Journal of Affective Disorders 147 (2013) 446–450

Contents lists available at SciVerse ScienceDirect

Journal of Affective Disorders

journal homepage: www.elsevier.com/locate/jad

Brief report

Reduced hedonic capacity in euthymic bipolar subjects: A trait-like feature?

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ARTICLE INFO

Article history:
Received 13 March 2012
Received in revised form
4 October 2012
Accepted 5 October 2012
Available online 2 November 2012

Keywords:
Anhedonia
Affective disorders
Dopaminergic system
Reward circuit

ABSTRACT

Background: The aim of our study was to assess hedonic capacity in euthymic bipolar subjects, identifying possible differences compared to remitted unipolar depressed patients and healthy controls.
Methods: 107 subjects with bipolar disorders, 86 with major depressive disorder and 106 healthy controls, homogeneous with respect to demographic characteristics, were enrolled. The following scales were administered: the Snaith–Hamilton pleasure scale (SHAPS), the subscale for 'anhedonia/asociality' of the scale for the assessment of negative symptoms (SANS) and the visual analogue scale (VAS) for hedonic capacity.
Results: Scores on SHAPS total, interests and social interactions, SANS 'anhedonia/asociality' and VAS were all significantly higher in affective disorder patients compared to healthy controls. No difference was found between clinical groups. 20.5% ($n=22$) of bipolar disorder subjects and 24.5% ($n=21$) of major depressed subjects showed a significant reduction in hedonic capacity (SHAPS total score ≥ 3), compared to 7.5% ($n=8$) of healthy controls ($\chi^2=12.03$; $p=.002$).
Limitations: Limitations include heterogeneity with respect to pharmacological status and longitudinal course (i.e., 'single' vs. 'recurrent' affective episodes).
Conclusions: The major finding of our study is that euthymic bipolar patients and remitted major depressed patients display residual anhedonic symptoms. This suggests that, in affective disorder patients, altered hedonic capacity could represent an enduring trait and that, possibly, dysfunctions in the neurobiological mechanisms underlying hedonic response and reward processing persist, irrespective of mood state.

1. Introduction

Ribot (1896) first described anhedonia as the inability to experience pleasure. The DSM recognized anhedonia in its third edition and currently defines it as diminished interest or pleasure in response to stimuli perceived as rewarding during the premorbid state (American Psychiatric Association, 2000).

Anhedonia has long been considered a core manifestation of depression, insomuch that Klein posited the existence of a subtype of major depression, referred to as *endogenomorphic* depression, marked by a characterological anhedonic trait (Klein, 1987). Anhedonia is currently one of two required symptoms for the diagnosis of major depressive disorder (American Psychiatric Association, 2000). The anhedonic experience is central to both unipolar and, to a lesser extent, bipolar depression; recent reports estimate that approximately 37% of individuals diagnosed with depression experience clinically significant anhedonia (Pelizza and Ferrari, 2009). Anhedonic symptoms are particularly difficult to treat (Dunlop and Nemeroff, 2007; Nutt et al., 2007; Price et al., 2009; Martinotti et al., 2012) and their presence predicts poor treatment outcome (Spijker et al., 2001).

A variety of brain regions, neural circuits and neurotransmitters have been implicated in the psychopathological expression of anhedonia. Dysfunction of the dopaminergic mesolimbic and mesocortical reward circuit has been singled out as the major neurobiological correlate of anhedonia (Isella et al., 2003; Willner et al., 2005).

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http://dx.doi.org/10.1016/j.jad.2012.10.004

Though a vast number of studies have stressed the importance of anhedonia in major depressive disorder (Loas, 1996; Hasler et al., 2004; Pizzagalli et al., 2008), few studies have explored the relationships occurring between anhedonia and bipolar disorder (Etain et al., 2007; Docherty and Sponheim, 2008). Excessive pleasure-seeking behavior and reduced hedonic capacity observed during manic and depressive episodes, respectively, point to changes in the neurobiological mechanisms underpinning pleasure, reward and motivation. Neuroimaging studies have, in fact, evidenced an association between bipolar disorder and dysfunction in brain regions coding the representation of reward values (mainly, the orbitofrontal cortex) (Drevets et al., 1998; Blumberg et al., 2003).

According to certain theoretical models, a noteworthy down-regulation of dopaminergic transmission follows the hyperdopaminergic state observed in mania, accounting for the emergence of anhedonia during depressive and even euthymic states (Berk et al., 2007). On this premise, some studies have provided preliminary evidence indicating that bipolar disorder patients present abnormalities in reward-processing circuitry, regardless of mood states. Euthymic children with bipolar disorder, relative to comparison subjects, made more mistakes on a response-reversal task and seemed to have greater difficulty adapting to changing reward contingencies (Gorrindo et al., 2005). Similarly, a significant portion of euthymic bipolar subjects exhibited blunted reward learning during a probabilistic reward task and reported residual anhedonic symptoms (Pizzagalli et al., 2008).

The aim of the present study was to assess hedonic capacity in euthymic bipolar subjects, given the relatively limited attention it has so far received. Based on the premises that bipolar disorder's euthymic phase is not entirely symptom-free but is actually an integral part of the disease and that anhedonia is a psychopathological dimension linked to specific neurobiological substrates, we hypothesized that reduced hedonic capacity would persist in euthymic patients, thereby constituting a trait abnormality. Furthermore, we compared bipolar disorder patients' hedonic response with that of remitted unipolar depressed patients and a group of non-psychiatrically ill individuals, in order to highlight any differences.

2. Methods

2.1. Participants and procedure

Data were obtained from 193 affective disorder outpatients (107 with bipolar disorders and 86 with major depressive disorder) admitted, from September 2009 to December 2011, to the affective disorders unit of the Day-Hospital of Psychiatry of the University General Hospital "A. Gemelli" in Rome. Also, 106 healthy controls were randomly recruited amongst community volunteers.

Inclusion criteria were: age 18–65; DSM-IV-TR diagnosis of bipolar disorder (type I, II, Cyclothymia, NOS) or major depressive disorder; euthymia/remission for at least 6 months, based on clinical and psychometric evaluations [clinical global impression-severity 1–2 (Guy, 1976); Hamilton depression rating scale <8 (Hamilton, 1960); Young mania rating scale <6 (Young et al., 1978)]. Exclusion criteria were: psychotic features; substance/alcohol abuse in the past 2 months; organic brain syndromes or any other medical condition with a possible psychiatric manifestation; mental retardation or documented IQ <70.

Affective disorders diagnoses were established using the structured clinical interview for DSM-IV Axis I disorders (SCID-I) (First et al., 1995). Personality disorders were diagnosed with the structured clinical interview for DSM-IV Axis II disorders (SCID-II) (First et al., 1990).

The study was conducted in accordance with the declaration of Helsinki and with good clinical practice guidelines, and was approved by the local ethical committee. All patients gave their written informed consent, after a complete description of the study was provided.

2.2. Psychometric assessment

The Snaith–Hamilton pleasure scale (SHAPS; Snaith et al., 1995) is a 14-item self-rating scale exploring hedonic responses in common pleasurable situations related to leisure pursuit and interests, eating and drinking, social interactions and sensory experiences. A total score ≥ 3 indicates a significant reduction in hedonic capacity (anhedonia).

The 'anhedonia/asociality' subscale of the scale for the assessment of negative symptoms (SANS; Andreasen, 1989) is a clinician-rated 5-item scale, rated on a 5-point Likert scale ranging from 0 (absent) to 4 (severe). It evaluates hedonic state deficit during pleasant activities.

The 10 cm visual analogue scale (VAS; Aitken, 1969; Mottola, 1993) for hedonic capacity: subjects specify their level of hedonic capacity by indicating a position along a continuous line between two end-points.

2.3. Statistical analysis

Statistical analysis was conducted using SPSS for Windows, Versions 15.0 (SPSS Inc, Chicago, Illinois). We compared demographic and clinical characteristics between bipolar disorder patients, major depressed patients and healthy controls. For all binary comparisons of categorical variables, either the chi-square test or Fisher's exact test was used. Because the number of subjects in the different groups was uneven and the assumptions for parametric testing (normal distribution of values, homogeneity of variances) were violated for most of the variables, Kruskal Wallis non-parametric one-way ANOVA was performed in order to compare continuous variables in the 3 independent groups. When the 3-group comparison was significant, Mann–Whitney U test with a Bonferroni-corrected significance level was applied to pairwise comparisons between groups in order to identify the source of the significance. Spearman's rank correlation coefficient was employed to examine the relationship between continuous variables. All tests were 2-tailed, with statistical significance set at $p<.05$.

3. Results

3.1. Demographic and clinical data

Subjects' demographic and clinical data are presented in Table 1. All participants were Caucasians and were homogeneous for age ($p=.284$), gender ($p=.446$), and level of education ($p=.111$).

23% ($n=25$) of bipolar subjects and 19% ($n=16$) of major depressed patients had a co-morbid axis I disorder (generalized anxiety disorder: $n=6$; social anxiety disorder: $n=7$; panic disorder: $n=9$; agoraphobia: $n=6$; anorexia nervosa: $n=7$; bulimia nervosa, $n=6$) with no significant difference between groups.

An Axis II diagnosis was observed in 23% ($n=24$) of bipolar subjects and in 19% ($n=16$) of unipolar depressed patients, with no significant differences between groups; in particular, histrionic ($n=6$), narcissistic ($n=5$), borderline ($n=6$), antisocial ($n=6$), obsessive–compulsive ($n=4$), dependent ($n=4$), avoidant ($n=4$),