

Cortisol response and autonomic nervous system function to repeated stress in social anxiety disorder

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Introduction

The purposes of this study were to determine cortisol response, autonomic nervous system function, and a psychological stress response to repeated psychological stress in social anxiety disorder (SAD) patients and healthy controls.

Methods

Participants

- (1) SAD patients (N=7) (Male:N=1, Female:N=6, Age: 29.14 ± 6.89)
- Patients met DSM-IV-TR (APA , 2000) diagnostic criteria for SAD.
- Patients were not on any psychoactive medication.
- (2) Controls (N=9) (Male:N=1, Female:N=8, Age: 28.78 ± 7.17)
- Normal control subjects were matched for age and sex with SAD patients.

Procedures

- On 3 days out of 4 consecutive days, the participants were subjected to the Trier Social Stress Test (TSST).
- All experiments were conducted in the afternoon to minimize the effects of the cortisol circadian rhythm.
- TSST: Adaptation period (Adap); 10min Anticipation period (Antic); 10min Test period (Test); 10min -A speech task and mental arithmetic Recovery period (Rec); 20min

Measures

- (1) Endocrine stress response (cortisol)
- (2) Autonomic nervous system function: systolic blood pressure (SBP), diastolic blood pressure (DBP), heart rate (HR)
- (3) Psychological stress response: Stress Response Scale-18 (SRS-18)
- (4) Severity of social anxiety symptomatology: Fear of Negative evaluation (FNE), Social Avoidance and Distress Scale (SADS)
 (5) Questionnaires on physical conditions

Results

Table 1Age and Severity of Social Anxiety Symptomatologyof SAD Patients and Normal Control Subjects

	SAD (n=7)		controls (n=9)		
	Μ	SD	Μ	SD	t
Age(years)	29.14	6.89	28.78	7.17	.10 n.s
FNE	22.14	4.81	10.78	7.64	3.43 **
SADS	19.14	7.10	6.11	3.25	4.91 **
** n<0.01					

Investigation on the *dysfunction* of stress responses in patients with SAD

- Salivary cortisol levels were lower in SAD patients than in controls.
- There was no significant difference in autonomic responses (SBP, DBP, and HR) and a psychological response (SRS-18) between the two groups.
- SAD patients and normal controls reacted properly to the TSST in autonomic and psychological responses.



Fig. 1 Mean **cortisol** in SAD and control subjects during the adaptation period (Adap), the anticipation period (Antic), the test period (Test), and the recovery period (Rec1, Rec2).



Fig. 2 Mean **SBP** in SAD and control subjects during the adaptation period (Adap), the anticipation period (Antic), the test period (Test1, Test2), and the recovery period (Rec).

Fig. 3 Mean **DBP** in SAD and control subjects during the adaptation period (Adap), the anticipation period (Antic), the test period (Test1, Test2), and the recovery period (Rec).

Fig. 4 Mean **HR** in SAD and control subjects during the adaptation period (Adap), the anticipation period (Antic), the test period (Test1, Test2), and the recovery period (Rec).

Fig. 5 Mean **total scores of the SRS-18** in SAD and control subjects during the adaptation period (Adap), the anticipation period (Antic), and the test period (Test).

Investigation on the *habituation* of stress responses in patients with SAD

- Both groups did not demonstrate decrease in physiological responses (cortisol, SBP, DBP, and HR) from day 1 to day 3.
- In SAD patients, HR during the adaptation period of day 3 were significantly higher than that of day 1.
- Although habituation of a psychological response occurred in control subjects, it did not in SAD patients.

Fig. 6 Mean cortisol responses over the five test periods (Area Under the Curve) to 3 days repeated psychological stress in SAD and control subjects.

Fig. 7 Mean **HR during the ADAPTATION period** to 3 days repeated psychological stress in SAD and control subjects.

Fig. 8 Mean **total scores of the SRS-18 during the TEST period** to 3 days repeated psychological stress in SAD and control subjects.

Conclusion

The present results indicate that expectation anxiety increases and habituation of psychological responses does not occur in repeated social stress in patients with SAD.

Therefore, the importance of cognitive intervention besides exposure is implied in the treatment of SAD.