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Distinguishing affective depersonalization from anhedonia in major depression and bipolar disorder

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Abstract

Background: Affective depersonalization has received limited attention in the literature, although its conceptualization may have implications in terms of identification of clinical endophenotypes of mood disorders. Thus, this study aims to test the hypothesis that anhedonia and affective depersonalization represent 2 distinct psychopathological dimensions and to investigate their clinical correlates in patients with major depressive disorder (MDD) and bipolar disorder (BD).

Methods: Using a data pool of 258 patients with mood and anxiety disorders, an item response theory–based factor analysis approach was carried out on 16 items derived from 2 clinical instruments developed in the Spectrum Project (the Structured Clinical Interview for Mood Spectrum and the Structured Clinical Interview for Derealization-Depersonalization Spectrum). Clinical correlates of these psychometrically derived dimensions were subsequently investigated in patients with BD or MDD.

Results: Using an item response theory–based factor analysis, a 2-factor solution was identified, accounting overall for the 47.0% of the variance. Patients with BD showed statistically significant higher affective depersonalization factor scores than those with MDD ($Z = 2.215$, $P = .027$), whereas there was no between-groups difference in anhedonia scores ($Z = 0.825$ $P = .411$). In patients with BD, age of onset of the disease correlated with affective depersonalization factor scores ($\rho = -0.330$, $P = .001$) but not with anhedonia factor scores ($\rho = -0.097$, $P = .361$).

Conclusions: Affective depersonalization and anhedonia seem to be 2 distinct psychopathological dimensions, although closely related, bearing the opportunity to identify patients with a specific profile for a better clinical and neurobiological definition.

1. Introduction

Current diagnostic practice is guided by *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition* (*DSM-IV*), which provides a polythetic, categorical approach to classification of psychiatric disorders, currently validated by empirical studies. However, *DSM-IV* classification has some important limitations. Among them is the failure of a categorical system to capture specific manifestations associated with a given disorder. The dimensional approach to psychiatric disorders has been proposed as complementary to the classical categorical one, for a better definition of clinical endophenotypes in terms of prognosis and better tailored therapies [1-4]. Moreover, psychopathological dimensions have been claimed to have a much more apparent biological foundation [5].

Although it can be assumed that specific psychopathological dimensions involve physiologic arousal, appraisal, subjective experience, and goal directed behavior, there is still limited understanding of their neurobiological bases [6]. This is partly due to the complexity of distinguishing psychopathological constructs that have a wide overlap.

Depression involves several psychopathological components, and different authors adopted a factor analysis approach to obtain a map of symptom components of depression [7-11]. However, the identification of specific dimensions is strictly related to the adopted assessment instrument. Although anhedonia is recognized as an important component of depression [12-14] and is one of the diagnostic criteria for the *DSM-IV* diagnosis of major depression with melancholia [15], affective depersonalization (AD) is not specifically investigated by most clinical instruments used to assess patients with mood disorders.

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Anhedonia is defined as a diminished or abolished capacity to experience pleasant emotions and was introduced as one of the core symptoms of the endogenous (or melancholic) subtype of major depression in *DSM-III* [16]. The development of anhedonia as a construct was influenced by Klein's conceptual framework of "endogenomorphic" depression, which referred to a particular type of depression characterized by a pervasive impairment of the capacity to experience pleasure or to respond affectively to the anticipation of pleasure [17]. Anhedonia was found to correlate significantly with neuroticism, introversion, and morbid risk of depression in first-degree relatives [18]. Furthermore, in his classification of chronic depression, Akiskal [19] described a condition characterized by the presence of neurovegetative change and anhedonia as an antidepressant-responsive form of chronic depression.

On the contrary, the conceptualization of AD referred to the classical definition given by Wernicke [20], subsequently elaborated by Jaspers [21], representing the inability of the self to imbue perceptions with emotional feelings. Although studies on patients with depersonalization disorder pointed out that AD is a homogenous construct [22,23], data on patients with mood disorders are still scanty. In classical psychiatric writings, AD can be connected with endogenous depression through the concept of loss of feelings or *Gefühl der Gefühllosigkeit* [24]. Its conceptualization needs to be differentiated from that of anhedonia because it goes beyond a general loss of affection or pleasure consisting of a number of different manifestations such as the loss of fear or disgust to situations previously avoided. Still, AD needs to be distinguished from the *délire des negations*, also known, in English literature, as Cotard syndrome [25]. In fact, although the "emotional anesthesia," typical of AD, is regarded as an important feature of the Cotard syndrome [26], the subsequent analysis by Young and Leafhead [27] of original Cotard cases revealed a series of other commonly occurring symptoms such as self-deprecatory delusions, suicidal ideation, feelings of guilt, and denial of body parts.

On this background, it is evident that both anhedonia and AD belong to depression, and a conceptual differentiation between them may have important practical implications not only in terms of a better definition of clinical endophenotypes of mood disorders for prognosis and response to treatments but also because it may allow further research on the neurobiology and neuropsychology of emotional processing. Therefore, this study is aimed to test the hypothesis that anhedonia and AD represent 2 different psychopathological dimensions in a large sample of patients with mood and anxiety disorders and to investigate their clinical correlates in patients with major depression and bipolar disorder (BD).

2. Methods

2.1. Participants

A consecutive sample of adult outpatients presenting for treatment of mood and anxiety disorders at the Department of Psychiatry in Pisa, Italy, from September 2006 to September 2007 were invited to participate in the study.

Exclusion criteria were severe medical illnesses, neurological diseases, and inability to participate because of the severity of psychiatric symptoms. Eligible subjects provided written informed consent after receiving a complete description of the study and having an opportunity to ask question. The Ethics Committee of the Azienda Ospedaliera Universitaria of Pisa approved all recruitment and assessment procedures.

2.2. Instruments

2.2.1. Diagnostic assessment

The diagnostic interviews consisted of the administration of the Structured Clinical Interview for *DSM-IV* Axis-I disorders [28] and a structured clinical interview consisting of 16 items derived from 2 instruments developed in the context of the Spectrum Project, namely, the Structured Clinical Interview for Mood Spectrum (SCI-MOODS) lifetime version [29] and the Structured Clinical Interview for Derealization-Depersonalization Spectrum (SCI-DER) lifetime version [30].

Cassano et al [3,31,32] observed that patients with psychiatric disorders may manifest a "spectrum" of associated clinical features not included in the criteria set. Some are "trait-like" symptoms that occur as subtle manifestations of an illness diathesi. Early onset of such symptoms may act to shape developing mental functions and change the personality. Failure to recognize and attend to residual and comorbid spectrum features in treating *DSM-IV* disorders may explain continued clinical impairment, even when core Axis I symptoms have been successfully treated [33]. To operationalize such spectrum model, researchers of the Spectrum Project developed a number of clinical instruments that proved to be valid and reliable (www.spectrum-project.net). All instruments have been developed with a similar structure to be used all together in a global dimensional assessment of psychopathology. In fact, all instruments explore the "presence" or "absence" of spectrum feature in the lifetime. Instrument versions exploring the last-month time frame are also available.

2.2.2. The Structured Clinical Interview for Derealization-Depersonalization Spectrum

The SCI-DER was developed and validated at the Department of Psychiatry, Neurobiology, Pharmacology and Biotechnologies of the University of Pisa by experienced psychiatrists. It includes 49 items exploring presence or absence of lifetime symptoms of depersonalization organized into 4 domains: (1) derealization, (2) somatopsychic depersonalization, (3) autopsychic depersonalization, and (4) AD. Items responses are coded in a dichotomous way (yes/no), and total and domain scores are obtained by counting the number of positive answers. The SCI-DER proved to have sound psychometric properties: an excellent internal consistency (0.92), high test-retest reliability at 15 to 20 days (r = 0.88), and high convergent validity with respect to the Dissociative Experiences Scale (r = 0.74), and