Course and moderators of emotional eating in anorectic and bulimic patients: A follow-up study

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Abstract

Emotion dysregulation has been found to be associated with specific eating attitudes and behavior in Eating Disorder (ED) patients. The present study evaluated whether emotional eating profile of ED patients changes over time and the possible effects of a psychotherapeutic intervention on the emotional eating dimension. One hundred and two ED patients (28 with Anorexia Nervosa restricting type [AN-R], 35 with Anorexia Nervosa binge/purging subtype [AN-B/P] and 39 with Bulimia Nervosa [BN]) were evaluated at baseline, at the end of a Cognitive Behavioral Therapy, at 3 and 6 year follow-up. The Structured Clinical Interview for DSM IV Axis I Disorders, the Emotional Eating Scale (EES) and several self-reported questionnaires for eating specific and general psychopathology were applied. A control group of 86 healthy subjects was also studied, in order to compare psychopathological variables at baseline. A significant EES total score reduction was observed among AN-B/P and BN patients, whereas no significant change was found in the AN-R group. Mixed Models analyses showed that a significant effect on EES total score variation was found for cocaine or amphetamine abuse (b = .25; p < .01). Patients who assumed these substances reported no significant EES reduction across time, unlike other patients. The present results suggest that ED patients with a history of cocaine or amphetamine abuse represent a subpopulation of patients with lasting dysfunctional mood modulatory mechanisms.

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1. Introduction

Emotion dysregulation has recently been considered a trans-diagnostic factor related to multiple types of psychopathology (Ehring & Watkins, 2008), and different associations between emotion dysregulation and symptoms of depression, anxiety, and substance abuse have been observed (Aldao, Nolen-Hoeksema, & Schweizer, 2010). As far as Eating Disorder (ED) are concerned, patients often show relevant difficulties in emotion regulation, due to the lack of the skills required to cope with negative affective states (Svaldi, Grievensthoef, Tuschens-Caffrey, & Ehring, 2012), and different authors consider ED symptoms (e.g. binge eating and purging behaviors) as pathological responses to regulate intense or relatively undifferentiated emotional states, to restrict the affective experience or to deviate attention from negative emotions (Torres et al., 2010; Treasure, Claudino, & Zucker, 2010; Zaitsoff & Grilo, 2010; Espeset, Guliksen, Nordbø, Skårderud, & Holte, 2012).

Emotional eating (EE) has been defined as the tendency to eat in response to emotional states (Arnow, Kenardy, & Agras, 1995), and it was identified as a possible factor triggering binge eating in Bulimia Nervosa (BN) (Engelberg, Steiger, Gauvin, & Wonderlich, 2007), and in Binge Eating Disorder (BED) (Masheb & Grilo, 2006; Zeeck, Steilzer, Linster, Joos, & Hartmann, 2010). This construct is not merely focused on eating behavior and overeating, but it specifically addresses the feelings that lead people to experience an urge to eat and the desire of consuming food in response to different emotions (Arnow et al., 1995). Recent studies suggested that EE is not only associated with the presence of binge eating, but is also a common dimension in all EDs (Courbasson, Rizea, & Weiskopf, 2008; Torres et al., 2010; Ricca et al., 2012). In particular, it has been observed that EE is significantly associated with specific eating attitudes and behavior, according to the different ED diagnoses: restraint for patients with Anorexia Nervosa restricting type (AN-R), subjective binge eating for patients with Anorexia Nervosa binge/purging type (AN-B/P), and objective binge eating for patients with BN (Ricca et al., 2012). Overall, these findings seem to support those models which conceptualize EDs as syndromes that are characterized by the impairment in the cognitive capacity to process and regulate emotions as the primary regulatory disturbance (Kenardy, Arnow, & Agras, 1996; Stein et al., 2007).

However, despite the relevant role of emotional eating in EDs, to our knowledge there are no previously published studies that have evaluated the course of emotional eating in a clinical setting. Therefore, it is unknown whether emotional eating profile of ED patients changes over time and which are the possible effects of a psychotherapeutic intervention on this psychopathological dimension.

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The aims of the present study were as follows:
- to evaluate the course of emotional eating over time in patients with AN and BN
- to assess the possible predictors of emotional eating change.

2. Methods

The study was performed at the Eating Disorder Outpatient Clinic of the Psychiatric Unit of the Careggi University Hospital of Florence, Italy. A written informed consent was obtained from each patient and the protocol was approved by the Ethics Committee of the Institution. All patients who attended the Outpatient Clinic between June 2003 and December 2005 were included in the study. The exclusion criteria were: 86 subjects, aged mean 28.86 ± 7.70 years, with a mean BMI of 22.02 ± 2.99 kg/m². Eight subjects were excluded from the initial list of controls because of the presence of any actual and lifetime eating disorder or other psychiatric diagnosis, according to DSM-IV criteria, evaluated by means of a face-to-face clinical interview, according to specific questions extracted from the Eating Disorder Examination Interview (EDE 12.00) (Fairburn & Cooper, 1993) and from DSM-IV (Text Revision) (American Psychiatric Association, 2000). Moreover participants completed the following self-reported questionnaires, in order to collect data on eating and general psychopathology: the Emotional Eating Scale (EES) (Arnow et al., 1995), the Eating Disorder Examination Questionnaire (EDE-Q) (Fairburn & Beglin, 1994), the Beck Depression Inventory (BDI) (Beck & Steer, 1987), the State-Trait Anxiety Inventory (STAI form Y-1) (Spielberger, Gorsuch, & Lushene, 1970), the Barratt Impulsivity Scale (BIS-11) (Patton, Stanford, & Barratt, 1995) and the Symptom Checklist (SCL-90-R) (Derogatis, Liptman, & Cov, 1973). Emotional Eating Scale is a 25-item self-report questionnaire that indicates the extent to which specific feelings lead a subject to feel an urge to eat. Each item consists of an emotion term (e.g., jittery, angry, and helpless), and the 5-point scale used was anchored on a continuum from “no desire to eat” to “an overwhelming urge to eat”. The 25 items form 3 subscales, reflecting eating in response to anger (Anger/Frustration), anxiety (Anxiety), and depressed mood (Depression). These 3 subscales refer to the emotional antecedents of binge eating, and on each scale, higher scores reflect a greater tendency to eat in response to emotional state. The EES is internally consistent, it demonstrates temporal stability and it has good construct, criterion, and discriminant validity (Arnow et al., 1995). The current sample internal consistency was good (Cronbach’s alpha: 0.92). The self-reported EDE-Q consists of 38 items, assessing the core psychopathological features of eating disorders, and contains 4 subscales: dietary restraint, eating concern, weight concern, and shape concern. The dietary restraint subscale is an admixture of cognitions and behaviors pertaining to dietary restriction. The three other subscales evaluate the dysfunctional attitudes regarding eating and overvalued thoughts regarding weight and shape. The global score represents the mean of the four subscale scores (Fairburn & Beglin, 1994). The EDE-Q has good concurrent validity (Mond, Hay, Rodgers, Owen, & Beumont, 2004a, 2004b), a high degree of temporal stability (Mond et al., 2004a, 2004b) and reliability (Berg, Peterson, Frazier, & Crow, 2012). In the current sample internal consistency was good (Cronbach’s alpha: 0.80). The Beck Depression Inventory (Beck & Steer, 1987) is a 21-item self-reporting scale that assesses the severity of affective, cognitive, motivational, vegetative, and psychomotor components of depression. Each item is a list of four statements arranged in increasing severity of a particular symptom of depression; the higher the score, the higher is the depression. The BDI showed excellent psychometric properties (Beck, Steer, & Carbin, 1988). In the current sample Cronbach’s alpha was 0.91. The STAI (Spielberger et al., 1970) is a 40-item measure that indicates the intensity of feelings of anxiety. It distinguishes between state anxiety (i.e., a temporary condition experienced in specific situations) and trait anxiety (i.e., a general tendency to perceive situations as threatening). It showed good psychometric properties (Spielberger et al., 1970) and in the present sample internal consistency was good (Cronbach’s alpha was 0.91). The BIS-11 (Patton et al., 1995) is a questionnaire designed to assess the personality/behavioral construct of impulsiveness. It is the most widely cited and psychometrically sound instrument for the assessment of impulsiveness. Cronbach’s alpha in the current study was 0.83.

Finally, SCL-90-R (Derogatis et al., 1973) is a good psychometric instrument devoted to the identification of the psychopathologic distress. In the current sample Cronbach’s alpha was 0.95.

Chi-square test and one-way analysis of variance (ANOVA) test for independent samples with Tukey’s post hoc test were performed to compare socio-demographic and clinical variables between groups. ANOVA Mixed Models with random intercept were performed to evaluate emotional eating levels over time and the effect of the baseline variables on emotional eating change. EES total score was entered as dependent variable and age, BMI, age of onset, psychiatric comorbidities, ED diagnosis, and psychopathological variables at the baseline were entered as covariates, considering them as potential moderators of emotional eating change across time.

The Statistical Package for the Social Sciences (SPSS; SPSS Inc., Chicago, IL) for Windows 18.0 was used for data analysis.

3. Results

3.1. Study variables at baseline

Descriptive statistics of the study variables at the baseline for the three groups and controls are reported in Table 1. Patients and controls
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