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SHORT COMMUNICATION

Relationship between alexithymia, alexithymia factors and salivary cortisol in men exposed to a social stress test

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Summary
Background: The fact that alexithymia is associated with several medical and psychiatric disorders suggests that it may be a vulnerability factor for various diseases, possibly by enhancing stress responses. To test this "alexithymia-stress hypothesis", we measured the influence of alexithymia and alexithymia subfactors on the cortisol response to an acute stressor.
Methods: Twenty-eight male students were exposed to the Trier Social Stress Test (TSST), during which saliva samples for cortisol determination were collected.
Results: Subjects reacted to the stressor with a significant cortisol response. Subjects scoring high on alexithymia evidenced an increased basal anticipatory cortisol level but their peak cortisol and area under the curve were similar to that of low scorers. Multiple regression analyses revealed that the increased cortisol in high scorers was due to only one subfactor of alexithymia, "the difficulty in describing feelings" factor (DDF). DDF high scorers reacted with a large increase in cortisol during anticipation but not during exposure to the stress test.
Conclusion: The observation that alexithymia scores were associated with differences in cortisol levels before social stress exposure raises the possibility that alexithymia modulates cortisol levels, possibly by affecting the anticipatory cognitive appraisal of situations. This may be essentially attributed to the DDF factor. This observation sheds new light on the "alexithymia-stress hypothesis", which may be of importance to better understand the relationship between alexithymia and diseases. Further studies to address this issue should focus on the factorial structure of the construct and on the importance of anticipation.
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1. Introduction

Alexithymia is a general deficit in the processing of one's own emotions, characterised by the following impairments: difficulty in differentiating feelings and distinguishing them from bodily sensations and emotional arousal (DIFF), difficulty in describing feelings to others (DDF), and an externally oriented way of thinking (EOT). Alexithymia has been found to be associated with several somatic and psychiatric disorders (Lumley et al., 1996), suggesting that it might be a vulnerability factor for various diseases. Because most of the previous research has shown relationships between alexithymia and stress-related diseases, some authors have suggested that the influence of alexithymia on the expression of stress-related pathological states might involve poor resistance to stress. This hypothesis, originally described as the "alexithymia-stress hypothesis" (Martin and Pihl, 1985) was tested empirically in more than 10 different electrophysiological studies which revealed inconsistent results. In subjects exposed to emotional stimuli or to situational stressors, alexithymia was associated with hyper-arousal or conversely with similar or less reactivity. Furthermore, at baseline, a high and stable level of autonomic reactivity was found in alexithymics compared to non-alexithymics in some but not all studies (Berthoz et al., 2002).

The aim of the present study was to test the "alexithymia-stress hypothesis", according to which alexithymia would enhance HPA reactivity in response to life stressors. To this end, we submitted subjects to a laboratory social stressor, the Trier Social Stress Task (TSST) (Kirschbaum et al., 1993) and measured salivary cortisol before, during and after stress exposure. Because previous studies have shown a differential association of specific alexithymia subfactors in diseases (Luminet et al., 2006) — suggesting the alexithymia construct is not homogenous in its effects on such illnesses —, we decided to test the effect of both alexithymia and its subfactors.

2. Methods

2.1. Sample

Twenty-eight students (all non-smokers, mean age: 20.86, S.D.: 2.38), recruited through advertisements, participated in the study in exchange for course credit or remuneration. Women were excluded due to the possible impact of menstrual cycle phase or oral contraceptives (Kirschbaum et al., 1999) on cortisol levels. Subjects who received medication and/or reported suffering from a somatic or a psychiatric illness were also excluded. Participants were informed that the study examined individual differences in job interviews and were instructed (1) not to abuse alcohol the day before the experiment, (2) to respect their usual sleeping hours, (3) not to ingest alcohol, caffeine, or soda on the day of the experiment and (4) not to ingest any food or drink 1 h before the experiment's onset (as detailed in Mikolajczak et al., 2007).

2.2. Procedure

The experiment was conducted in accordance with the Declaration of Helsinki and approved by the ethical committee of the Psychology Department, which follows the rules of the IRB. The effect of circadian hormone rhythms was minimized by conducting all sessions between 1400 h and 1800 h. As detailed in Mikolajczak et al. (2007), after providing written informed consent and a basal saliva sample, participants underwent a short relaxation procedure (1 min) and then were left alone for 10 min in a comfortable room with several magazines at their disposal. After providing a second basal sample of saliva, subjects were introduced to the TSST. This stressor — which induces profound endocrine responses in 70—80% of subjects — consists of both a 5 min public speech and a 5 min cognitive task in front of two examiners and a video camera. Participants then provided a sample of saliva and spent the rest of the experiment reading magazines, and were interrupted only for five saliva collections (timing in Fig. 1) before debriefing. They completed the 20-item

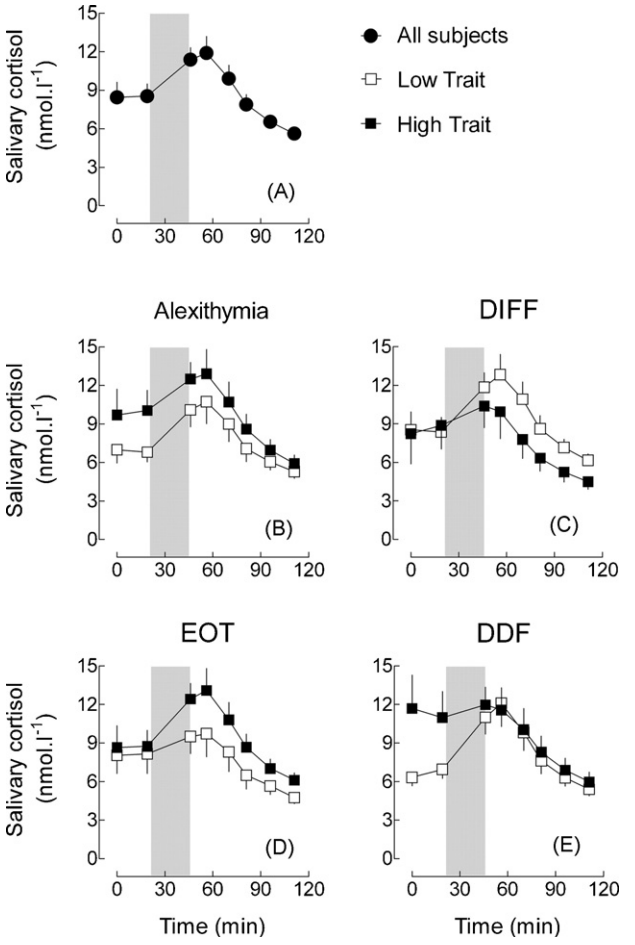

Figure 1 Cortisol response to TSST (A) and moderation by alexithymia (B), DIFF (C), EOT (D) and DDF (E). Groups were created via a median-split. They were named low and high trait for alexithymia and its subfactors. ANOVA on time and alexithymia yielded a significant main effect of time ($F_{1,\ 27} = 13.24$, $p < .001$) and no effect of alexithymia (time × alexithymia interaction: $F_{2,\ 26} = 0.014$, $p = 0.9$). The medians for alexithymia, DIFF, EOT and DDF were 48, 16, 18 and 15, respectively. The mean scores ± S.D. obtained in the low vs. high groups for alexithymia, DIFF, EOT and DDF were 39.54 ± 6.58 vs. 55.6 ± 6.21, 12.47 ± 3.27 vs. 20.44 ± 2.56, 15.70 ± 1.42 vs. 21.72 ± 4.14 and 10.41 ± 2.87 vs. 18.36 ± 2.50, respectively.

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Toronto Alexithymia Scale (TAS-20) in a separate questionnaire session.

2.3. Measures

Alexithymia was measured with the French version of the TAS-20 (Bagby et al., 1994; Loas et al., 1996), the most widely used measure of the alexithymia construct. This questionnaire consists of 20 items answered on a 5-point scale, targeting three specific dimensions: DIFF (e.g., "When I am upset, I do not know if I am sad, frightened or angry"), DDF (e.g., "I find it hard to describe how I feel about people"), and EOT (e.g., "I prefer talking to people about daily activities rather than their feelings").

Cortisol secretion. Saliva samples were collected using Sarstedt® Salivettes (Nümbrecht, Germany) and stored at room temperature until completion of the session and at—20 °C until assay. Saliva was extracted by centrifugation (1000 × *g*, 2 min) and cortisol measured using a competitive polyclonal immunoassay, comprising an electromagnetic separation step followed by electrochemiluminescence quantitation with the Elecsys 1010/2010 analyser (Roche Diagnostics, Mannheim, Germany). Results are expressed in nanomoles per litre (nmol/l).

2.4. Statistical procedures

To assess the relationship between alexithymia and endocrine parameters, bivariate and multivariate regressions were computed. In order to assess anticipatory, current and overall effects of the TSST and alexithymia on cortisol levels we computed three indicators: (1) baseline cortisol, obtained by averaging the first two samplings; (2) peak cortisol (i.e., the value obtained on fourth sampling); (3) the area under the curve with respect to ground (AUCg), which was computed using the trapezoidal method recommended by Pruessner et al. (2003). We also computed ANOVAS for repeated measures with time as within-subject factor and level of alexithymia (two categories: below and above the median value, named low and high trait, respectively) as between-subject factors. The graphs obtained in this manner allow a good visual presentation of the effects of alexithymia and its subfactors on cortisol levels.

3. Results

The means scores ± S.D. for the TAS-20 and subfactors were 48.14 ± 10.28 for alexithymia, 15.04 ± 4.84 for DIFF, 13.54 ± 4.78 for DDF and 19.57 ± 4.48 for EOT. Out of the 28 subjects, 5 (17.8%) and 6 (21.4%) scored below 39 or above 57 on the TAS-20, which are the cut-off scores for low or high alexithymia, respectively (Vermeulen et al., 2006). An ANOVA conducted on cortisol responses and alexithymia yielded a significant main effect of time ($F_{1,\ 27} = 13.24$, $p < .001$) indicating that cortisol secretion increased in response to stress and then decreased during the recovery period (Fig. 1) but no effect of alexithymia (time × alexithymia interaction: $F_{2,\ 26} = 0.014$, $p = 0.9$). There was no effect of alexithymia on the AUCg (Beta = .171, $t = .885$, NS). Conversely, the analysis revealed a significant effect of alexithymia on baseline (Beta = .370, $t = 2.028$, $p = 0.05$), but not on peak secretion (Fig. 1). To evaluate the weight of alexithymia subfactors we performed three multiple regression analyses with AUCg, Baseline and Peak cortisol as dependent variables. The independent variables, DIFF, DDF and EOT, were entered simultaneously into the model. As shown in Table 1, only the DDF factor was a significant predictor of cortisol response (note that it significantly predicted AUCg and basal cortisol but not peak cortisol). The cortisol response to stress was increased in high EOT scorers and decreased in high DIFF scorers, but in a non-significant manner. The time-dependent cortisol responses in subjects presenting high or low trait alexithymia and subfactors are depicted in (Fig. 1).

Table 1 Multiple regression analyses predicting the cortisol responses by alexithymia subfactors

	Area under the curve (AUCg)		Baseline cortisol		Peak cortisol	
	$F_{(3,\ 24)} = 1.22$, $R^2 = .132$		$F_{(3,\ 24)} = 3.098$*, $R^2 = .279$		$F_{(3,\ 24)} = .201$, $R^2 = .024$	
	Beta	*t*	Beta	*t*	Beta	*t*
DIFF	−.186	−.825	−.135	−.657	−.110	−459
DDF	.448	1.899*	.605	2.811**	.163	.651
EOT	−.117	−.580	−.068	−.372	−.124	−.582

Note: **$p \leq .01$; *$p \leq .05$; †$p \leq .10$.

4. Discussion

Only a few studies to date have tested the relationship between alexithymia and cortisol responses. Lindholm et al. (1990) have shown an association between alexithymia and a positive dexamethasone suppression test. Conversely, McCaslin et al. (2006) have shown no association between alexithymia and cortisol reactivity to a video stress challenge. A positive correlation between alexithymia and norepinephrine/cortisol ratio has been shown in formerly alcohol-dependent men (Henry et al., 1992) and in depressed patients (Spitzer et al., 2005). Our pilot study was novel in that it examined the association of alexithymia, but also of its subfactors on cortisol response to a situation of controlled social stress (TSST). It permitted us to observe that alexithymia was associated with significantly increased cortisol at baseline but not during stress exposure. This effect was mainly due to the effect of DDF. It is of note that subjects scoring low on this factor increased their cortisol from low value at onset to high value at peak whereas high scorers already evidenced maximal cortisol in anticipation of stress (Fig. 1). The possibility that baseline differences could be attributed to differences in the tonic functioning of the HPA axis rather than differences in anticipation may be ruled out by the fact that alexithymia and DDF did not exert a similar effect on cortisol under resting conditions (Mikolajczak, Roy and de Timary, unpublished data). These data are in keeping with the view that biological factors underlie alexithymia's effects on responses to stress or emotions, as suggested by previous imaging studies (see Aleman, 2005 for review). However these studies did not examine the relevance of alexithymia subfactors. The effects of DDF for instance might be related to the finding that affect labelling decreases amygdala activation in response to affective stimuli (Lieber-

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man et al., 2007). Overall these data suggest that alexithymia, and in particular the DDF factor, mainly modulates the anticipation of the stressor rather than the response to the stressor itself and thus likely the cognitive processing of emotions. Such anticipatory appraisals have been shown to contribute largely to the modulation of blood cortisol levels (Gaab et al., 2005). Further studies should test how DDF influences anticipation, in particular by testing how it affects the appraisal of a threat or of a challenge (Gaab et al., 2005). Concerning the clinical relevance of anticipation, Brosschot and colleagues support the importance of perseverative cognitions rather than current stress exposure for the development of somatic diseases. These cognitions, as manifested in worry and rumination in anticipation or in response to stress, will activate the cardiovascular, immune, endocrine and neurovisceral systems and hence prolong stress-related activation, with a possible effect on the expression of somatic diseases (Brosschot et al., 2005). Another interesting aspect is that subjects scoring high on DIFF or EOT had decreased or increased cortisol responses to stress respectively, further supporting the importance of studying the effects of alexithymia at the level of the subfactors. It would be worth studying these effects in larger samples where the effect could be significant.

As above described, in clinical situations like alcoholism (Henry et al., 1992) and depression (Spitzer et al., 2005) the authors observed a positive correlation between alexithymia and norepinephrine/cortisol ratio, which seems not to fit with our observation. However, the observation that the alexithymia subfactors are differentially related to cortisol, raises the possibility that clinical situations that have an effect on some but not all alexithymia subfactors might alter the relationship between alexithymia and cortisol. For instance, DIFF values are known to be increased in subjects presenting with anxious or depressive symptoms, in particular in alcohol-dependent subjects (de Timary et al., 2008). If, as our study suggest, DIFF is negatively correlated to cortisol, it may at least partially explain the observations of Henry et al. (1992) and Spitzer et al. (2005) of an apparently negative correlation between alexithymia and cortisol.

However, our study is not without its limitations. Firstly, our work was not meant to study anticipation or perseverative cognitions and it focussed only on a small sample of normal male subjects. Further studies would therefore greatly benefit from testing whether the current findings can be replicated in clinical populations and in female subjects (females often evidence lower DDF scores (Parker et al., 2003) and lower cortisol responses to public speaking (Kirschbaum et al., 1992)). A second limitation concerns the absence of specific instrument to exclude psychiatric illness and in particular the evaluation of a possible mediating role of depressive mood on the relationship between alexithymia and its subfactors and cortisol. For instance, Finset et al. (2006) observed that cortisol levels increased after a medical interview in alexithymic females suffering from fibromyalgia, but that the observed increase was associated with depressive affect. Because the present sample was composed *a priori* of normal individuals, we decided to control for personality traits instead of anxiety and depression. However, given their association with alexithymia, DIFF and DDF, future studies should control for mood variables such as negative affectivity.

Nonetheless, our findings provide a novel examination of the "alexithymia-stress hypothesis", a hypothesis that had been nearly abandoned, due to the serious discrepancies observed in the numerous electrophysiological studies that had tested it. We suggest that further studies on this topic should take into account the importance of alexithymia subfactors and focus on how alexithymia can influence appraisal, anticipation of stressors or perseverative stress-related cognitions. Such studies may increase the understanding of the relationship between alexithymia and diseases, a clinically relevant question, as psychological interventions designed to improve the appraisal of stressful situations might also have an effect on the expression of diseases.

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Conflict of interest

None declared.

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