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Alexithymia and anxiety sensitivity in populations at high risk for panic disorder

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Abstract

Objective: Populations at high risk for panic disorder (PD) probably share with subjects with PD an underlying vulnerability involving features like anxiety sensitivity (AS) and alexithymia. The present study would verify if PD relatives (R) and subjects who have experienced 1 or more panic attacks (PAs) show different levels of AS and alexithymia with respect to healthy controls (HC).
Methods: One hundred fifty-seven HCs, 30 subjects with PA, 64 R subjects, and 139 outpatients with PD were evaluated and compared on AS, alexithymia, and control variables.
Results: Subjects with PD show higher alexithymia and AS levels compared with HCs; R subjects do not differ on ASI total score; and R females show more alexithymic features. Subjects with PA are comparable with HCs both on AS and alexithymia.
Conclusions: Results confirm an impairment in emotional and bodily sensations information processing in subjects with PD but partially disconfirm the expectation of a difference between R subjects and subjects with PA with respect to HCs on AS and alexithymia. Emotional and bodily sensation competencies could be protective factors for PD in high-risk populations.

1. Introduction

Several authors have pointed out that in anxiety disorders, in particular panic disorder (PD), amplification of somatic sensations is often associated to subsequent dysfunctional cognitive appraisal of these sensations with a significant bias toward a danger-related and catastrophizing interpretational style [1,2]. The construct of *anxiety sensitivity* (AS) [3] refers to the tendency to fear anxiety-related sensations. According to Reiss [4], individuals with elevated AS experience amplified fear in response to stimuli that elicit anxiety and find their own anxiety symptoms to be particularly aversive. Many studies have found large associations between AS and PD features [5-8] (for a meta-analysis, see Naragon-Gainey [9]). Some authors evidenced that the way that individuals appraise anxiety- and panic-related symptoms may increase the risk of developing agoraphobia, over and above the risk of developing PD [10]. Prospective studies have shown that AS predicts the onset of anxiety and panic in both nonclinical and clinical populations [11].

A second interesting psychological construct pertaining to bodily sensations (related to emotion-related activation) information processing is alexithymia. The *alexithymia construct*, as defined by Nemiah et al [12], has 3 salient features: (1) difficulty identifying and describing subjective feelings, (2) difficulty distinguishing between feelings and the bodily sensations of emotional arousal, and (3) constricted imaginal capacities, as evidenced by paucity of fantasies and externally oriented cognitive style. The alexithymia construct reflect deficits in the cognitive processing and regulation of emotions [13].

As reported by Galderisi and colleagues [14], empirical evidence of poor emotion processing in subjects with PD has been provided, including a high prevalence of alexithymia. Regarding PD, the psychological construct of alexithymia has been extensively studied from different points of view [15-18]: overall results proved an alexithymic deficit in subjects with PD. In particular, the first feature of alexithymia seems to distinguish healthy control (HC) subjects from subjects with PD and to predict symptom severity. However, as evidenced in the study by Marchesi et al [18], the possibility of a trait or state interpretation of this construct remains controversial.

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Converging lines of evidence suggest that PD in adulthood may represent manifestations of an underlying constitutional vulnerability or diathesis for anxiety that is partly genetic and variably expressed during a life cycle [19]. It is well known that PD runs in families [20,21].

Individual differences in AS and alexithymia are hypothesized to emerge from both genetic and maladaptive learning experiences that lead to the acquisition of beliefs about the aversive consequences of arousal and anxiety-related states [22].

A recent study by Stein and colleagues, examining the heritability of AS in a group of 179 monozygotic and 158 dizygotic twin pairs, suggests that variation in AS (particularly the physical aspect of AS) is explained by unique environmental and additive genetic influences [23]. Emotional maltreatment in childhood, particularly when it includes parental threatening, hostile, and rejecting behaviors, is associated with higher levels of AS in young adults [24]. In a familial study, van Beek and Griez [25] found that the first-degree relatives of subjects with PD were significantly more anxiety sensitive than HC subjects, but less anxiety sensitive than patients with PD, suggesting that AS runs in families.

Moreover, Stein et al [26] observed a statistically significant interaction between levels of childhood emotional (or physical) maltreatment and 5-HTTLPR genotype. Specifically, S/S individuals with higher levels of maltreatment had significantly higher levels of AS than subjects in other groups, evidencing a gene-by-environment (serotonin transporter and childhood maltreatment) interaction process influencing AS.

In the same way, several authors studied genetic and environmental factors associated with alexithymia. For example, Ham et al [27] showed an association between the Val/Val COMT genotype and higher Toronto Alexithymia Scale (20-item version; TAS-20) scores in a sample of 109 students. Valera and Berenbaum [28], in a study involving 45 monozygotic and 32 dizygotic twin pairs, concluded that familial influences contribute to alexithymia, with a specific effect of environmental factors on difficulty in identifying feelings and communicating emotions and a specific effect of shared genetic factors on externally oriented thinking. In a larger sample composed of 8785 twin pairs, Jørgensen et al [29], using a structural equation modeling, selected an ACE model including additive genetic, shared environmental, and nonshared environmental effects providing the best fit for all 3 facets of alexithymia as well as total alexithymia scores, with heritabilities of 30% to 33% and the remaining variance being explained by shared (12%-20%) and nonshared environmental effects (50%-56%).

Two categories of subjects have been proposed to be populations at high risk for the development of PD: relatives of subjects affected by PD [20] and subjects who have had almost a panic attack without developing the psychiatric syndrome [30].

Genetic and environmental variables are thought to be involved both in the etiology of PD and influencing the development and regulation of metacognitive abilities such as the access to one's own emotions and feelings and the interpretation of bodily sensations, contributing to a description of a liability spectrum for developing the clinical features of the disorder.

Actually, no studies have systematically investigated the differences in these variables between subjects with PD, high-risk populations (first-degree relatives and subjects who have had 1 or more panic attacks without developing PD), and healthy subjects.

We hypothesize that the distribution of alexithymia and AS among these categories of subjects could provide important information about their specific role in characterizing low- and high-risk populations.

In particular, we would verify if first-degree relatives of subjects with PD and subjects who have had almost a panic attack without developing the psychiatric syndrome show higher levels of AS and alexithymia with respect to HCs.

2. Methods

2.1. Sample

After the approval of the study protocol by the local ethics committee, 4 distinct samples were recruited for the study purposes. The clinical sample, composed of 139 outpatients affected by PD with agoraphobia, was consecutively recruited from the Unit of General Psychiatry of the Department of Clinical Neurosciences of the University Vita-Salute, San Raffaele Hospital of Milan, using a subsequent admission method of recruitment. All subjects undertook an average of 1-hour assessment, including a diagnostic and anamnestic interview, and, afterward, another 1-hour session with the administration of the Beck Depression Inventory II (BDI-II) [31], the TAS-20 [32] (Italian version; [33]), and the Anxiety Sensitivity Index (ASI) [34].

Axis I and II diagnoses were defined according to *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition*'s (DSM-IV) criteria, using the Italian version of the Structured Clinical Interview for *DSM-IV* Axis I and II Disorders. All the interviews are administered by trained clinicians who are expert in anxiety disorders' diagnosis and treatment, whereas the BDI-II, the TAS-20, and the ASI are self-administered and followed by trainee psychologists. Patients with comorbid diagnoses were not excluded, provided that PD was the main problem for which treatment was sought. Exclusion criteria were brain injury or trauma, any neurologic condition, psychosis, and substance abuse. Patients with PD were recruited before starting therapy (which could have been pharmacologic therapy, CBT, CBT plus pharmacotherapy, and hospitalization).

Sixty-three patients were under medication at the time of the assessment, 32 of whom were taking selective serotonin reuptake inhibitors, 15 benzodiazepine, and 16 selective