

Depressive and Anxiety Symptoms in the Outcome of Eating Disorders: 8-Year Follow-Up

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Abstract

Introduction: Eating Disorders (EDs) are severe and treatment-resistant conditions whose psychopharmacological treatments are still limited. Anxiety and depressive symptoms and specific personality traits play a key role in ED outcome. This 8-year follow-up study on a sample of ED patients treated with a multimodal model aims to produce a better insight in the intertwined aspects of anxiety, depression, and eating psychopathology.

Methods: n=107 female individuals with EDs were assessed face-to-face 8 years after discharge from the outpatient facility of the Center for EDs using: Temperament and Character Inventory, Eating Disorder Inventory-2, Symptom Checklist 90, and Beck Depression Inventory-II. All outpatients underwent a multimodal treatment. Recovered and non-recovered subgroups were compared with ANCOVA. Multivariate regression analyses were performed between changes in personality, anxiety, and depression with those in eating symptoms and psychopathology.

Results: Both ED samples were found to be improved in depression and anxiety along with ED symptoms and eating psychopathology at follow-up. Also the non-recovered individuals showed a significant improvement of anxiety and depression scores along with eating symptoms and psychopathology and personality. The relationship between changes in personality traits and eating psychopathology was stronger than the one we found between personality and anxiety and depression.

Conclusion: The multimodal treatment may be effective to significantly improve mood and anxiety features of ED patients jointly with those in eating symptoms and psychopathology, and related personality traits. Even though the changes in personality may be relevant in order to stabilize eating symptoms, such changes do not significantly correlate with those of anxiety and depression in AN or BN subgroups. Moreover, the course of eating psychopathology is rather independent from that of anxiety and depression over the long-run. The relationship between psychopathology of anxiety and depression with eating symptoms and treatment resistance deserves further exploration to help clinicians in treatment planning.

Keywords: Anorexia nervosa; Bulimia nervosa; Anxiety; Depression, Eating psychopathology, Personality traits; Outcome; Multimodal treatment

Introduction

Eating Disorders (EDs), anorexia nervosa (AN) and bulimia nervosa (BN) are severe, enduring, and treatment resistant conditions with the highest mortality rates of any psychiatric disorders [1,2]. Despite their severity, according to NICE guidelines [3] there is yet no evidence-based treatment for these illnesses. Psychopharmacological treatments for EDs are limited and show little efficacy [4], maybe as a consequence of severe low weight, psychiatric comorbidity, and specific personality traits of those affected by an ED, and patients' suboptimal cognitive abilities and ambivalence towards change [5-7]. However, multimodal strategies focused on multidisciplinary models encompassing individual/group psychotherapy, family therapy/ counseling, psychopharmacological treatment, and nutritional rehabilitation [8,9] were found to improve ED symptoms in affected individuals [10,11]. Psychiatric comorbidity is relevant with respect to treatment and outcomes. In fact, major depression in ED patients varies from 13 to 70% in AN and it is about 40% in BN [12]. If we consider also subthreshold mood disorders, the percentage rises up to 98% [13]. Recently, studies on weight-restored AN patients, demonstrated a significant reduction of depressive symptoms [14]. Studies on bingeeating disorders demonstrate a relationship between a reduction in Body Mass Index (BMI) and a reduction in depressive symptoms [15].

Also anxiety disorders affect patients with EDs more often than the general population [16]. Avoidance strategies and safety behaviors have been suggested as mechanisms linking EDs and anxiety [17] with a body of evidence highlighting early onset anxiety as a predisposing factor for developing EDs [18].

Comorbidity with anxiety and depression also influences the outcome of EDs. In fact, according to outcome studies [19-21] comorbid depression or anxiety are related to persisting ED symptoms, poorer health and general functioning, and higher rates of mortality. Comorbid psychiatric disorders, including depression, also predict lower rates of remission with family-based treatment for AN

[22,23]. Nevertheless, findings are overall mixed, and the relationship between anxiety and depressive symptoms with eating psychopathology has still to be defined mostly over the long-run [24-26].

Another point of interest concerns the relationship between anxious and depressive symptoms with personality traits and onset, prognosis, and outcome of EDs [27,28]. In fact, a specific pattern of personality traits, as measured by the Temperament and Character Inventory (TCI), strongly characterizes EDs. High harm avoidance (HA) and low self-directedness (SD) are associated with low novelty seeking (NS) in AN and with high NS and reward dependence in BN [29,30]. On the other hand, high HA and low SD also characterize depressive and the majority of anxiety disorders potentially representing a crucial crossroad for the evolution of both disorders [6,31].

We hypothesized that the multimodal treatment would mainly affect personality traits in order to produce long-lasting symptom changes in patients with EDs. If so, a linear relationship between changes in anxiety and depression and those of personality as well as eating psychopathology could emerge. Moreover, a direct and extensive relationship between depression and anxiety traits and eating psychopathology was expected to be due to the changes in personality.

The aim of the present study was to perform an 8-year follow-up of a sample of patients affected by an ED who had been treated with an integrated model, with a specific focus on the changes of depression and anxiety. In particular it aimed: 1) to assess the outcome of depressive and anxiety symptoms at the 8-year follow-up using standardized measures (e.g. Symptom Checklist 90, and Beck Depression Inventory-II), and 2) to verify as to whether changes in general psychopathology could be related with those in personality traits on the TCI and in eating psychopathology (as measured by the Eating Disorder Inventory-2, EDI-2). The possible explicative model would consider personality as a possible predictor of the changes in general and eating psychopathology.

We expected to find a decrease in anxiety and depression scores at follow-up along with a reduction in eating symptoms and psychopathology severity. Even if the study design does not allow to make any inference on causal relationships, we hypothesized that eating, anxiety, and depressive features may influence each other, with personality as an underlying factor. The exploration of such relationships may generate insights in the long-term course of these aspects potentially helping clinicians plan individualized treatments.

Material and Methods

Participants

We considered for this study n=412 female outpatients seeking treatment at the Eating Disorder Centre of the University of Turin, between January, 1st 2003 and December, 31st 2005 (T0). Between January 2012 and January 2014 a clinical psychologist contacted over the phone all eligible individuals to inform them about the outcome study, and to verify their interest in study participation. All those interested in participating were then asked to be interviewed in person once (T1) by a member of the research team (psychiatrist or trained psychologist). Out of those who were eligible (n=412), only n=170 agreed with participating in our study; we failed to contact n=144

(35%) individuals, while n=98 (37%) declined study participation for personal reasons.

Inclusion criteria were defined as follows: 1) Receiving treatment at the Eating Disorder Centre of the University of Turin between January 1st, 2003 and December 31st, 2005; 2) Meeting AN or BN criteria according to the Structured Clinical Interview for Diagnostic and Statistical Manual of Mental Disorders, Revised Third Edition (SCID-I) [32] as assessed by a psychiatrist during the first interview (T0); 3) Absence of lifetime or current psychiatric diagnoses occurring before the ED onset (e.g., major atypical depression with overeating, or Obsessive Compulsive Disorder with eating rituals, somatoform disorder with gastric polarization); were instead included individuals with comorbid depression, anxiety or other disorders without any causal correlation with the eating disorder; 4) schizophrenia and bipolar disorders; 5) female gender; 6) absence of dropout over a 2year period of time; 7) good compliance to the delivered treatment interventions [33] monthly visits with a psychiatrist and registered dietitian, and two cycles (20 weekly sessions each) of Brief-Adlerian Psychodynamic Psychotherapy [34]; 8) no other treatments occurring after discharge.

Out of the recruited sample (n=170), n=107 (63%) individuals participated in this follow-up study whilst n=63 (37%) were excluded because of the aforementioned inclusion criteria. With more detail, n=25 dropped out from treatment, n=12 showed poor compliance to treatment; n=4 were affected by schizophrenia, and N=3 by bipolar disorder, n=3 were still undergoing treatment at time of follow-up, n=16 did not complete the assessments.

All participants with previous diagnosis of EDs (respectively n=60 with AN and n=47 with BN) were evaluated at T1 by a psychiatrist to assess the presence of an ED according to DSM IV-TR criteria as evaluated using the SCID-I [32]. Out of n=107 participants, n=61 met diagnostic criteria for an ED (n=31 AN, n=6 BN, and n=24 ED Not Otherwise Specified, EDNOS) and n=46 resulted to be recovered.

The multimodal treatment

All patients underwent a multimodal treatment for EDs, as described by our group [33] consisting of psychiatric clinical management (including medications), regular visits with a registered dietitian, and individual Brief Adlerian Psychodynamic Psychotherapy (B-APP) [34].

In particular, patients were visited by a psychiatrist and a psychiatry resident and then monthly followed-up. All residents were regularly supervised (case management and psychodynamic supervisions). During follow-up all medications (tranquillizers, antidepressants or antipsychotics) which were prescribed on the basis of comorbid anxiety or depressive psychopathology were titrated as much as needed per clinical decision in order to achieve their desirable effects.

The treatment team included also a registered dietitian and a physician trained in internal medicine with specific expertise in EDs who performed monthly and bi-monthly visits, respectively.

Finally, patients were treated with B-APP [34] which lasted 15-20 sessions depending on clinical severity. At the end of psychotherapy both psychiatrists re-assessed the patients and evaluated the following steps of the treatment plan. Per clinical judgment, the B-APP cycle could be repeated once after an observation period ranging from 3 to 9 months (mean 6 months) according to patients' needs. The therapists

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usually decided whether the second cycle could have the same focus of the previous one or a different one.

As an adjunctive intervention, all patients' parents or significant others (e.g. husbands or stable partners, grandmothers/fathers, uncles) were offered a counselling intervention. The counselling was addressed to both management of the eating behaviors and relatives' emotional and relational problems. Family-based therapy [35] was not specifically adopted since patients were adults and family autonomy was specifically encouraged [36].

The treatment period generally lasted from six months to three years depending on the eating and general psychopathology, personality structure and traits, treatment compliance, and response to treatment. Dropout from treatment ranged from 31% for those affected by AN to 34% for those with a diagnosis of BN, as reported in previous papers [37,38].

Ethics

All participants provided written informed consent. The research was reviewed and approved by the Ethics Committee of the hospital "AOU Città della Salute e della Scienza" of the University of Turin, Italy.

Materials

Participants' socio-demographic and clinical characteristics (e.g., age, diagnosis, BMI, binge/purging behaviors) were recorded at T0 (i.e., first visit at the ED Center) and T1 (face-to-face interview conducted 8 years after discharge).

All participants completed the following self-report questionnaires at both time-points:

Temperament and Character Inventory (TCI) [39], 240 items: it is an instrument used for the dimensional assessment of personality. It is divided into 7 independent dimensions, 4 of which assess temperament (novelty seeking, harm avoidance, reward dependence and persistence) the other 3 assess character (self-directedness, cooperativeness, and self-transcendence).

Eating Disorder Inventory-2 (EDI-2), 91 items [40]. A well-known instrument with 11 subscales measuring attitudes, behaviors, and eating traits common to individuals with EDs.

Symptom Checklist 90 (SCL-90), 90 items [41]. It assesses general psychopathology. It considers nine dimensions (somatization, obsessive-compulsive, interpersonal sensitivity, depression, anxiety, hostility, phobic anxiety, paranoid ideation, and psychoticism) and a total score that indicates global severity of psychopathology is provided.

Beck Depression Inventory (BDI-II), 21 items [42]. It is used to assess the severity of depressive symptoms. This tests investigates somatic aspects of depression like fatigue, asthenia, decrease of appetite, weight loss, sleep disturbances, sexual impairment, and psychological aspects like sadness, sense of failure, sense of guilt, self-incrimination. Cut-off for clinical attention is fixed at 16 [42].

We used the SCL-90 to assess anxiety since two scales (phobic anxiety and general anxiety) separately assess different forms of anxiety. However, we used also the BDI-II in addition to the SCL-90 aiming at performing a broader assessment of depression.

Statistical Analysis

All the analysis on the ED participants were first conducted comparing AN and BN patients and then separately for recovered versus non-recovered AN and BN individuals.

A GLM ANCOVA for repeated measures was performed to compare anxiety and depression scores, and eating psychopathology and personality scores between intake (T0) and 8-year follow-up (T1). The age of onset of the disorder, the age at the intake in the study and the age since the first visit (duration of the follow-up), and also the performed drug treatment (three categories: no drug, tranquillizers, antidepressants, antipsychotics) were considered as confounding variables comparing eating symptoms and psychopathology between T0 and T1.

Multivariate regression analysis was performed to ascertain the relationship of the changes in personality with anxiety and depression scores, and eating psychopathology. Moreover the delta values (e.g. the difference between the T0 and the T1 of the study) of depression and anxiety were correlated with a multiple regression analysis with those of eating symptoms and eating psychopathology. As a consequence of the high number of variables in the analysis a Bonferroni correction with p<.05/(22 variables)=p<.002 significance level was adopted for the GLM ANCOVAs to reduce Type I errors. Since multivariate regression statistically corrects the interaction between multiple variables considered in the analysis the value of p<.05 was considered acceptable for second level analysis. Statistical Analysis was conducted using SPSS 17th software package [43].

Results

Demographic and clinical characteristic of the sample

At T0 n=60 out of n=107 participants (44%) were diagnosed with AN (both AN-Restricting and AN-Binge-Purging subtypes; age=29.65 \pm 8.83 years), n=47 (56%) with (both BN-Purging and BN-Non-Purging subtypes; age= 29.81 \pm 9.86 years). See Table 1 for further details.

| Group Variable | | то | | T1 | | Paired sample t- test | |
|------------------------|----|--------------|------------|--------------|----|--------------------------|-------|
| | | Mean | SD | Mean | SD | t | Р |
| BMI | AN | 16.3 ± 1.18 | | 17.89 ± 2.03 | | -6.149 | 0 |
| | BN | 21.28 ± 3.04 | | 21.47 ± 3.69 | | -0.476 | 0.636 |
| B-P/week | AN | 3.21 ± 5.33 | | 0.63 ± 2.09 | | 4.396 | 0 |
| | BN | 5.43 ± 5.07 | | 0.47 ± 1.68 | | 6.511 | 0 |
| Age of onset | AN | 19.83 ± 6.8 | | n.a | | n.a | n.a |
| | BN | 18.61 ± | 5.61 | | | | |
| Duration of illness | AN | 9.78 ± 8.84 | | n.a | | n.a | n.a |
| | BN | 11.4 ± 9 | 11.4 ± 9.5 | | | | |

Table 1: Demographic and clinical characteristics and paired sample t-test of between T0 and T1

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Anxiety, depressive, eating psychopathology, and personality changes between T0 and T1 in AN and BN samples

Results of the GLM ANCOVA for repeated measures as regards BDI-II and SCL-90 anxiety and depression scores between T0 and T1 with respect to AN versus BN groups and recovered versus non-recovered subgroups are summarized in Tables 2 and 3.

| Group Variable | | то | | T1 | | | |
|----------------------------|-------|--------------|--------|---------------|--------------|--------|-------|
| | | Mea n | SD | Mean | SD | F | Р |
| BDI II | AN | 16.08 ± 5.87 | | 9.3 ± 7.72 | | 4.516 | 0 |
| | AN h | 13.88 ± 4.96 | | 8.52 ± 3.39 | | ns | ns |
| | AN nh | 17.66 | ± 6.02 | 9.86 ± 4.6 | | 7.708 | 0 |
| Depression (SCL90) | AN | 22.78 | ± 7.92 | 19.78 ± 10.16 | | 4.389 | 0 |
| | AN h | 20.92 | ± 6.87 | 14.75 ± 9.53 | | 6.657 | 0 |
| | AN nh | 24.09 | ± 8.44 | 23.44 ± | 9.09 | 6.208 | 0 |
| Anxiety (SCL90) | AN | 16.69 | ± 5.8 | 13.18 ± 7.25 | | 7.417 | 0 |
| | AN h | 14.75 | ± 5.84 | 11.03 ± | 6.54 | 12.401 | 0 |
| | AN nh | 18.1 ± | 5.42 | 15.36 ± | 7.28 | 15.003 | 0 |
| Phobic anxiety (SCL90) | AN | 5.57 ± 3.57 | | 4.92 ± 3.88 | | 7.639 | 0 |
| | AN h | 5.11 ± | 3.31 | 4.23 ± 4.16 | | ns | ns |
| | AN nh | 5.84 ± | 3.77 | 5.42 ± 3.67 | | | |
| Drive of thinness | AN | 10.12 | ± 8.27 | 6.82 ± 6.55 | | 3.782 | 0 |
| (EDI-2) | AN h | 8.68 ± | 7.83 | 4.4 ± 5.93 | | 5.338 | 0 |
| | AN nh | 11.14 | ± 8.53 | 8.54 ± 6 | 6.49 | ns | ns |
| Bulimia | AN | 4.17 ± | 5.3 | 2.57 ± 4.18 | | 3.471 | 0.001 |
| (EDI-2) | AN h | 2.68 ± | 4.14 | 1.6 ± 3. | 1.6 ± 3.73 | | 0 |
| | AN nh | 5.23 ± | 5.83 | 3.26 ± 4 | 1.31 | 5.707 | 0 |
| Interoceptive Awareness | AN | 10.5 ± | 7.33 | 5.68 ± 6 | 6.44 | 5.362 | 0 |
| (EDI-2) | AN h | 9.88 ± | 7.06 | 7.84 ± 6 | 6.87 | ns | ns |
| | AN nh | 12.11 | ± 6.35 | 10.09 ± | 10.09 ± 8.42 | | ns |
| Ineffectivenes s | AN | 9.88 ± | 7.99 | 8.06 ± 8 | 3.01 | ns | ns |
| (EDI-2) | AN h | 6.4 ± 6 | 6.34 | 5.72 ± 6.21 | | ns | ns |
| | AN nh | 12.37 | ± 8.2 | 9.74 ± 8 | 3.77 | 3.732 | 0 |
| Social Insecurity | AN | 7.83 ± | 4.88 | 5.95 ± 4.33 | | 3.348 | 0.001 |
| (EDI-2) | AN h | 6.16 ± 3.62 | | 4.52 ± 3.12 | | ns | ns |
| | AN nh | 9.03 ± | 5.35 | 6.97 ± 4 | 1.8 | ns | ns |

| Impulse Regulation | AN | 6.9 ± 6.24 | 4.02 ± 4.3 | 3.411 | 0 |
|------------------------|-------|--------------|--------------|--------|-------|
| (EDI-2) | AN h | 5.44 ± 5.96 | 2.48 ± 2.96 | ns | ns |
| | AN nh | 7.94 ± 6.31 | 5.11 ± 4.8 | 4.204 | 0 |
| Interpersonal distrust | AN | 6.51 ± 4.88 | 4.43 ± 4.21 | 3.85 | 0 |
| (EDI-2) | AN h | 5 ± 3.59 | 3. 72 ± 3.41 | ns | ns |
| | AN nh | ± 5.45 | 4.94 ± 4.68 | 3.807 | 0.001 |
| Harm avoidance | AN | 24.42 ± 7.12 | 21.57 ± 6.9 | 3.149 | 0.002 |
| (TCI) | AN h | 21.68 ± 7.65 | 19.6 ± 6.19 | ns | ns |
| | AN nh | 26.44 ± 6.06 | 22.77 ± 7.18 | ns | ns |
| Self directedness | AN | 21.27 ± 8.56 | 26.5 ± 8.12 | -5.386 | 0 |
| (TCI) | AN h | 25.64 ± 8.06 | 30.4 ± 6.71 | ns | ns |
| | AN nh | 18.5 ± 7.93 | 23.54 ± 7.84 | ns | ns |

Table 2: ANCOVA of clinical, psychopathological and personalityimprovement between T0 and T1 in AN patients

AN: Anorexia Nervosa; AN h: Anorexia Nervosa healed; AN nh: Anorexia Nervosa non healed.

| Group Variable | то | | T1 | | | | |
|---------------------------|----------|--------------|--------|--------------|-------|--------|------|
| | | | SD | Mean | SD | F | Р |
| BDI II | BN | 13.30 ± 4.62 | | 8.30 ± 3.28 | | 8.245 | .000 |
| | BN h | 13.86 ± | 4.75 | 8.17 ± 3.30 | | 8.259 | .000 |
| | BN nh | 11.45 ± 3.83 | | 8.73 ± 3.37 | | 1.087 | .000 |
| Depression (SCL90) | BN | 22.84 ± 6.32 | | 19.61 ± 6.63 | | 3.343 | .001 |
| | BN h | 23.21 ± | 6.42 | 19.96 ± 6.58 | | ns | ns |
| | BN nh | 21.64 ± 6.13 | | 18.44 ± 6.99 | | 6.634 | .001 |
| Anxiety (SCL90) | BN | 16.64 ± 4.71 | | 11.39 ± 4.40 | | 13.950 | .000 |
| | BN h | 16.66 ± | 5.01 | 11.28 ± | 14.56 | 6.495 | .000 |
| | BN nh | 16.48 ± | : 3.74 | 11.77 ± | 4.17 | 8.664 | .000 |
| Phobic anxiety (SCL90) | BN | 5.07 ± | 2.97 | 3.60 ± 2 | 50 | 8.409 | .000 |
| | BN h | 4.25 ± | 1.63 | 3.97 ± 2 | 61 | ns | ns |
| | BN nh | 5.07 ± | 2.97 | 2.49 ± 1 | .78 | 3.605 | .000 |
| Drive of thinness | BN | 14.17 ± | 5.96 | 8.00 ± 6 | 5.91 | 5.257 | .000 |
| (EDI-2) | BN h | 14.75 ± 5.98 | | 7.09 ± 6 | .30 | ns | ns |

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| | BN nh | 12.27 ± 5.55 | 10.73 ± 8.21 | ns | ns |
|----------------------------|----------|---------------|--------------|--------|------|
| Bulimia | BN | 9.93 ± 5.94 | 3.13 ± 3.99 | 7.341 | .000 |
| (EDI-2) | BN h | 9.53 ± 6.17 | 2.85 ± 3.71 | ns | ns |
| | BN nh | 11.45 ± 4. 68 | 4.00 ± 4.86 | ns | ns |
| Body dissatisfaction | BN | 14.43 ± 8.60 | 8.91 ± 5.59 | 4.749 | .000 |
| (EDI-2) | BN h | 15.28 ± 8.51 | 8.54 ± 6.39 | 5.184 | .000 |
| | BN nh | 12.54 ± 9.12 | 8.91 ± 5.59 | ns | ns |
| Interoceptive Awareness | BN | 10.82 ± 6.20 | 3.59 ± 4.43 | 6.706 | .000 |
| (EDI-2) | BN h | 11.28 ± 6.35 | 3.30 ± 4.60 | 3.467 | .000 |
| | BN nh | 9.36 ± 4.98 | 4.45 ± 3.96 | 4.186 | .000 |
| Ascetism | | 8.11 ± 4.18 | 4.02 ± 3.93 | 5.960 | .000 |
| (EDI-2) | | 8.53 ± 4.57 | 4.09 ± 4.01 | ns | ns |
| | | 6.73 ± 2.50 | 3.82 ± 3.84 | ns | ns |
| Ineffectiveness | BN | 9.90 ± 6.07 | 6.02 ± 5.35 | 3.934 | .000 |
| (EDI-2) | BN h | 10.08 ± 6.39 | 5.76 ± 5.22 | 4.139 | .000 |
| | BN nh | 9.18 ± 4.89 | 6.82 ± 5.91 | ns | ns |
| Social Insecurity | BN | 6.68 ± 4.30 | 4.36 ± 3.82 | 3.501 | .001 |
| (EDI-2) | BN h | 7.67 ± 4.32 | 4.54 ± 3.68 | ns | ns |
| | BN nh | 4.00 ± 2.37 | 3.81 ± 4.33 | ns | ns |
| Impulse Regulation | BN | 7.18 ± 5.59 | 3.79 ± 4.31 | 4.798 | .000 |
| (EDI-2) | BN h | 7.53 ± 6.02 | 4.06 ± 4.80 | ns | ns |
| | BN nh | 5.54 ± 3.24 | 3.00 ± 2.28 | ns | ns |
| Interpersonal distrust | BN | 5.16 ± 3.35 | 3.22 ± 3.85 | ns | ns |
| (EDI-2) | BN h | 5.56 ± 3.40 | 3.13 ± 3.84 | 3.888 | .000 |
| | BN nh | 4.36 ± 2.73 | 3.45 ± 4.05 | ns | ns |
| Self directedness | BN | 23.04 ± 8.63 | 27.51 ± 6.73 | -3.033 | .000 |
| (TCI) | BN h | 21.94 ± 9.18 | 27.18 ± 6.83 | -3.558 | .000 |
| | BN nh | 25.09 ± 5.43 | 28.00 ± 6.59 | -4.397 | .000 |

Table 3: ANCOVA of clinical, psychopathological and personalityimprovement between T0 and T1 in BN patients

BN: Bulimia Nervosa; BN h: Bulimia Nervosa healed; BN nh: Bulimia Nervosa non healed.

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Multivariate regression analysis in the AN group

As concerns personality, the changes in HA statistically predicted those in inadequacy (F= 11.264; p<.002), body dissatisfaction (F=8.829, p<.005), drive to thinness (F=5.345, p<.026), and asceticism (F=4.550, p<.039). The changes in SD predicted those in bulimia (F=11.198, p<.002), and impulsiveness (F=6.171, p<.017) whilst cooperativeness predicted the changes in interpersonal distrust (F=9.495, p<.004), and asceticism (F=6.532, p<.015).

With respect to the relationship between psychopathology traits, multivariate regression with anxiety and depression scores as statistical predictors of eating psychopathology showed that depression on the SCL-90 was significantly related to perfectionism (F=9.310; p<.004), while anxiety to weekly episodes of vomit (F=6.437, p<.015). If the analysis were conducted in the opposite direction the relationships persisted although they were weaker (F=5.809, p<.022; F=5.967, p<. 020, respectively).

Finally, depression on the SCL-90 significantly predicted phobic anxiety (F=7.621, p<.008), while in the opposite sense the relationship lost significance (F=4.147, p<.048).

Multivariate regression analysis in the BN group

As regards personality, SD predicted the changes in social insecurity (F=13.254, p<.001), interoceptive awareness (F=12.123, p<.001), interpersonal distrust (F=7.270, p<.009), asceticism (F=5.426, p<.024), impulsiveness (F=4.868, p<.032), and inadequacy (F=4.266, p<.044). Changes in cooperativeness statistically predicted those in depression (F=4.599, p<.036).

When assessing anxiety and depression scores as statistical predictors of eating psychopathology with multivariate regression analysis we found that depression on the SCL-90 was significantly related to perfectionism (F=4.851, p<.032), while anxiety to interpersonal distrust (F=5.391, p<.024), and social insecurity (F=4.558, p<.037), and phobic anxiety to weekly episodes of vomit (F=5.780, p<.020). If the analysis were conducted in the opposite direction the relationships between weekly episodes of vomit and phobic anxiety persisted but it was less significant (F=4.376, p<.046). A new significant association emerged with body dissatisfaction predicting changes on the BDI-II (F=4.694, p<.036).

As concerns the relationship between anxiety and depression, depression on the SCL-90 significantly predicted anxiety (F=47.019, p<.001), and phobic anxiety (F=41.137, p<.001), while in the opposite direction the relationship persisted, although losing some significance (F=19.112, p<.001 and F=11.076, p<.001, respectively).

Discussion

With this study we performed an 8-year follow-up that garnered encouraging results overall in line with earlier literature [44,45]. Depressive and anxiety symptoms significantly improved at time of follow-up in both AN and BN subgroups although medications [46-48] may have played a relevant role in this regard. Nevertheless, the majority of those who were assessed at follow-up were medication free.

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Moreover, in line with the body of evidence on the effectiveness of psychotherapy in the outcome of depression and anxiety [49,50] the delivered multimodal treatment addressed not only the ED but also self-esteem, resourcefulness, and relational functioning by means of the B-APP [34,51], eventually improving also some pathogenic features of both depression and anxiety. The possibility that the decrease of eating symptoms (which often causes depression or anxiety) or recovery may have positively influenced also depressive and anxious features is possible but it cannot be confirmed by multivariate regression analysis except for body dissatisfaction in bulimic women, potentially suggesting nonlinear mechanisms. Personality traits showed a long-term improvement characterized by reduction of HA, and increase of SD as well as cooperativeness as already found in other outcome studies [46]. In particular, HA and SD are the personality core features of EDs and other mental disorders as well, eventually representing risk factors for recurrence, partial remission or treatment resistance [31,47]. In particular, their improvement may have played a relevant role in the evolution of eating psychopathology.

Anorectic individuals

Notwithstanding the severity of AN and its high rates of recurrence and partial response to treatment [45,52-54], we found encouraging results as regards eating psychopathology which was significantly improved at follow-up. The comparison of AN recovered versus nonrecovered individuals highlighted some between-group differences in the improvement of anxiety and depression or eating psychopathology and personality traits. Nevertheless, in both ED groups the recovered group was more likely to report a less relevant improvement then the non-recovered one.

This finding could be explained by baseline measures; in fact, the recovered group scored lower at T0. Psychopathology may have had an effect on recovery in a threshold-related manner: sub-threshold changes in psychopathology could not help recovery. Similar findings in those affected by BN confirm the relevance of general and eating psychopathology traits in the course of EDs, independently from diagnostic subgroup.

All in all, our findings confirm that, even though medications in AN are, at-best, poorly effective [55-58] and psychotherapy requires specific focus also on psychopathology features which are not strictly ED-related [37,59], the multimodal treatment facilitated the improvement of eating psychopathology and symptoms, even though not achieving full recovery [57]. Since our sample included severely ill patients with AN (BMI<16, namely severe or extreme AN according to the current DSM-5 classification) these findings suggest that such a comprehensive approach represents on opportunity to survive and at least partially recover from AN, regardless of its severity [10].

Bulimic individuals

The treatment of patients with BN resulted more favorable than that of AN, substantially confirming existing data in literature [60]. In particular, as specific focus of the present research, we found that anxiety and depressive symptoms were significantly improved at follow-up. The changes in personality, particularly SD which is related to the overall character development, seem to play a heavier role in the BN group than in the AN group and this, along with the higher response to drug treatments [61], may explain the greater response to psychological treatments of those with BN [62,63]. These results provide support to the long-lasting response to treatment of BN individuals; relatedly, the multimodal treatment was found to be helpful in changing the course of the eating symptoms. Also in the BN group the non-recovered individuals sometimes reported larger improvements than the recovered ones, highlighting that, regardless of the complete symptom remission, the treatment was effective in reducing distress and psychopathology in all participants.

Relationship between improvement of anxiety and depression symptoms, personality, and eating psychopathology

The relationship between changes in HA and SD and also in cooperativeness with many eating psychopathology traits in both ED groups is consistent with previous literature supporting EDs to be entrenched with these personality features [31]. On the other hand, the weak correlation we found between personality changes and general psychopathology is somehow in contrast with the aforementioned hypothesis. However, the psychopathology process based on the liability of personality traits [31,47] seemed more directly related to eating psychopathology than to anxiety and depressive symptoms. Even though anxiety and depression levels decreased along with personality traits evolution, they were less linearly related to personality evolution than expected. In fact, anxiety and depression could fluctuate more (e.g., environmental stressors and medications) than eating psychopathology.

The relationship of anxiety and depression with eating psychopathology resulted to be less relevant than hypothesized. The direction of the relationship was statistically stronger from general to eating psychopathology, with the only exception of body dissatisfaction eventually producing a sort of relief on depressive feelings in BN women. This supports that anxiety and depression tend to evolve in a rather similar way during treatment, even though they represent a relevant complication to be treated to favor the recovery process. As concerns the relationship between anxiety and depression the relatedness of the SCL-90 scales suggests that the use of another instrument (such as the BDI-II) for the assessment of depression is worth of interest in the ED field.

Conclusion

The present study supports the effectiveness of the multimodal integrated treatment in the improvement of mood and anxiety features, eating symptoms and psychopathology [57,64-66]. It could be the result of the combination of both medications and psychodynamic psychotherapy which has been demonstrated to be effective in depression and anxiety disorders [67-69].

Changes in anxiety and depressive symptoms are accompanied by the improvement of those personality traits which are mostly related to general and eating psychopathology [1,31,47]. We hypothesized that such changes in personality may be partly responsible for the stabilization of the improvements in anxiety and depression. Nevertheless, we could not demonstrate a linear correlation between the improvement of depression and anxiety, personality, and eating psychopathology.

The substantial independence between the improvement in anxiety and depressive features with eating symptoms and psychopathology, and personality traits suggests that multimodal approach does not act hierarchically from personality to general psychopathology to eating

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symptoms but it could be rather underpinned by complex dynamics which future exploration may want to examine [34,57].

Limitations

The study was conducted on a relatively small sample of individuals with a lifetime history of AN and BN; a sample with mixed diagnoses hampers the exploration of diagnosis-specific dynamics. Moreover, not all patients who sought treatment at the ED Center of the University of Turin agreed with participating so a recruitment bias may have occurred. In particular, we could have included those who were satisfied with the delivered intervention or those who were still under treatment (e.g., individuals with EDNOS). Still, some recovered individuals may have declined to avoid psychological distress related to their history of illness. Finally, it was impossible to contact a substantial number of treatment-seekers whose outcome (including mortality rates) could not be taken into account.

Clinical implications and research perspectives

Our data are substantially in keeping with current literature and support that a multimodal treatment is at the moment the treatment of choice of EDs, almost independently from the applied form of psychotherapy [57,59,70]. Depressive and anxiety psychopathology improves along with the course of the ED, but the absence of both control group and intermediate time-points makes it impossible to exclude physiological symptom fluctuations and to verify the efficacy of the multimodal treatment also in reducing anxiety and depression in those with an ED. Nevertheless, the data of this 8-year follow-up study provide support to the overall reduction of the levels of anxiety and depression in both AN and BN individuals.

These results encourage further research on the relationship between different aspects of general and eating psychopathology with the personality traits of HA and SD [31,47]. In particular, the nonlinear mechanisms by which multimodal treatment could produce changes in different psychopathology domains should be explored [54, 59]. The changes in patients' functioning which influence changes in anxiety, depression or eating symptoms should be recognized and addressed with specific approaches [54,57]. The study of factors related to treatment-resistance may be extremely useful to improve the understanding of the underlying dynamics needing specific therapeutic care [1,7]. Regular clinical follow-up strategies to produce epidemiologically solid data are much needed.

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