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Original article

Insight and depressive symptoms in eating disorders: the mediating role of disorder-specific psychopathology

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ABSTRACT

Objective: To study whether the insight described in psychosis also occurs in eating disorders (EDs), whether it differs between anorexia nervosa (AN) and bulimia nervosa (BN), and whether diagnosis-specific ED psychopathology mediates this relationship.
Methods: Cross-sectional baseline data from a prospective cohort of 103 day-hospital patients with AN or BN were analyzed. Standardized measures of insight, depressive symptoms, and ED psychopathology were administered. Exploratory factor analysis was conducted on depressive symptoms, followed by diagnosis-stratified correlation analyses between insight dimensions and depressive symptom factors. Mediation models were then applied to examine whether diagnosis-specific ED psychopathology accounted for significant associations.
Results: Three depressive dimensions were identified: emotional, apathetic, and self-critical, explaining 56.26% of the variance. In AN, affective distress was associated with poorer insight into hypothetical contradiction and treatment engagement, whereas negative self-cognitions where associated with better recognition and relabeling of ED pathology. In BN, depressive symptoms were associated with reduced insight into body weight concerns and treatment engagement. ED–specific psychopathology significantly mediated the relationship between depressive symptoms and insight in both AN and BN.
Conclusion: These findings reveal that depressive symptoms and insight are associated in distinct patterns in anorexia and bulimia, and appear to be linked through different ED-specific psychopathological pathways. Highlighting these diagnosis-specific associations contributes to a more nuanced understanding of insight in EDs and underscores the need for tailored clinical approaches.

1. Introduction

The relationship between insight into illness and depressive symptoms has attracted particular attention in psychiatric research, as depression appears to be the only group of symptoms that shows a positive association with clinical insight - that is, better insight is related to higher levels of depression. This unique association distinguishes depression from other psychiatric symptoms, which often exhibit an opposite pattern. The term "insight paradox" has been used to describe the co-occurrence of depressive symptoms among patients who demonstrate good levels of insight (Belvederi Murri et al., 2015; Lysaker et al., 2007). Explanations for this paradox typically point in two opposite directions. On one hand, it has been suggested that awareness of having a severe psychiatric disorder - often with significant social, functional, and existential consequences - may induce or exacerbate depression. On the other hand, low mood or depressive states may themselves promote a more realistic or pessimistic view of the self and the world, leading to increased awareness of illness and its implications (David, 2018).

The relationship between clinical insight and depressive symptoms has been extensively studied in psychotic disorders, particularly schizophrenia, where greater insight - especially awareness of illness and its implications - is consistently associated with higher levels of depression, hopelessness, and suicidality, although meta-analyses indicate that the overall effect size is modest (Belvederi Murri et al., 2015; Misdrahi et al., 2014; Murri et al., 2016). This paradoxical association

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has been linked to psychological mechanisms such as internalized stigma, hopelessness, and illness engulfment - where one's identity becomes overtaken by the illness (Konsztowicz & Lepage, 2019; Lysaker et al., 2013) and may be further shaped by moderating factors, such as clinical severity, socioeconomic status, and engagement with mental health services (Murri et al., 2016). Additionally, emerging unpublished data from psychosis research suggests that the direction and strength of this relationship may differ across depressive symptom domains, with cognitive and somatic symptoms potentially showing divergent associations with insight and contributing to the heterogeneity of findings in the literature.

In psychosis, insight is conceptualized as a multidimensional construct encompassing recognition of having a mental illness, the ability to re-label pathological experiences, attribution of symptoms to illness, awareness of social consequences, and commitment to treatment (David, 1990). In eating disorders (EDs), insight is often shaped by ego-syntonic symptoms, identity investment in the disorder, and internalized weight- and shape-related beliefs, which may at times reach delusion-like intensity but typically occur in the absence of broader psychotic symptoms. These similarities and differences provide a rationale for examining whether the insight paradox described in psychosis also applies to EDs.

In the context of EDs, the literature presents contradictory findings regarding the relationship between insight and depression. Several studies have reported no association between these variables in anorexia nervosa (AN) (Arbel et al., 2013, 2014) or bulimia nervosa (BN) (Konstantakopoulos et al., 2011, 2020). Other studies have found a negative association between depression and insight (Gawron et al., 2025), whereas a positive association has also been described (Sciarrillo et al., 2024). Notably, the only prospective study in AN, found that reductions in depressive symptoms during treatment were accompanied by improvements in insight (Gorwood et al., 2019), highlighting a potential causal link that supports investigating these associations further.

In individuals with EDs, depressive symptoms are common but may stem from heterogeneous and disorder-specific mechanisms (Leblé et al., 2017). For example, during recovery, patients may experience a loss of self-esteem and identity as they abandon behaviors that were experienced as ego-syntonic (Gregertsen et al., 2017), which would lead to depressive symptoms (Mischoulon et al., 2011).

Furthermore, malnutrition itself can produce mood and cognitive disturbances, including anxiety and depression-like symptoms such as insomnia, irritability, poor concentration, social isolation, and apathy (Brozek J., 1990; Stanga et al., 2007). These diverse etiologies suggest that depressive symptoms in EDs may differentially influence insight-related processes. While emotional distress might heighten illness awareness, somatic or neurovegetative symptoms may hinder self-reflection or treatment engagement. Recent clinical data also suggest that guilt may play a central role in ED psychopathology, interacting with self-critical cognitions and potentially influencing insight (Raffone et al., 2025). Consequently, disentangling these symptom dimensions is crucial for understanding how depression shapes clinical insight in EDs.

Taken together, this clinical and etiological heterogeneity suggests that depressive symptoms in EDs cannot be treated as a unitary construct. This provided the rationale for adopting a data-driven approach to depressive symptoms, using exploratory factor analysis (EFA) to identify distinct symptom dimensions, and for examining diagnosis-specific pathways between depression and insight.

Given the clinical relevance of insight for treatment engagement and prognosis in EDs and the inconsistent findings in previous research, this study aimed to clarify how different depressive symptoms relate to specific components of clinical insight in EDs. We hypothesized that distinct symptom clusters may show divergent associations with insight, reflecting the heterogeneous nature of depressive experiences in this population. Specifically, we expected that affective symptoms of depression, such as sadness or crying might relate to greater awareness of illness, whereas somatic symptoms (e.g., fatigue, sleep disturbance, loss of energy) could be linked to lower levels of insight; however, exploratory analyses also examined cognitive self-critical symptoms which could show distinct patterns.

2. Method

2.1. Participants

The sample consisted of day-hospital patients from the EDs Unit of the Psychiatry Department of the Hospital de la Santa Creu i Sant Pau (HSCSP) and the Hospital Mutua de Terrassa (HUMT), (Catalonia, Spain). All consecutive admissions among those seeking voluntary admission between October 2022 and June 2024 were invited to participate. Inclusion criteria were: a) confirmed diagnosis of AN, BN or atypical AN classified under Other Specified Feeding or Eating Disorder (OSFED), included to avoid excluding patients with AN psychopathology who present with body weight within the normal range; according to the Diagnostic and Statistical Manual of Mental Disorders, 5th edition, Text Revision (DSM-5-tr), b) a minimum age of 17 years, and c) ability to speak Spanish or Catalan with adequate fluency. The participants had moderate to severe ED psychopathology, typically presenting with a long-standing illness. Relevant measures of ED psychopathology (EAT-40 and EDI-BN scores) are reported in Table 1.

2.2. Procedure

The study was approved by the Research Ethics Committee (CEIm) of HSCSP (approval number IIBSP-INS-2021-122) and the study was registered at https://clinicaltrials.gov/ (NCT06177262). All participants gave informed consent. We collected the data during the baseline phase of a prospective study examining the characteristics and course of illness awareness in EDs. As part of the data collection, participants completed self-administered questionnaires. Diagnosis was established during the initial clinical assessment by a clinical psychologist or psychiatrist specializing in EDs, following the unit's standard diagnostic protocol. Height and weight were measured during the consultation.

2.3. Measures

Eating Attitudes Test (EAT-40)

The EAT is a widely used standardized self-report questionnaire designed to assess ED symptoms, particularly those related to AN (Garner & Garfinkel, 1979). The test consists of 40 items that evaluate attitudes, thoughts, and behaviors associated with restriction, body image, and food preoccupation, using a 6-point Likert scale, where higher scores indicate a greater risk of ED. The Spanish version of the EAT-40 presents adequate psychometric properties (Castro J et al., 1991).

Eating Disorder Inventory - (EDI)

The EDI is a self-administrated questionnaire designed to assess ED specific symptoms. It comprises eight subscales, three of which evaluate attitudes and behaviors related to eating (Drive for Thinness (DT), Bulimia (B), and Body Dissatisfaction (BD)) while the remaining five assess broader psychological constructs related to EDs. For the present study, we used the B subscale. A previous study has confirmed the reliability and validity of the EDI subscales (Espelage et al., 2003). The Spanish validation demonstrated excellent internal consistency, with a Cronbach's α of 0.92 (Guimerà & Torrubia, 1987).

Beck Depression Inventory-II (BDI-II)

Depression was measured using the Spanish adaptation of the BDI-II (Sanz et al., 2003). The inventory was designed to evaluate depressive symptomatology on a 21 items scale ranged from 0 to 3. The Spanish version of the BDI-II presents adequate psychometric properties similar to the original (Beck et al., 1996) with a Cronbach's α of 0.87.

Schedule for the Assessment of Insight in Eating Disorders (SAI-ED)

Table 1
Clinical and sociodemographic characteristics of the total sample (N=103).

Characteristics	
Age (M; SD)	30.18 (13.21)
Self-identified gender (*n*; %)	
Women	102 (99.03)
Men	0 (0)
Non binary /other gender identities	1 (.97)
Self-reported sexual orientation (*n*; %)	
Heterosexual	82 (79.60)
Homosexual	1 (1.00)
Bisexual	14 (13.60)
Other orientations	2 (1.90)
Preferred not to say	4 (3.90)
Educational status (*n*; %)	
No formal education/ incomplete primary	1 (1.00)
Primary	18 (17.50)
Secondary	55 (53.40)
University	29 (28.20)
Civil status (*n*; %)	
Single	78 (75.70)
Married/living with partner	17 (16.50)
Divorced/separated	7 (6.80)
Widowed	1 (1.00)
Employment status (*n*; %)	
Employed	16 (15.50)
Student	40 (38.80)
Sick leave	28 (27.20)
Unemployed	2 (1.90)
Retired	2 (1.90)
On disability pension	7 (6.80)
Other	8 (7.80)
BMI (kg/m2) (M; SD)	
Current	20.88 (6.04)
Duration of illness (years) *(M; SD)*	11.43 (12.77)
ED diagnosis (*n*; %)	
AN	48 (46.60)
Atypical anorexia	5 (4.85)
BN	50 (48.54)
Any psychotropic medication *(n; %)*	68 (66.00)
Antidepressants	58 (56.30)
Antipsychotics	12 (11.70)
Anxiolytics/Hypnotics	27 (26.20)
Mood stabilizers (1)	18 (17.50)
EAT (M; SD)	55.37 (23.59)
EDI-BN (M; SD)	18.17 (7.75)
BDI-II depression severity (n; %)	
Minimal or no depression (0-13)	12 (11.70)
Mild (14-18)	5 (4.9)
Moderate (19-27)	22 (21.4)
Severe (28-63)	64 (62.1)

Abbreviations: M; SD (mean; standard deviation); BMI (body mass index); AN (anorexia nervosa); BN (bulimia nervosa); EAT-40 (Eating Attitudes Test); EDI-BN (Eating Disorder Inventory - Bulimia scale); BDI-II (Beck depression inventory).
Note: The "Atypical Anorexia" subgroup includes patients with a BMI in the normal range (18.5–24) admitted to the day hospital. This category was included under OSFED in accordance with the study's inclusion criteria for atypical anorexia presentations. Mood stabilizers were defined as lithium, valproate, carbamazepine, lamotrigine, and certain anticonvulsants (e.g. topiramate, gabapentin) when used in a psychiatric context.

The SAI-ED is a semi-structured interview consisting of eight items that assess the level of insight in EDs patients. The first two questions assess the awareness of illness and the need for treatment, with responses rated on a 0-2 scale; four questions evaluate the recognition of ED symptoms and their reassignment as related to the illness reinterpretation as symptoms of the illness; another item explores the hypothetical conflict between the individual's perception of their condition and others' concerns, all rated on a 0-4 scale. Additionally, there is a final question focused on adherence to treatment, collecting therapist impressions, and is rated on a 0-5 scale. All items except the treatment engagement item are clinician-administered based on patient responses, while the treatment engagement item is rated directly by the clinician. The SAI-ED generates two global scores: a subtotal score for awareness of illness and symptoms, derived from the first seven items (ranging from 0 to 24), and a total score that includes all items, covering the adherence item as well (ranging from 0 to 29).

The SAI-ED demonstrated good psychometric properties in a validation study, with the best cutoff points indicating lack of insight (score < 17.5; sensitivity of 100% and specificity of 90%) and for impaired insight (score < 23.5; sensitivity of 83% and specificity of 70%) (Konstantakopoulos et al., 2020). The Spanish version was validated with a Cronbach's alpha of $\alpha = 0.79$ (Gawron et al., 2025).

2.4. Statistical analysis

An exploratory factor analysis (EFA) was conducted on the 21 items of the BDI-II to identify latent structures underlying depressive symptoms in our sample. Preliminary analyses were performed to examine potential differences in BDI-II scores by diagnostic group (AN and BN; for analytical purposes, patients with atypical AN (OSFED) were included in the AN group). Although BDI-II total scores did not differ significantly between AN and BN (Table S2, Suppl. material), potential differences in factor structure across diagnostic subgroups were not examined and are acknowledged as a limitation. Internal consistency for each of the derived factors was assessed using Cronbach's alpha, yielding values of 0.90, 0.80, and 0.88 for Factors 1, 2, and 3, respectively, supporting adequate reliability. Based on these results, the EFA was conducted using the full sample. Prior to extraction, sampling adequacy was to be assessed using the Kaiser–Meyer–Olkin (KMO) measure and Bartlett's test of sphericity. The factor extraction method used was Principal Axis Factoring with oblique rotation (Oblimin), given the expected intercorrelation between factors. The number of factors was not predefined; instead, components with eigenvalues >1 were retained. Items were assigned to factors if they exhibited loadings >0.4 and a loading difference of at least 0.1 between factors. After factor extraction, factor scores were computed for each participant.

Correlation analyses were then performed to explore associations between depressive symptoms (total and factors) and total and individual items score of insight, using both Spearman's rho and Pearson's correlation coefficients, depending on the nature or distribution of the data. Additionally, correlations between depression scores and BMI were also assessed.

Post-hoc mediation analyses were conducted to explore whether AN and BN eating pathology mediated the relationship between depressive symptoms and insight. Mediation models were restricted to variable combinations that showed significant bivariate correlations within diagnostic groups, in order to reduce the risk of type I error and to focus on theoretically meaningful associations.

A significance threshold of $p < 0.05$ was applied. All analyses were conducted using IBM SPSS Statistics version 26.

3. Results

3.1. Sample characteristics

The final sample comprised 103 participants diagnosed with an ED, recruited from a specialized treatment program. The majority identified as women, and the sample showed a broad range of ages, educational levels, and employment statuses. Clinical and demographic characteristics, including diagnostic subtypes, medication use, and self-reported psychopathology, are presented in Table 1.

3.2. EFA

Preliminary analyses indicated no significant differences in total BDI-II scores between ED diagnosis ($t(101) = -1.01$, $p = 0.32$). Based on this, the full sample was used to conduct an EFA with oblimin rotation of the 21 BDI-II items. Sampling adequacy was excellent (KMO = 0.926), and

Bartlett's test of sphericity was significant ($\chi^2 = 1409.81$, df = 210, $p < 0.001$), supporting the suitability of the data for factor analysis. Three components with eigenvalues greater than 1 were extracted, accounting for 56.26% of the total variance. Items were assigned to a factor if they exhibited a loading ≥ 0.40 and a difference of at least 0.10 from loadings on the other factors. Items that did not meet these criteria - specifically items 2 (pessimism), 11 (agitation), 13 (indecisiveness) and, 16 (changes in sleep) - were not retained in any of the final components (See Table 2).

Based on the item content, the components were labeled as follows: Factor 1 - Affective distress: reflects the emotional and interpersonal aspects of depression, characterized by intense subjective suffering and emotional reactivity. This factor encompasses core affective symptoms often seen in acute depressive episodes and captures both internal emotional suffering and externally observable distress. Factor 2 - Hypoactivation/Apathy captures a marked reduction in energy, initiative, and motivational drive. The items loading on this factor reflect a state of disengagement and diminished activation rather than somatic or vegetative symptoms per se, suggesting a more apathetic or blunted presentation of depressive states.

Factor 3 - Negative self-cognitions reflects a cognitive dimension of depression characterized by pervasive self-critical thoughts, negative self-evaluation, and low self-worth. This component aligns with Beck's cognitive triad and captures the internalization of failure and self-blame.

3.3. Correlations

BMI was significantly correlated with Total BDI-II (r=0.28; p=0.01) and with the three factors (F1 (r=0.24; p=0.02), F2 (r=0.27, p=0.01 and F3 (r= .27; p=0.01)) in the total sample. However, no significant differences were found between AN group and BN group in total BDI-II scores (Table S2, Suppl. material).

Regarding insight, significant differences were found between diagnostic groups on several items and total scale (Table S1, suppl. material). Therefore, correlations between depressive symptoms and insight were analyzed separately by diagnostic groups using Pearson or Sperman coeficients. In patients with AN (Table 3), item 5 (Hypothetical contradiction) showed a moderate negative correlation with depressive symptoms (r = –0.42, p < 0.01), and item 4b (Relabeling of eating pathology) showed a moderate positive correlation (r = 0.35, p < 0.05).

In patients with BN (Table 4), item 3b (Relabeling of body weight concerns) showed a moderate negative correlation with depressive symptoms (r = –0.38, p < 0.05). Overall, fewer significant associations were observed in BN than in AN.

Table 2
Factors loadings for EFA with oblimin rotation of BDI-II items.

BDI-II items	F1 Affective distress	F2 Hypoactivation/ Apathy	F3 Negative self-cognitions
1. Sadness	**0.62**	0.21	0.03
2. Pessimism	0.32	0.19	0.41
3. Past failure	0.30	0.13	**0.48**
4. Loss of pleasure	**0.73**	0.18	-0.05
5. Guilty feelings	**0.64**	-0.16	0.30
6. Punishment feelings	**0.82**	-0.07	-0.06
7. Self-dislike	-0.15	0.19	**0.89**
8. Self-criticalness	0.25	-0.06	**0.61**
9. Suicidal thoughts	**0.57**	-0.03	0.19
10. Crying	**0.46**	0.07	0.14
11. Agitation	0.33	-0.05	0.29
12. Loss of interest	**0.66**	0.37	-0.11
13. Indecisiveness	0.32	0.28	0.27
14. Worthlessness	0.25	0.06	**0.60**
15. Loss of energy	0.19	**0.71**	-0.02
16. Changes in sleep	0.26	0.23	0.13
17. Irritability	**0.69**	0.10	0.01
18. Changes in appetite	**0.47**	0.18	0.06
19. Concentration difficulty	0.30	**0.41**	0.24
20. Tiredness or fatigue	-0.02	**0.87**	0.04
21. Loss of interest in sex	0.01	**0.43**	0.07

* Note: Factor loadings > 0.40 and a loading difference of at least 0.1 between factors are in boldface.

3.4. Mediation analyses

Significant mediation effects were found in both diagnostic groups. In the AN group, negative self-cognitions (F3) was associated with lower awareness of the disorder (item 5, Hypothetical contradiction) through increased restrictive eating symptoms - fear of gaining weight, dieting behaviors, and food-related preoccupations- as measured by the EAT-40. The indirect effect was statistically significant and of large magnitude (B = –0.40, 95% CI [–0.71, –0.13]), consistent with the hypothesis that cognitive self-distortions related to depression may be linked to lower insight via the intensification of restrictive ED pathology (Fig.1).

In the BN group, both affective (F1) and cognitive (F3) depressive symptoms were associated with reduced treatment engagement (item 6) through greater severity of bulimic symptoms, measured by the EDI-BN. The indirect effects were also significant (F1: B = –0.29, 95% CI [–0.62, –0.00]; F3: B = –0.28, 95% CI [–0.53, –0.06]), consistent with the hypothesis that different facets of depression may be associated with reduced motivation for treatment by exacerbating the emotional, cognitive and behavioral components of BN. (See Fig.2, Fig.3 and Table S3- Suppl. material).

4. Discussion

In our study, exploratory factor analysis (EFA) identified a three-factor structure of the BDI-II - Affective Distress, Hypoactivation/ Apathy, and Negative Self-Cognitions. These dimensions showed distinct associations with specific aspects of clinical insight, which varied across diagnostic groups (AN and BN). Mediation analyses further revealed that these associations were partially explained by diagnosis-

Table 3
Correlations of the schedule for the assessment of insight in eating disorders (SAI-ED) items with depressive symptoms in patients with Anorexia Nervosa.

SAI-ED items	BDI-II Total score	F1 Affective distress	F2 Hypoactivation/ Apathy	F3 Negative self-cognitions
1. Recognition of mental illness	0.01	-0.07	0.06	0.15
2. Awareness of need for treatment	-0.14	-0.25	-0.06	-0.02
3a. Recognitions of body weight concerns	-0.08	-0.09	-0.06	0.06
3b. Relabeling of body weight concerns	-0.05	-0.05	-0.06	0.10
4a. Recognitions of eating pathology	0.24	0.21	0.09	**0.28***
4b. Relabeling of eating pathology	0.19	0.17	-0.02	**0.29***
5. Hypothetical contradiction	**-0.51****	**-0.55****	-0.26	**-0.44****
6. Treatment engagement	-0.26	**-0.32***	-0.07	-0.22
Subtotal score	-0.03	-0.10	-0.06	0.14
Total score	-0.09	-0.16	-0.08	0.07

Note: Correlations are reported using Spearman's Rho and Pearson's *r*.
Significance (2-tailed): *$p < 0.05$; **$p < 0.01$

Table 4
Correlations of the schedule for the assessment of insight in eating disorders (SAI-ED) items with depressive symptoms in patients with Bulimia Nervosa.

SAI-ED items	BDI-II Total score	F1 Affective distress	F2 Hypoactivation/ Apathy	F3 Negative self-cognitions
1. Recognition of mental illness	0.01	-0.02	-0.01	-0.01
2. Awareness of need for treatment	-0.02	-0.07	-0.06	0.05
3a. Recognitions of body weight concerns	0.20	0.22	0.04	0.28
3b. Relabeling of body weight concerns	**-0.30***	**-0.31***	**-0.36***	-0.18
4a. Recognitions of eating pathology	0.23	0.23	0.07	0.23
4b. Relabeling of eating pathology	-0.13	-0.14	-0.23	-0.02
5. Hypothetical contradiction	-0.13	-0.15	-0.23	-0.06
6. Treatment engagement	**-0.40****	**-0.42****	-0.24	**-0.37***
Subtotal score	-0.14	-0.14	-0.23	-0.02
Total score	-0.23	-0.24	-0.27	-0.12

Note: Correlations are reported using Spearman's Rho and Pearson's *r*.
Significance (2-tailed): $*p < 0.05$; $**p < 0.01$

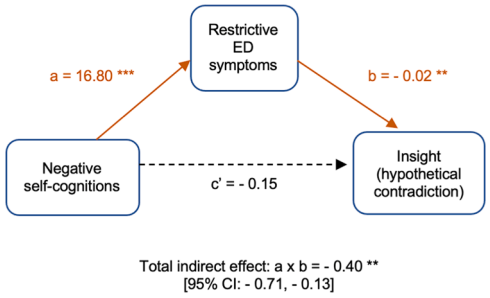

Fig. 1. Mediation model of the association between cognitive depressive symptoms and insight in AN, with restrictive eating pathology as mediator.

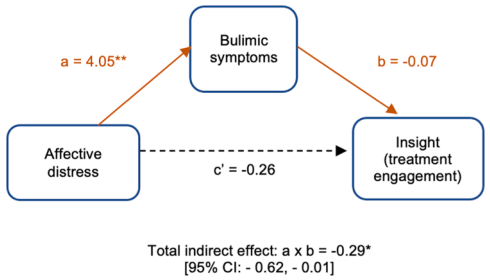

Fig. 2. Mediation model of the association between pathology as mediator.

specific ED symptomatology, partially explained by diagnosis-specific ED symptomatology, consistent with the hypothesis that depressive symptoms may be associated with lower insight through different ED-specific pathways depending on the clinical profile.

Several studies have evaluated the factor structure of the BDI-II in different populations, identifying some or all of the factors labeled Cognitive, Somatic, and Affective or its combinations in in two-factor models (Brouwer et al., 2013). This broadly aligns with our findings, where the Negative Self-Cognitions factor corresponds to Beck's cognitive triad, reflecting internalized failure and self-blame, emphasizing the

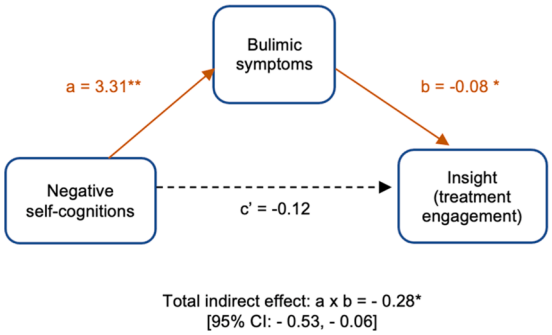

Fig. 3. Mediation model of the association between cognitive depressive symptoms and insight in BN, with bulimic pathology as mediator.

"self" aspect of cognitive symptoms. The second factor resembles the somatic-vegetative factor previously identified but seems to capture diminished activation without including other somatic aspects such as irritability or sleep disturbances.

Although the BDI is commonly used as a self-report measure of depression in EDs, its factorial structure in this population has been scarcely explored. One study with 437 patients with AN (Fuss et al., 2015) found the emergence of a factor related to agitation and irritability (Restlessness factor). However, their results suggest that, despite overlap between somatic symptoms of depression and the physical effects of malnutrition and starvation, the variance explained by the somatic symptoms of the BDI-II was similar to that found in non-ED samples. This supports the adequacy of the identified structure for the aims of our study.

BMI showed positive correlation with depressive symptoms with the total BDI score and when BDI were analyzed by factors. All correlations were similar in magnitude, statistically significant, and followed the same direction. Although there is some evidence suggesting associations between malnutrition and depressive symptoms in ED (Mattar et al., 2011; Meehan et al., 2006; Pleplé et al., 2021), may be more apparent in individuals with more severe AN (Stroe-Kunold et al., 2016), and BDI scores could remain elevated following weight restoration (Meehan et al., 2006; Pollice et al., 1997). Interestingly, in our study, no significant differences were found on BDI or its factors between AN and BN groups, and the correlations indicated that higher BMI was related to greater depressive symptomatology in the total sample. This pattern may suggest that higher body weight is associated with increased emotional distress in patients with EDs in day-hospital treatment, whereas individuals with lower BMI might experience difficulties in identifying or reporting depressive symptoms.

However, some of the insight aspects that differed between AN and BN groups were not consistently associated with depressive symptom scores in either group. This suggests that the observed variations are not solely explained by mean differences in depressive symptoms or insight, but may reflect distinct interaction profiles between depressive symptom dimensions and insight across diagnoses.

The recognition and attribution of disordered eating behaviors in individuals with AN was positively associated with negative self-cognitions. This was the only aspect of insight that showed a significant positive association with increasing depressive symptoms. This finding suggests a distinction between cognitive recognition of ED behaviors and emotional acceptance or motivation for change. Some patients may recognize their ED behaviors but, they may be integrated into a self-critical framework. In such cases, greater behavioral insight may be accompanied by self-directed hostility or guilt, and may serve to reinforce control, identity, or self-worth rather than readiness for change. Thus, greater recognition of symptoms does not necessarily imply readiness for treatment, but may instead reflect internalized critical standards characteristic of AN (Bäck et al., 2025).

Moreover, the *hypothetical contradiction* item, which reflects the

patient's ability to consider another person's perspective, showed a distinctive pattern in individuals with AN, but not in those with BN. Specifically, higher levels of depressive symptoms were associated with poorer awareness of illness in AN. The capacity to adopt others' perspectives is a metacognitive function closely linked to theory of mind (ToM), an area in which individuals with AN have demonstrated greater deficits compared to other EDs subtypes (Bora & Köse, 2016; Mason et al., 2021) although ToM was not directly assessed in the present study and this interpretation should therefore be considered hypothetical.

In the BN group, a negative association emerged between relabeling of body weight concerns and depressive symptoms as affective distress and hypoactivation/apathy. The ability to recognize body and weight concerns was previously identify as a con of the illness by BN patients and, a contribution aspect to enhance motivation to change (Serpell & Treasure, 2002). Our finding suggests that, higher levels of depressive symptoms are associated with greater prominence of alternative attributions for these concerns. For example, patients may increasingly explain their ED symptoms by referring to societal beauty ideals or internalized thinness norms, rather than recognizing them as illness-driven (Thompson & Stice, 2001). This could reflect a reduced capacity to "blame" the ED for their preoccupations, or even a lack of clarity about the origin of such concerns, although this interpretation remains speculative and warrants further empirical examination.

These diagnostic differences may suggest that depressive symptomatology interacts with qualitatively distinct cognitive processes underpinning insight. In BN, depressive symptoms may be associated with alterations in more basic cognitive-emotional processes such as emotional salience or self-referential processing, affecting the reattribution of body-related concerns. In contrast, in AN, depressive symptoms appear to be linked to higher-order cognitive functions such as metacognition and theory of mind, influencing the ability to adopt alternative perspectives and recognize contradictions.

Mediation analyses further supported this interpretation. In the AN group, cognitive depressive symptoms were associated with lower illness awareness through the intensification of restrictive eating pathology. In BN, both affective and cognitive depressive symptoms were associated with reduced treatment engagement via increased bulimic symptomatology. Notably, this mediator reflects not only behavioral expressions (e.g., bingeing or purging), but also the emotional and cognitive dysregulation surrounding these behaviors. These findings suggest that depressive symptoms are associated with lower clinical insight and motivation through disorder-specific mechanisms - amplifying rigid, self-punitive behaviors in AN, and emotional dysregulation in BN. Understanding these distinct pathways may guide more targeted therapeutic strategies, emphasizing the need for depression-informed, diagnosis-specific interventions in the treatment of ED.

From a clinical perspective, targeting negative self-cognitions in AN may help improve insight and could also contribute to reducing depressive symptoms, although this potential therapeutic effect should be confirmed in future longitudinal studies.

A potential methodological limitation of this study is the use of a self-report measure (BDI-II) to assess depressive symptoms, which may be influenced by the patient's ability to recognize them and report their severity. While clinician-administered interviews may offer greater objectivity, the BDI-II has shown more consistent performance in female populations compared to other self-reports, such as the CES-D or HADS which have demonstrated gender-related bias (Macêdo et al., 2018). Given that our sample was exclusively female and recruited from specialized hospital programs in Catalonia, the BDI-II may be a suitable choice. Future research should consider complementary assessment methods and the potential influence of comorbid mental disorders, which were not systematically controlled.

The cross-sectional design of this study precludes causal inferences regarding the temporal relationships between depressive symptoms, eating disorder psychopathology, and insight. Moreover, several mechanisms discussed (e.g., ToM) were not directly measured.

Although the sample size could be considered modest for conducting factor analysis, the data met established criteria for sampling adequacy and factorability. Moreover, prior research has shown that when communalities are moderate and factors are well determined, acceptable recovery of factor structures can be achieved with sample sizes in the range of 100–200 (Maccallum et al., 1999; MacCallum et al., 2001).

In addition, the sample size may have limited the power to detect interaction effects in the mediation analyses, particularly those with small effect sizes. Replication in larger samples is warranted to clarify the potential role of diagnostic status in moderating the relationship between depressive symptoms and insight.

Finally, the use of psychotropic medication by a notable proportion of participants may have influenced depressive symptoms and insight, representing a potential confounding factor. Additionally, the generalizability of our findings may be limited to females, hospital-based samples, or individuals with more severe forms of the disorder.

5. Conclusions

Our findings reveal diagnosis-specific patterns in how depressive symptoms relate to clinical insight in eating disorders. Depressive symptoms appear to be associated with lower insight through distinct mechanisms in anorexia and bulimia, mediated by disorder-specific symptomatology. These results underscore the importance of tailoring interventions to both the affective profile and diagnostic subtype of ED patients.

Public Significance Statement

Depression can change how people with eating disorders recognize and understand their illness. This study found that in anorexia and bulimia, depression influences insight in different ways, depending on the specific symptoms of each disorder. These results show why treatments should be tailored to both the diagnosis and the emotional profile of each patient, helping to improve engagement and recovery outcomes.

Declaration of generative AI and AI-assisted technologies in the manuscript preparation process

During the preparation of this work the authors used ChatGPT (OpenAI) in order to improve the clarity and readability of the manuscript text. After using this tool, the authors reviewed and edited the content as needed and takes full responsibility for the content of the published article.

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Ethical standards

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration to 2025, as revised in 2025.

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CRediT authorship contribution statement

Leonor P. Gawron: Conceptualization, Data curation, Formal analysis, Methodology, Writing – original draft. **Maria J. Portella:** Formal analysis, Resources, Supervision, Writing – review & editing. **Esther Pousa:** Writing – review & editing.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.psychres.2026.116965.

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