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Salivary cortisol and DHEA reactivity to psychosocial stress in socially anxious males

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ABSTRACT

The purpose of the present study was to examine Hypothalamus–Pituitary–Adrenal (HPA) axis reactivity in social anxiety. The present study used a standardized psychosocial stress protocol (the Trier Social Stress Test; TSST; [Kirschbaum, C., Pirke, K.M., Hellhammer, D.H., 1993. The 'Trier Social Stress Test'—a tool for investigating psychobiological stress responses in a laboratory setting. Neuropsychobiology 28, 76–81.]) with 11 higher-social-anxiety and 11 lower-social-anxiety male college students. Psychological responses and salivary cortisol and dehydroepiandrosterone (DHEA) reactivity and cortisol/DHEA ratio were assessed at seven different times. The results showed that there was a significantly lower cortisol responsiveness in the higher social anxiety group but there was no significant difference of DHEA responsiveness. Further analyses showed lower responses for the cortisol/DHEA ratio in the higher-social-anxiety group to the TSST. These results suggest that there may be reduced HPA axis reactivity to psychosocial stress in socially anxious people.

1. Introduction

Social anxiety is a highly prevalent condition in both clinical and non-clinical populations. Social Anxiety Disorder (SAD), or Social Phobia is the commonest of the anxiety disorders, and it has a high rate of lifetime prevalence (Kessler et al., 1994; Magee et al., 1998; Pellisso et al., 2000). Social anxiety is characterized by a fear of negative evaluation by others (Clark and Wells, 1995; Rapee and Heimberg, 1997). One of the most fearful situations for SAD patients and for individuals with social anxiety is public speaking (Stein et al., 1996). SAD patients and individuals with high social anxiety have a number of similar psychological and physiological features that differ only in the degree of intensity (Turner et al., 1986, 1990), therefore, it has been suggested that there is an overlap between shyness, social anxiety and SAD (Rapee, 1995). It is possible that there are common features and a continuum from low to high social anxiety and SAD. Many previous studies have examined the specific psychological and physiological features of SAD patients, as well as of individuals with high social anxiety. These studies have clarified the psychopathology and the pathophysiology of SAD.

Recently, an abnormality of the Hypothalamic–Pituitary–Adrenal (HPA) axis has been implicated in major depressive and anxiety disorders (Marshall et al., 2002; Peeters et al., 2004; Stones et al., 1999). Cortisol is the glucocorticoid hormone that is secreted by the adrenal gland and it has been suggested that cortisol secretion is stimulated by psychological distress and social evaluative threats (Dickerson and Kemeny, 2004). As a result abnormalities and the reactivity of the HPA axis have been the focus of many investigations on psychiatric disorders. Furthermore, cortisol is considered to be the endocrine hormone modulating mental and physical states that are associated with psychosocial stressors.

Uhde et al. (1994) found no difference in basal cortisol values between SAD patients and normal controls. Furlan et al. (2001) examined differences in HPA axis reactivity between high and low responding SAD patients and healthy controls using speech and physical tasks and found specific HPA axis responses to speech tasks, but not for physical tasks. Condren et al. (2003) found significantly greater delta max cortisol values (the difference between baseline and maximum cortisol level during stress) in SAD patients in comparison to healthy controls, when a serial subtraction test and a digit span test were conducted. Martel et al. (1999) reported that both social phobia patients and controls had a significantly elevated level of cortisol prior to the Trier Social Stress Test (TSST: Kirschbaum et al., 1993). However, this study did not find a difference in HPA axis reactivity resulting from psychosocial stressors between SAD adolescents and the control

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group. On the other hand, Beaton et al. (2006) reported lower salivary cortisol levels in a clinically diagnosed social phobia group, compared to a non-clinical control group, in a speech task. Tops et al. (2008) suggested that the fear of negative evaluation, which is core cognition in SAD, was related to a low cortisol mobilization response. The above studies suggest two patterns of HPA axis reactivity to situations causing social anxiety: high and low cortisol responses, and as such, have been inconsistent. Such differences may arise from differences in psychosocial stressors, or from the protocols that have been used in different studies. It is suggested that cortisol response to standardized psychosocial stressors should be evaluated to clarify the role of endocrine reactivity to social anxiety.

It is meaningful to examine the salivary dehydroepiandrosterone (DHEA) response to situations causing social anxiety. DHEA, in addition to cortisol, is a major steroid produced by the zona reticularis of the adrenal cortex. Some studies have suggested that lower DHEA levels are associated with lower psychological wellbeing (van Niekerk et al., 2001). One study has shown that DHEA was negatively correlated with depression (Michael et al., 2000). Another investigation has indicated that the administration of DHEA improved mood of healthy young men (Alhaj et al., 2006). Conversely, other studies have reported that the administration of DHEA has little effect on the mood of healthy elderly people (Kudielka et al., 1998; Wolf and Kirschbaum, 1999). In conclusion, these findings have implicated DHEA in the regulation of mental states. However, the mechanism of stress-induced DHEA secretion (Oberbeck et al., 1998) and DHEA reactivity in SAD to psychosocial stressors has not been sufficiently understood.

Moreover, a difference in the ratio of cortisol to DHEA has been found in individuals with mental disorders. Blauer et al. (1991) indicated that DHEA could antagonize cortisol activity. Therefore, it is considered that the cortisol to DHEA ratio may be a marker of endocrine imbalance. For example, Goodyer et al. (2003) showed that persistently depressed individuals had a higher cortisol to DHEA ratio than non-depressed and remitted individuals. Goodyer et al. suggested that the high ratio might be a marker of persistent psychiatric disorders. Other studies have found that a higher morning cortisol to DHEA ratio is associated with higher anxiety (van Niekerk et al., 2001). Young et al. (2002) showed that the cortisol to DHEA ratio from saliva samples correlated with the length of the current depressive episode and suggested that the cortisol to DHEA ratio could be a marker of depressive states. Thus, it has been speculated that the cortisol to DHEA ratio would represent an endocrine imbalance of the HPA axis function and may be a marker of the state of other psychiatric disorders. However, no studies have investigated the cortisol to DHEA ratio in social anxiety and SAD.

Above-discussed findings suggest that there might be an abnormality of the HPA axis function in individuals with high social anxiety and in SAD patients. Furthermore, this abnormality may influence the regulation of their mental states. Therefore, investigating the HPA axis reactivity in relation to social anxiety, including the roles of cortisol, DHEA and cortisol to DHEA ratio, may be important for understanding the psychopathology of SAD and social anxiety. The present study investigated HPA axis and psychological reactivity to standardized psychosocial stresses as measured by the Trier Social Stress Test in male college students with high and low social anxieties.

2. Method

2.1. Participants

Twenty-two Japanese male students who did not smoke (mean age = 21.62, SD = 2.46) were recruited for the present study via advertisements. Only males were recruited to control for the fluctuating influence in females of estradiol and progesterone. Before the experiments, we administered Japanese version of the Short Fear of Negative Evaluation scale (SFNE; Sasagawa et al., 2004) for pre-screening of high and low socially anxious males. Thirteen males were excluded from the present study because they did not meet the criteria of high and low social anxious which was previously shown.

Watson and Friend (1969) designed original Fear of Negative Evaluation scale (FNE) which consists of 30 true–false items. The FNE and SFNE assess the core cognition of SAD, the "fear of negative evaluation by others". The SFNE consists of 12 items rated on 5-point Likert-type scale. Sasagawa et al. (2003) reported that the mean score of SFNE in college students is 38.20 (SD = 10.28). In another study, Chen (2005) reported that the mean score of Japanese SAD patients in Cognitive Behavioral Group Therapy program for SAD is 45.59 and the score decreased to 36.93 at 3-month follow-up period after the program. The statistical examination by Graded Response Model (Samejima, 1969) showed that SFNE has sufficient measurement accuracy in comparison to the original FNE. Sasagawa et al. (2003) reported that SFNE showed moderate correlation with Liebowitz Social Anxiety Scale (Liebowitz, 1987) ($r = .45$) or Social Phobia Scale and Social Interaction Anxiety Scale (Mattick and Clarke, 1998) ($r = .47$, .49, respectively) and high internal consistency ($\alpha = .92$). Therefore, it was considered that SFNE has good reliability and discriminant validity of SFNE.

Therefore, participants were included only if they were higher socially anxious (HS group) or lower socially anxious (LS group) based on their scores on SFNE. The HS group (11 males; mean age = 21.91, SD = 2.84) scored more than 44 points, which is +0.5 SD above the SFNE mean (Sasagawa et al., 2003). The LS group (11 males; mean age = 21.30, SD = 2.06) scored less than 34 points, which is −0.5 SD below the SFNE mean. In the present study, SFNE scores of HS group (mean = 48.73, SD = 3.41) was significantly higher than the scores of LS group (mean = 28.00, SD = 3.44), $t(20) = 14.20$, $p < .01$. There was no significant difference in age ($t(20) = .56$, $p = .58$) and Body Mass Index (BMI: $t(20) = 1.59$, $p = .13$) between HS group and LS group (Table 1).

2.2. Procedure

First, all participants were told that the purpose of the present study is to examine psychological and physiological reactivity to psychosocial stress. After that, the procedure of the present study was told and written informed consent was obtained.

All experiments were conducted after 15:00 to control for the circadian rhythm of the HPA axis. Meals, coffee or tea, exercise, and brushing teeth were prohibited 1 h before the experiments. A semi-structured interview about medical history and current or lifetime of any psychiatric disorder was conducted before the experiments to check that all participants were physically healthy. It revealed that none had any clinically significant mental disorders and took medications that may alter HPA activity.

Participants were tested individually. Before the stress tasks, participants rested 10 min for a baseline. Then we conducted TSST following the procedure of Kirschbaum et al. (1993) to produce psychological and physiological stress. The TSST consists of a speech task and a serial subtraction test. First for the speech task, participants had a 10-minute preparation period and then gave a 5-minute speech to two assessors (male and female) about a job. Then participants did a serial subtraction test for 5 min. They had to count backwards from 2083 by 17 s as quickly and correctly as possible. Every time they made

Table 1
Means and standard deviations of demographic data of participants.

	HS group ($n = 11$)	LS group ($n = 11$)	t-values	p
BMI (kg/m^2)	20.75 (1.33)	22.84 (4.16)	1.59	n.s.
Age (year)	21.30 (2.06)	21.91 (2.84)	.56	n.s.
SFNE	48.73 (3.41)	28.00 (3.44)	14.20	$p < .01$