



Review

Biological and psychological markers of stress in humans: Focus on the Trier Social Stress Test



Andrew P. Allen^{a,b}, Paul J. Kennedy^{a,b}, John F. Cryan^{a,c},
Timothy G. Dinan^{a,b}, Gerard Clarke^{a,b,*}

^a Alimentary Pharmabiotic Centre, University College Cork, Cork, Ireland

^b Department of Psychiatry, University College Cork, Cork, Ireland

^c Department of Anatomy and Neuroscience, University College Cork, Cork, Ireland

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ABSTRACT

Validated biological and psychological markers of acute stress in humans are an important tool in translational research. The Trier Social Stress Test (TSST), involving public interview and mental arithmetic performance, is among the most popular methods of inducing acute stress in experimental settings, and reliably increases hypothalamic–pituitary–adrenal axis activation. However, although much research has focused on HPA axis activity, the TSST also affects the sympathetic-adrenal-medullary system, the immune system, cardiovascular outputs, gastric function and cognition. We critically assess the utility of different biological and psychological markers, with guidance for future research, and discuss factors which can moderate TSST effects. We outline the effects of the TSST in stress-related disorders, and if these responses can be abrogated by pharmacological and psychological treatments. Modified TSST protocols are discussed, and the TSST is compared to alternative methods of inducing acute stress. Our analysis suggests that multiple readouts are necessary to derive maximum information; this strategy will enhance our understanding of the psychobiology of stress and provide the means to assess novel therapeutic agents.

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* Corresponding author at: Department of Psychiatry, 1.15 Biosciences Institute, University College Cork, Cork, Ireland. Tel.: +353 21 4901408.
E-mail address: G.Clarke@ucc.ie (G. Clarke).

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1. Introduction

1.1. General introduction

Exposure to a situation perceived as threatening or excessively demanding (a stressor) leads to the release of chemicals which can help cope with the stressor (e.g. Joels and Baram, 2009; Schwabe et al., 2012). Acute stress can alter numerous biological functions, such as the hypothalamic-pituitary-adrenal (HPA) axis (Foley and Kirschbaum, 2010), the immune system (Steptoe et al., 2007), the autonomic nervous system (Xhyheri et al., 2012), and the enteric nervous system (Ziegler, 2012). Psychologically, acute stress is a subjectively negative experience (but see Jamieson et al., 2013), and can have positive as well as negative effects on cognition (Lupien

et al., 2007; Starcke and Brand, 2012). Although acute stress is important for responding to threatening situations, chronic stress is damaging to health (McEwen, 2007). Over time, stressful life events can induce chronic stimulation of the HPA axis, leading to depression (Checkley, 1992; Juruena et al., 2011), and poorer prognosis for cancer and heart disease (Maddock and Pariente, 2001). Early-life stress is associated with disorders such as depression (Batten et al., 2004; Dinan, 2005), post-traumatic stress disorder (Koenen et al., 2007), and irritable bowel syndrome (Mayer et al., 2001), and chronic work stress can exacerbate anxiety and depression (Smith, 2000). Moreover, chronic stress will result in changes in subsequent reactivity to an acute stressor (e.g. Chatkoff et al., 2010; Low et al., 2009; Roth et al., 2012). Given the contrast between acute and chronic stress, it is of interest to examine the effects

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