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HPA axis response to a psychological stressor in generalised social phobia

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Abstract

Social phobia may be associated with a dysfunction of the hypothalamic–pituitary–adrenal (HPA) axis. In this study we determined HPA axis responsivity to a psychological stressor in patients with social phobia and compared them to healthy controls. Fifteen patients with DSM IV social phobia with a mean score of 77.7 on the Liebowitz Social Anxiety Scale and 15 age and sex matched controls underwent the stressor consisting of mental arithmetic and a short term memory test performed in front of an audience. Plasma levels of cortisol and corticotropin were measured at various intervals throughout the test. Although baseline measures of cortisol did not differ between patients (319.8±34.6 nmol/l) and controls (279.5±42.7 nmol/l)(t=0.7, df=28, $P<0.5$) nor did baseline corticotropin values (8.6±2.1 pg/ml vs 13.7±2.0 pg/ml respectively) ($t=-1.8$, df=28, $P<0.08$) this stressor resulted in a significantly greater delta max cortisol response (the difference between baseline values and the maximum increase during the stressor) in patients (167.1±23.7 nmol/l) than in controls (106.7±16 nmol/l) (t=2.1, df=28, $P<0.04$). There was no significant difference in delta max corticotropin between groups (patients 8.8±2.1 pg/ml vs controls 9.1±1.9 pg/ml) ($t=-0.08$, df=28, $P<0.9$). This preliminary study indicates that patients with social phobia appear to have a hyper-responsive adrenocortical response to psychological stress. © 2002 Elsevier Science Ltd. All rights reserved.

Keywords: Social phobia; Cortisol; Corticotropin; Psychological stressor; Mental arithmetic; Corticotropin releasing factor

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1. Introduction

Social phobia, also known as Social Anxiety Disorder is a common psychiatric disorder, which results in significant distress and social disability (Stein, 1998). It is characterized by a persistent fear of situations in which the individual may be exposed to the scrutiny of others, fearing that he or she may act in a way that is humiliating or embarrassing (APA, 1994). Social phobia has been subdivided into two types, generalised social phobia and "specific" or "discrete" social phobia, the former subtype being associated with the most severe disability (Brunello et al., 2000).

Although the exact cause of this disorder is unknown, certain biological theories have been posited. Several investigators have implicated a possible role for dopamine, serotonin and noradrenaline (for reviews, see Stein, 1998; Bell et al., 1999) and neuroimaging studies (Tiihonen et al., 1997; Schneier et al., 2000), neuroendocrine challenge tests (Tancer et al. 1993, 1994/1995; Hollander et al., 1998) and laboratory stressors (Heimberg et al., 1990; Levin et al., 1993; Stein et al., 1994) have supported some of these theories although the findings are by no means uniform (Tancer et al., 1994/1995; Stein et al., 1995).

An alternative theory is that social phobia may be a stress-related condition, with the core endocrine reaction seen in man and other animals in response to stress activating the hypothalamic–pituitary–adrenal (HPA) axis which results in an increase in cortisol (Axelrod and Reisine, 1984). Stress, both physical and emotional (Chrousos and Gold, 1992), acute or chronic (Akil and Morano, 1995) results in HPA axis activation. Corticotropin releasing factor (CRF), a hypothalamic neuropeptide which can activate the pituitary–adrenal system, has been implicated in the pathophysiology of anxiety disorders, (Chrousos and Gold, 1992; Arborelius et al., 1999) enhanced release of which may account for many of the symptoms of anxiety (Butler and Nemeroff, 1990).

Basal cortisol measures provide an index of the resting activity of the HPA system at one or multiple points in time. However, reaction to laboratory stressors, provide a way of assessing its ability to respond to perturbations and as numerous investigators have shown (Leyton et al., 1996; Petrides et al., 1997; Pike et al., 1997; Singh et al., 1999), normal basal HPA indices do not preclude abnormalities which may be seen on exposure to these stressors. The study of endocrine reactivity to stressors within the laboratory permits standardisation of the stressor and the ability to control better for known confounding factors (Biondi and Picardi, 1999). The use of stressors to examine HPA axis reactivity in social phobia has provided conflicting results. Martel et al. (1999) examined salivary cortisol levels in socially phobic adolescent girls in response to a modified Trier Social Stress Test (TSST) but found no difference between patients and controls. Similarly, Levin et al. (1993) measured plasma cortisol in response to a public speaking task in patients with social phobia and found decreases in cortisol levels in both generalised and discrete social phobics and controls with no differences between groups. Lastly, Granger et al. (1994) found a positive correlation between cortisol response to a parent–child conflict task and measures of social anxiety in children.

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