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Psychophysiological correlates of chronic worry: Cued versus non-cued fear reaction

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ABSTRACT

Worry has been defined as a chain of thoughts and images that promote mental attempts to avoid anticipation of potential threats. From this perspective worry can be conceptualized as a state of anticipatory anxiety or non-cued fear reaction. The present study examines high and low chronic worriers during cued and non-cued defense reaction paradigms and during resting and self-induced worry periods. The non-cued procedure was based on the cardiac defense paradigm, whereas the cued procedure was based on the startle probe paradigm using pleasant, neutral and unpleasant pictures as cues. High worriers, compared to low worriers, showed (a) a greater cardiac defense response in the non-cued fear response paradigm, (b) no differences in eye-blink in the startle probe paradigm, (c) reduced skin conductance reactivity during the startle probe paradigm and (d) reduced Respiratory Sinus Arrhythmia, accompanied by increased respiratory rate and decreased expiratory period, during the resting period. These results support the notion of chronic worry as a state of anticipatory anxiety, accompanied by indices of reduced vagal control, that modulates non-cued defense reactions.

1. Introduction

Worry has been conceptualized as a chain of thoughts and images, negatively affect laden and relatively uncontrollable, that promotes mental attempts to avoid anticipation of potential threats (Borkovec, 2002). This conceptualization emphasizes a key aspect of worry: anticipation of threat. Anticipation of threat activates defense reactions, either the fight–flight response or the freezing response. Continuous activation of this type of defense reactions is a form of being permanently stressed and vigilant to emotional negative information, thus increasing the risk of physical and mental problems (Brosschot et al., 2006; Knepp and Friedman, 2008).

The psychophysiological correlates of worry have been investigated in a number of studies using as participants non clinical high trait worriers and patients with generalized anxiety disorder (GAD) (Hoehn-Saric et al., 1993; Dua and King, 1987; Borkovec et al., 1983, Borkovec and Roemer, 1995; Karteroliotis and Gil, 1987; Lyonfields et al., 1995; Thayer et al., 1996, 2000; Segerstrom et al., 1999; Wilhelm et al., 2001; Davis et al., 2002; Brosschot et al., 2003; Hofmann et al., 2005; Jönsson, 2007; Conrad et al., 2008). Although the data are not totally consistent, the two most repeated findings are the absence of sympathetic hyper-reactivity (indexed mainly by skin conductance measures) and the presence of reduced vagal control (indexed by HR variability measures) in high worry people. Confirming previous findings, Thayer and Brosschot (2008) have recently reported increased HR and decreased HR variability, monitored over a 24 h period, associated with daytime worry and the subsequent nighttime. As regards brain mechanisms, a recent study using functional MRI in GAD patients (Oathes, 2008) found greater amygdala activation during anticipation of emotional pictures, but hyporeactivity in the same region during the actual presentation of the pictures.

A relevant psychophysiological research area, insufficiently investigated in relation to chronic worry, is the modulation of defense reactions. As mentioned above, a central feature of chronic worry is anticipation of threat. Therefore, it should be expected that anticipation of threat in high worriers would modulate defense reactions. Two specific defense reactions, eye-blink startle and cardiac defense, have been widely investigated in recent years in the context of fear and anxiety research (for a review, see Lang et al., 2000; Grillon, 2002; Bradley and Lang, 2007; Vila et al., 2007). The startle reflex in humans involves a quick closing of the eyes accompanied by stiffening of the head, dorsal neck, body walls, and limbs, as if to protect from a predator (Graziano and Cooke, 2006). Cardiac defense, on the other hand, refers to the heart rate response to intense or aversive stimulation. It consists of a complex response pattern, observed within the 80 s after stimulus onset, with a short latency acceleration/deceleration (peak around second 3), followed by a long latency acceleration/deceleration (peak between 30 and 40 s) (Vila et al., 2007).

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