heart rate variability (HRV). HRV has been assigned metaanalytically to the ventromedial prefrontal cortex and the amygdala as core regions and it is identified as an important pathway linked, for example, to work-related stress (Chandola et al., 2010; Thayer et al., 2012). Decreased HRV promotes the development of a series of risk factors for cardiovascular disease (Thayer et al., 2010).

2.1.2. The hypothalamic-pituitary-adrenal (HPA) axis

The HPA system is the second major system that is activated in the process of allostasis (McEwen, 2003, 2004) and