Asian Journal of Psychiatry 14 (2015) 57–60

Contents lists available at ScienceDirect

Asian Journal of Psychiatry

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

Short communication

The cortisol response to social stress in social anxiety disorder

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ARTICLE INFO

Article history:
Received 28 October 2014
Received in revised form 13 March 2015
Accepted 18 March 2015

Keywords:
Social anxiety disorder
Cortisol
Social stress
Childhood trauma
Emotional abuse

ABSTRACT

This study evaluated the cortisol stress response (CSR) following the Trier Social Stress Test in Social Anxiety Disorder (SAD) and control participants, to determine whether individual differences in CSR associate more with SAD diagnosis or dimensional characteristics [i.e. childhood trauma (CT)]. Twenty-one participants (11 with SAD) had full data available for both CT-scores and cortisol area-under-the-curve (AUC). Linear regression produced significant results: predicting AUCG with study group, emotional abuse (EA) scores and their interaction ($F = 3.14$, df = 5,15; $p = .039$); of note, the study group by EA interaction was significant at $p = .015$, driven by a strong positive association between EA and cortisol AUCG in the control group, and a negative association between these variables in the SAD group (standardized-beta = 1.56, $t = 2.75$, $p = .015$). This suggests that EA in SAD patients is associated with altered CSR, highlighting need to measure dimensional characteristics.

1. Introduction

Social Anxiety Disorder (SAD) is a common disorder defined by an intense fear of being negatively evaluated by others, and fear of embarrassment in social situations (Antony and Stein, 2009; Woody and Nosen, 2009). The estimated lifetime prevalence of SAD is 10 percent (Kessler, 2003; Stein and Stein, 2008). Individuals with SAD often experience negative self-focused cognitions that tend to worsen anxiety when social threat is anticipated (Schulz, 2008).

Given the fundamental role of impaired social evaluation in the pathophysiology of SAD, it would be important to determine whether SAD patients exhibit any unique pattern of stress responsivity to a social challenge. This can be studied empirically using standardized social stress protocols measuring neurohormonal changes. The literature to date examining the cortisol stress response (CSR) following a social challenge in SAD has reported mixed results (Beaton et al., 2006; Condren et al., 2002; Furlan et al., 2001; Levin, 1993; Martel et al., 1999; Roelofs et al., 2009; Shirotsuki et al., 2009; van West et al., 2008; Yoon, 2012). One possible explanation for this inconsistency is that moderating factors such as childhood trauma (CT) have a greater influence on social stress responses and/or overall cortisol secretion than does the condition, itself. For example, Elzinga et al. (2010) showed that individuals with SAD and a history of childhood abuse had greatly enhanced cortisol reactivity to a psychosocial stress task in comparison to those with SAD alone. Similarly, van der Vegt et al. (2010) examined awakening cortisol levels in anxiety disorders, and stressed the effects of early childhood adversity on cortisol reactivity in these conditions. This is in keeping with the notion that emotional abuse (EA), more so than physical or sexual abuse, may be associated with the pathophysiology of SAD (Asher and Coie, 1990; Lochner et al., 2010). More recently, a study has found that different forms of CT are associated with different CSR (Kuhlman et al., 2015). Failure to assess and control for such moderating factors could help explain previous heterogeneous findings.

The goal of the current study was to examine the relationship between the CSR and CT, as well as its subcomponent childhood EA. To this end, the present study examined cortisol responses in SAD and control participants in response to the TSST. It further evaluated whether individual differences in the experience of CT were associated with the CSR in this context. Thus, it was hypothesized that individual differences in early adversity would

Abbreviations: SAD, social anxiety disorder; CSR, cortisol stress response; CT, childhood trauma; EA, emotional abuse; CAMH, Centre for Addiction and Mental Health; SCID, structured clinical interview for DSM disorders; TSST, trier social stress test; CTQ, Childhood Trauma Questionnaire; AUC, area under the curve.

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http://dx.doi.org/10.1016/j.ajp.2015.03.004
1876-2018/

be a strong predictor of the CSR following the TSST, whether or not SAD participants differed from controls overall.

2. Methods and materials

The study sample consisted of 24 participants (12 SAD, 12 controls) aged 18 to 65. Of those 24 participants, 8 were female and 16 were male. SAD was established as the primary diagnosis by a psychiatrist at CAMH then confirmed by a trained interviewer using the SCID (First et al., 1995). Control participants had no current or past history of Axis I or II disorders. Exclusion criteria for both groups included recent major life stressors, pregnancy, breastfeeding, and chronic medical disease. Controls were matched to SAD participants by gender and age. Participants underwent an informed consent process and signed a consent form prior to enrolment. This study received CAMH Research Ethics Board approval and complied with the Code of Ethics of the World Medical Association.

The study consisted of two visits. During visit 1, all participants underwent a diagnostic interview (SCID) (First et al., 1995) to confirm the diagnosis of SAD and rule out other Axis I and II disorders. Demographic and current treatment information, medical and psychiatric history were obtained. Participants completed questionnaires including standardized measures of anxiety, depression, and the CTQ (Bernstein and Fink, 1998). The CTQ is a self-report questionnaire assessing CT with subscales examining emotional, physical, and sexual childhood abuse and neglect.

During visit 2, The TSST (Kirschbaum et al., 1993) was administered in the early afternoon (to account for diurnal variations). Based on the procedures developed by Kirschbaum et al., (1993), social stress was induced by placing participants in a novel situation where they believed they were being socially evaluated. This study followed the same protocol reported in Roelofs et al. (2009). Based on Roelofs et al. (2009), TSST modifications were available to participants who felt unable to complete the TSST. Participants were reminded that they could stop participating at any time. Serial plasma cortisol samples were taken two minutes after arrival, immediately prior to TSST, and ten, thirty, forty-five, and sixty minutes post-TSST using an indwelling line connected to a blood pump.

A total of 21 participants [11 with SAD including 4 females, and 10 controls including 4 females] had full data available for both CT scores and cortisol area under the curve (AUC). To examine the influence of study group, CT scores and their interaction on cortisol responses during the TSST, we used linear regression including each of these three independent predictors while controlling for age and BMI. Separate models were examined for AUC_G (AUC with respect to ground) and AUC_I (AUC with respect to increase) (Pruessner et al., 2003). Based on (1) the high rates of emotional abuse reported by patients with SAD (Asher and Coie, 1990; Lochner et al., 2010) and (2) recent evidence suggesting that different forms of childhood adversity are associated with different cortisol stress responses (Kuhlman et al., 2015), we performed one set of analyses based on total CTQ scores and another based on childhood EA scores only.

3. Results

When total CTQ scores were included in the models, no significant effects were noted. In contrast, a significant result was produced when predicting cortisol AUC_G with study group, emotional abuse scores and their interaction, controlling for age and BMI ($F = 3.14$, df = 5.15; $p = .039$). Of particular interest was the finding of a highly significant study group by emotional abuse interaction in predicting AUC_G (standardized beta = 1.56, $t = 2.75$, $p = .015$). As shown in Fig. 1, this was driven by a very strong positive relationship between emotional abuse scores and AUC_G in the control group ($r^2 = 49$) and a moderately strong negative relationship between emotional abuse scores and AUC_G in the

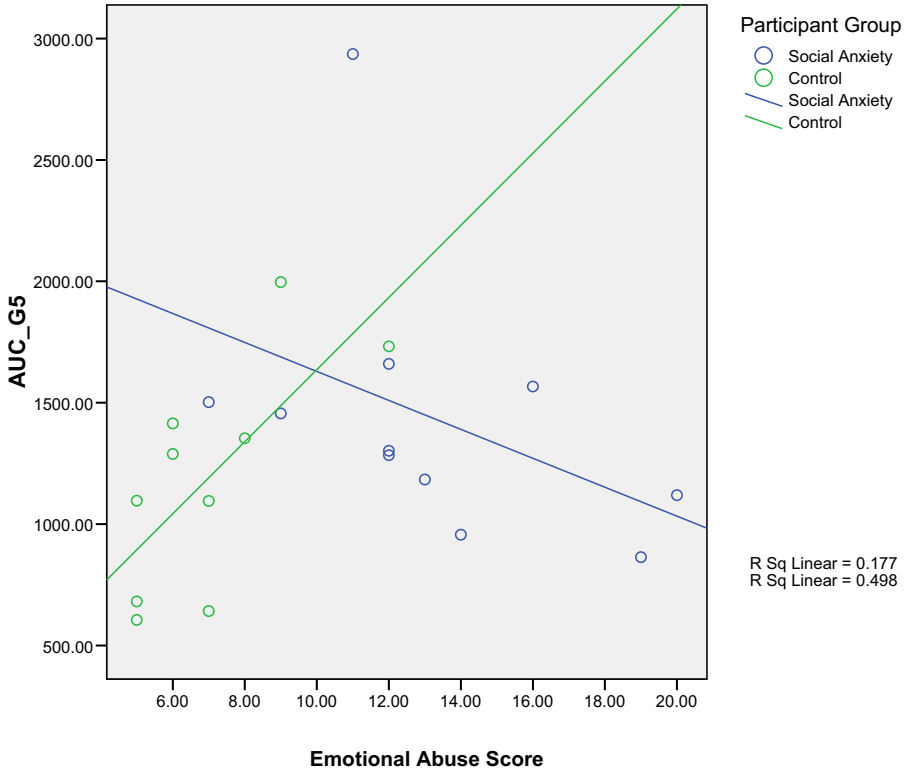

Fig. 1. Relationship between emotional abuse scores and cortisol (AUC_G) in response to TSST in SAD patients and controls.