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Fear of Food in Eating Disorders: Its Role in Eating Disorder Symptomatology, Psychopathology, and Early Outpatient Treatment

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Anxiety is known to be correlated with eating disorder (ED) symptoms, but less is known about anxiety in terms of fear of food in EDs. The study aims to compare different ED groups, investigate early changes in fear of food following cognitive-behavioral-based treatment (CBT) with integrated nutritional rehabilitation and counseling and test whether changes in fear of food are associated with changes in eating symptomatology and general psychopathology. ED outpatients ($n = 282$) and controls ($n = 68$) were assessed with the Fear of Food Measure (FOFM), Eating Attitude Test-40 (EAT-40), and Eating Disorder

Inventory-3 (EDI-3 risk and General Psychological Maladjustment, GPM). MANOVA with post hoc comparisons was run to compare controls and ED subgroups in FOFM. Paired sample *t*-test evaluated FOFM changes between baseline and mid-treatment. Multiple regression analyses were conducted to test whether changes in FOFM were associated with changes in EAT-40, EDI-3 risk, and EDI-3 GPM. Results demonstrated all EDs exhibited significantly higher FOFM scores compared to controls. In EDs, AN and BN showed significantly greater scores in FOFM-anxiety about eating (AE) and FOFM-food avoidance behaviors (FAB) compared to BED and OSFED. Significant changes in all FOFM subscale scores were observed in patients who completed mid-treatment ($n = 79$). FOFM-AE emerged as the most significant predictor of early improvements not only in EAT-40 total and EDI-3 risk scores but also in EDI-3-GPM scores. Results indicate that fear of food is a transdiagnostic construct characterizing all EDs, in particular AN and BN. Outpatient interventions assessing and targeting early fear of food, in particular in anxiety about eating, may help improve prognosis and support recovery in EDs. The role of fear of food in general psychopathology, beyond EDspecific symptoms, represents a valuable addition to the field and highlights the interplay between fear-related and ED symptoms, providing new directions for future ED research and treatment.

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EATING DISORDERS (EDS) ARE COMPLEX PSYCHIATRIC ILLNESSES characterized by dysfunctional eating or weight-control behaviors, with serious consequences both on physical health and psychological functioning (Treasure et al., 2020). According to the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-5-TR; American Psychiatric Association, 2022) and the *International Classification of Diseases* (ICD-11; World Health Organization, 2022) the main ED diagnoses include anorexia nervosa (AN), bulimia nervosa (BN), binge-eating disorder (BED) and other specified feeding or eating disorders (OSFED). Although there is high heterogeneity in symptom presentation and clinical characteristics across ED diagnoses, recent studies (Calugi et al., 2018; DuBois et al., 2017; Forrest et al., 2018; Lampard et al., 2011) aligned with the enhanced transdiagnostic cognitive behavioral theory (CBT-E) for EDs (Fairburn et al., 2003) have highlighted the pivotal role of the over-evaluation of eating, weight, and shape as central features of EDs. Beyond these core elements, EDs are also characterized by fear (e.g., fear of overeating, fatness and weight gain), anxiety (e.g., pre-meal anxiety and negative beliefs about the consequences of eating), and behavioral avoidance (e.g., avoiding caloric intake; Fairburn et al., 2003; Steinglass et al., 2011, 2014). These features have been operationalized in the construct of fear of food, an anxiety-related construct defined by heightened anxiety about eating, avoidance of food, and distressing concerns related to eating (Levinson & Byrne, 2015). A recent network analysis confirmed the centrality of food-related anxiety as one of the core fears in EDs, underscoring its role in maintaining ED psychopathology, predicting symptom persistence, and contributing to relapse (Levinson & Williams, 2020). Furthermore, fear of food has been observed across diagnostic categories in ED patients, suggesting it represents a transdiagnostic feature of these disorders (Levinson & Byrne, 2015).

Increased anxiety related to eating has been documented in individuals with AN, BN, BED and OSFED (Levinson & Byrne, 2015). In patients with AN, eating-related anxiety is particularly evident between the presentation and consumption of a meal. In these situations, heightened anxiety is often observed in response to foods of higher caloric content, beliefs about their consequences on shape, weight, and mood (Steinglass et al., 2010), the lack of control over eating environment (e.g., eating food prepared from someone else) and planning (e.g., eating more than planned; Webb et al., 2011). In this sense, eating-related anxiety experienced by patients with AN leads to a maintenance of restrained eating behaviors. In contrast, in ED groups characterized by binge-eating, such as BN, BED and certain forms of OSFED, such as short-duration BED and night eating syndrome, anxiety is associated with the loss of control over eating, leading to disinhibited eating behaviors, such as binge eating episodes (Aimé et al., 2017). Although the specific features of eating-related anxiety in these populations remain less well understood compared to AN and BN, accumulating evidence suggests that, in contrast to disorders not characterized by binge-eating, those who engage in binge-eating may experience eating-related anxiety that is less tied to a perceived loss of planning during eating episodes (Webb et al., 2011) and more closely linked to emotional triggers such as stress or negative affect (Goldschmidt et al., 2014; Leehr et al., 2015). Furthermore, it has been shown that weight- and shape-related concerns appear to play an important role in shaping anxiety in this clinical population (Melisse & Dingemans, 2025). Taken together, these findings suggest that eating-related anxiety in binge-spectrum disorders may reflect a distinct clinical profile characterized by both emotional dysregulation and weight/shape concerns, highlighting the importance of addressing these mechanisms in treatment approaches.

Eating-related avoidance behaviors are another defining and transdiagnostic feature across all EDs (Melles & Jansen, 2023). For instance, in AN these behaviors include food avoidance and refusal to be weighed (Radix et al., 2019). In BN, they manifest as phobias toward "forbidden" foods, leading to their elimination from the diet, except during binge episodes (Alvarenga et al., 2008; Guertin & Conger, 1999). Avoidance behaviors have also been reported in patients with BED, especially in terms of cognitive avoidance, such as suppressing thoughts related to food (Barnes et al., 2013). Behavioral avoidance has also been documented in patients with BED, but mostly in terms of body-avoidance, such as avoiding wearing clothes that make them particularly aware of their body, rather than food-related (Grilo et al., 2005; Reas et al., 2005).

In addition to fear of food and food avoidance behaviors, EDs are also characterized by maladaptive thoughts underlying food- and eating-related anxiety. These thoughts often result in intense worry and rumination (Smith et al., 2018), reflecting a broader pattern of feared concerns that persist across various ED presentations (Sapuppo et al., 2018; Startup et al., 2013; Sun et al., 2017).

The multifaceted construct of fear of food is significantly associated with core ED symptomatology such as body dissatisfaction, drive for thinness, and bulimic symptoms across different EDs (Levinson et al., 2019; Levinson & Byrne, 2015). A recent time-series investigation study also showed that in remitted patients with AN fear-related symptoms were positively associated with urges to restrict, purge, binge, and exercise, with food avoidance behaviors in particular predicting future binge eating and excessive exercise (Christian et al., 2024). Despite the centrality of this construct both in the acute and remission stages of EDs, studies addressing this aspect show room for improvement. Specifically, existing research has often focused on limited samples, predominantly involving patients with AN (Levinson et al., 2017, 2019), while neglecting other diagnoses such as BN, BED, and OSFED (Levinson & Byrne, 2015; Steinglass et al., 2010). Moreover, there is a lack of longitudinal studies, particularly those investigating changes in fear of food during the early stages of treatment. Given that early improvement in core symptoms are often strong indicators of treatment outcomes in EDs (Vall & Wade, 2016), an early reduction in fear of food could hold clinical significance due to its established links with core ED symptomatology. To date, no studies have yet explored whether fear of food changes early during treatment, and whether these changes are associated with changes beyond ED-specific symptoms.

Therefore, given the limited prior research on fear of food across different ED diagnoses, its early changes during treatment, and its associations with both ED symptomatology and general psychopathology, the present study adopts an exploratory approach. Specifically, it aims to:

(a) describe the differences in fear of food between controls from the general population and outpatients with varying ED diagnoses (specifically AN, BN, BED and OSFED); (b) examine early modifications in fear of food, ED symptomatology and ED-related psychopathology by midtreatment (4 months after treatment initiation), consisting of cognitive-behavioral-based therapy (CBT) with integrated nutritional rehabilitation and counseling (NRC) in a multidisciplinary outpatient setting; (c) explore whether mid-treatment changes in fear of food are associated with concurrent changes in ED symptomatology and ED-related psychopathology.

Materials and Methods

The project was approved by the University Bioethics Committee (Statement of Approval date 6/30/2016). Informed consent was obtained from all participants who agreed to participate in the study.

The study was conducted in Italy, at the Centro Gruber in Bologna (Fondazione Gruber Onlus), a multidisciplinary outpatient clinic specialized in the treatment of EDs.

SAMPLE

A convenience sample of consecutively screened outpatients who met DSM-5 diagnostic criteria for EDs seeking treatment in a specialized outpatient clinic for the treatment of EDs was recruited for the study. ED diagnoses were established at intake by the consensus of a psychiatrist and a clinical psychologist independently using the Structured Clinical Interview for DSM-5 (First et al., 2016). In the event of disagreement, the case was reviewed and discussed until a consensus diagnosis was reached between professionals. An ad hoc questionnaire was administered to collect sociodemographic data, while medical records were consulted to collect illness duration and body mass index (BMI) based on objective measurements (i.e., height and weight) taken by healthcare professionals during the patients' clinical intake assessment. Inclusion criteria for participation in the study were: (a) age ≥ 18 and (b) having a diagnosis of AN, BN, BED, or OSFED. The exclusion criteria were: (a) lack of capacity to consent for research, (b) ED diagnosis secondary to a physical health or metabolic condition, (c) comorbid drug/alcohol abuse, psychotic or neurocognitive disorders, and pregnancy.

A total of approximately 348 patients were screened for eligibility during the recruitment period. Of these, 282 (81%) met inclusion criteria and provided informed consent to participate in the study. Out of the 282 ED patients included in the study, a subsample of 79 patients who completed midtreatment at the time of analyses was analyzed to examine preliminary early changes in ED symptomatology and fear of food components. See Figure 1 for a graphical representation of the recruitment process.

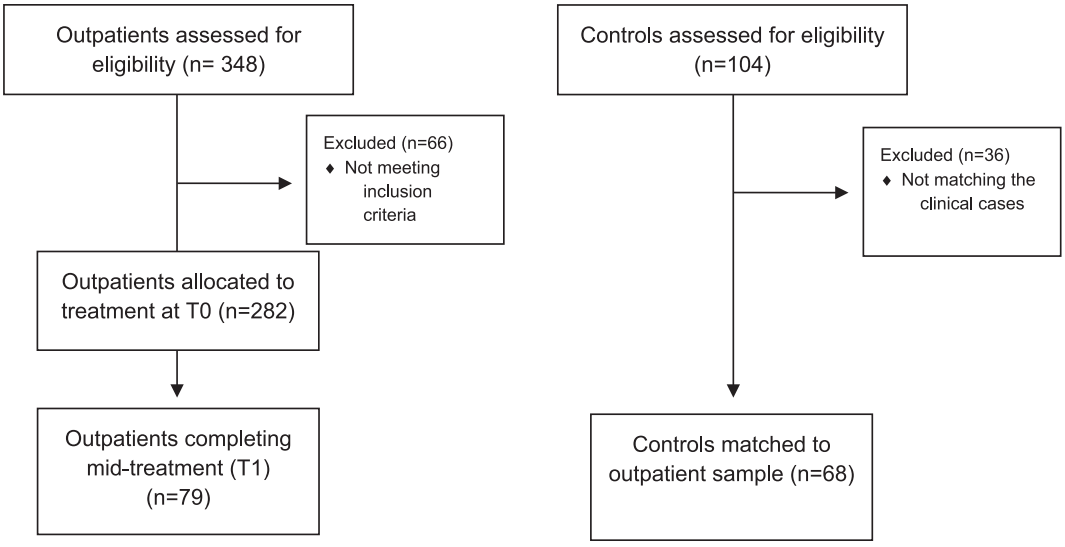

FIGURE 1 Flow diagram of the recruitment process.

The control group of adults from the general population was recruited online through questionnaires distributed via the Google Forms platform using the following inclusion criteria: (a) aged ≥ 18 and (b) no prior diagnosis of any ED according to DSM-5 diagnostic criteria. Exclusion criteria were: (a) lack of capacity to consent for research and (b) lifetime history of EDs according to DSM-5 diagnostic criteria either as primary diagnosis or in comorbidity to other mental health or due to a physical condition. Recruitment was carried out by posting the Google Forms link on various online communities and social media platforms to reach participants from general population with sociodemographic characteristics similar to those of the clinical sample (in terms of gender and age). However, as this approach did not fully achieve the desired demographic similarity, post hoc matching was performed to align controls with clinical participants on gender and age. This procedure aimed to improve group comparability for subsequent analyses. Out of the total controls recruited (n = 104), 68 participants were successfully matched to clinical cases (Figure 1).

TREATMENT

Treatment consisted of a multidisciplinary outpatient program integrating CBT for EDs (Ballardini & Schumann, 2011), consisting of weekly individual sessions of one hour each conducted by trained psychotherapists, and a program conducted by nutritional physicians specialized in EDs. The integration of an NRC program follows the recommendations of the American Psychiatric Association Practice guidelines for the treatment of ED patients (Crone et al., 2023), which specify that nutritional rehabilitation should be provided for underweight patients and nutritional counseling should be provided for normal weight and overweight patients (Abbate-Daga et al., 2007; Garner & Garfinkel, 1979).

The CBT psychotherapeutic sessions refer to the traditional CBT protocol for EDs including: education about the ED, orientation to the treatment and to the cognitive model of the disorder, the structure of therapy and possible outcomes; the identification of patients' goals; behavioral techniques including exposure to avoided foods, self-monitoring, self-regulatory strategies to reduce frequency of dysfunctional eating behaviors, and normalization of eating patterns; teaching patients to identify and alter dysfunctional cognitions through the use of the diary, behavioral experiments, and cognitive restructuring; relapse prevention (Ballardini & Schumann, 2011).

NRC was conducted by a nutritional physician specialized in EDs and included: the management of specific medical and nutritional issues and physical comorbidities; psychoeducation about dysfunctional thoughts regarding food, weight and body; psychoeducation about weight restoration, stabilization or weight loss; psychoeducation about health consequences of the illness following the clinical recommendations for the specific ED diagnosis (Crone et al., 2023). Nutritional goals were individually tailored based on diagnosis and clinical presentation, with regular monitoring of weight, nutritional intake, and relevant medical parameters (e.g., electrolytes, vital signs), in accordance with APA (2022) and NICE (2017) guideli-

nes (Crone et al., 2023; NICE, 2017). NRC also aimed to support the gradual development of autonomous and flexible eating behaviors, promoting patient collaboration in meal planning and reflection on bodily needs. The overall therapeutic stance shared key principles with Specialist Supportive Clinical Management (SSCM) (McIntosh et al., 2006) adapted for all EDs, such as a patient-centered approach, clinical monitoring, and the integration of psychoeducation and supportive dialogue to foster engagement and motivation.

NRC sessions were either preceded or followed by one-hour CBT psychotherapeutic sessions. Multidisciplinary collaboration was ensured through daily case discussions between psychotherapists and nutritional physicians, allowing the integration of nutritional and psychological insights and the adjustment of treatment targets based on evolving patient needs. Average duration of the entire treatment program for patients is roughly 24 CBT psychotherapeutic sessions and 24 sessions of NRC. Midtreatment corresponds to 12 CBT psychotherapeutic sessions integrated with 12 sessions of NRC, generally spanning about 4 months, depending on individual patient attendance and clinical needs (Ballardini & Schumann, 2011).

All treatments were delivered in person at our outpatient clinic by a multidisciplinary team specialized in EDs. Data collection took place between 2021 and 2024, over a period of approximately 3 years. Although the study was initially planned to begin earlier, the COVID-19 pandemic caused delays in both recruitment and treatment implementation. Recruitment resumed once in-person services could be safely offered again in accordance with public health guidelines.

ASSESSMENT

ED outpatients before treatment intake (T0 = baseline; $n = 282$) and matched controls ($n = 68$) were assessed with the following self-report questionnaires:

Fear of Food Measure (FOFM; Levinson & Byrne, 2015)

A self-report questionnaire that assesses the presence of fear of food in terms of emotional, cognitive, and behavioral components: Anxiety about Eating (AE), Food Avoidance Behaviors (FAB), and Feared Concerns related to eating (FC). Authors have not identified a cut-off that indicates the need for clinical attention. Higher scores indicate greater severity of fear of food. In the original validation study the internal consistency was excellent for two scales (AE $\alpha = 0.95$ and FC $\alpha = 0.90$) and good for the last scale (FAB $\alpha = 0.74$), and the test exhibited excellent test-retest reliability. Similarly, in our sample the internal consistency was excellent for AE ($\alpha = 0.96$) and FC ($\alpha = 0.90$) and good for FAB ($\alpha = 0.81$). Since the FOFM has not been translated and validated in Italian yet, for the present study the questionnaire was translated into Italian using the Back-Translation method. This method involves an initial translation of the items from the original language into Italian by at least two researchers who must agree on the final version. This version is then reviewed by a bilingual individual (L.T.) who, through a retranslation into the original language, confirms the comparability between the two versions (Behling & Law, 2000).

Eating Attitudes Test-40 (EAT-40; Cuzzolaro & Petrilli, 1988; Garner & Garfinkel, 1979)

A 40-item screening measure identifying behavioral and cognitive patterns of EDs. The measure yields three subscale scores for dimensions of dieting, bulimia and food preoccupations, oral control, and a total score ranging from 0 to 120. Higher scores indicate greater ED psychopathology. It has been widely used in adolescent (Miotto et al., 2003) and Italian populations (Cuzzolaro & Petrilli, 1988; Garner, 2004). Cronbach's alpha coefficient ranged from 0.79 to 0.94 in female patients with AN. Test-retest reliability was 0.84, and the validity coefficient was 0.87 (Garner & Garfinkel, 1979). In our sample, Cronbach's alpha showed excellent internal consistency for dieting ($\alpha = 0.90$) and bulimia and food preoccupations ($\alpha = 0.92$) and good for oral contral ($\alpha = 0.81$).

Eating Disorder Inventory-3 (EDI-3; Fairburn & Garner, 1986; Giannini et al., 2008)

A widely used 91-item self-report measure of symptomatology associated with EDs which contains six composite scales. EDI-3 Risk and EDI-3 General Psychological Maladjustment (GPM) were used in the current study, representing ED symptomatology and ED-related psychopathology. Higher scores indicate greater severity. The EDI-3 showed good psychometric properties, with Cronbach's alpha coefficients for subscales of 0.90-0.97 and test-retest $r = 0.98$ (Fairburn & Garner, 1986; Miller & Chapman, 2001). In our sample, Cronbach's alpha coefficient was good for EDI-3 Risk ($\alpha = 0.83$) and excellent for EDI-3-GPM ($\alpha = 0.90$).

ED outpatients completing mid-treatment (T1; $n = 78$) filled in the aforementioned questionnaires

generally after 4 months from intake, corresponding to 12 CBT psychotherapeutic sessions integrated with 12 sessions of NRC.

STATISTICAL ANALYSES

Statistical analyses were performed using the Statistical Package for Social Science (SPSS). Sample size was determined a priori through a power analysis performed with G*Power 3.1, assuming $\alpha = 0.05$, power = 0.80, and a medium effect size (f = 0.25 for MANOVA, Cohen's $d = 0.5$ for *t*-test, $f^2 = 0.15$ for regressions), resulting in a minimum estimated sample of $N = 206$ participants for MANOVA, $N = 27$ for t-test, and $N = 68$ for regressions. Descriptive analyses were run to examine sample characteristics. Multivariate Analysis of Variance (MANOVA) with post-hoc comparisons and Bonferroni corrections were run to compare controls and ED diagnostic groups in FOFM subscales at baseline (T0; Aim A).

Independent *t*-test was performed to compare patients who did and did not complete midtreatment in terms of BMI, illness duration, ED symptomatology and fear of food. A t-test for paired samples was performed on patients who had completed midtreatment (T1) at time of analyses ($n = 79$) to evaluate the modifications between baseline (T0) and midtreatment (T1) in ED symptomatology in terms of EAT-40-Total, EDI-3-Risk, and EDI-3-GPM scores, as well as the examined changes in FOFM subscale scores (Aim B).

Linear regression analyses were performed to test whether changes in FOFM-AE, FOFM-FAB and FOFM-FC scores were associated with changes in eating symptomatology (Aim C). Changes were operationalized as the difference (Δ) between midtreatment (T1) and baseline (T0) scores. Midtreatment corresponded to approximately 4 months after treatment initiation, including 12 sessions of CBT and 12 sessions of nutritional rehabilitation and counseling (NRC).

Additional analyses were also carried out in a subgroup of underweight patients (BMI lower than 18.5 at baseline). In particular, a *t*-test for paired samples was performed to evaluate whether changes in BMI occurred in this sub-group between T0 and T1 and a linear regression analysis was performed to test whether changes between T0 and T1 in FOFM-AE, FOFM-FAB and FOFM-FC scores were associated with changes between T0 and T1 in BMI in this sub-group.

The level of statistical significance adopted in this study is $\alpha < 0.05$.

Results

SOCIODEMOGRAPHIC AND CLINICAL CHARACTERISTICS OF THE SAMPLE

ED patients ($n = 282$) were all female, with mean age of 26.38 ± 8.68 years and predominantly with an AN diagnosis (32.26%). The mean BMI of the total ED sample was 23.17 ± 8.08 (16.87 ± 2.54 for AN, 22.32 ± 5.28 for BN, 33.73 ± 9.21 for BED, and 21.86 ± 6.88 for OSFED diagnoses). Controls ($n = 68$) were all female, with a mean age of 25.09 ± 4.53 years. Patients and controls were matched for sex, age, marital status, and occupation, while they significantly differed in education, BMI, and scores in FOFM, EAT, and EDI-3 measures. See Table 1 for further details on the sociodemographic and clinical characteristics of the sample.

All participants self-identified as White/European, reflecting the demographic composition of the clinical and general population from which the sample was drawn.

DIFFERENCES IN FEAR OF FOOD BETWEEN ED DIAGNOSTIC SUBGROUPS AND CONTROLS

To address the first aim of the study—investigating differences in fear of food assessed through the FOFM (anxiety about eating, food avoidance behaviors and feared concerns) between controls and outpatients with varying ED diagnoses (AN, BN, BED and OSFED)—MANOVA and ANOVA analyses were performed. There was a statistically significant difference in FOFM subscales based on the group variable (control, AN, BN, BED, OSFED; F (12, 881.327) = 17.483, $p < 0.0001$; Wilk's $\Lambda = 0.568$, partial $\eta^2 = 0.172$). A statistically significant effect for the group variable was found on FOFM-anxiety about eating (F (4, 311) = 47.366; $p < 0.0001$; partial $\eta^2 = 0.361$), food avoidance behaviors (F (4, 311) = 35.730; $p < 0.0001$; partial $\eta^2 = 0.299$), and feared concerns (F (4, 311) = 40.260; $p < 0.0001$; partial $\eta^2 = 0.325$). See Table 2 and 3 for further details.

Means and standard deviations for groups in FOFM subscales are shown in Table 2. Post hoc comparisons with Bonferroni corrections (Table 3) showed that controls differed significantly from all four ED diagnostic subgroups with very large effect sizes in all FOFM subscales, except for comparisons between BED and controls in FOFM-food avoidance behaviors.

Concerning differences between diagnostic subgroups, while significant differences did not emerge between outpatients with AN and BN, those with AN reported significantly higher scores

Table 1
Sociodemographic and Clinical Characteristics of the Sample

	ED patients ($n = 282$)	Controls ($n = 68$)	p	d
		***t*-test**		
Age in years (M ± SD)	26.38 ± 8.68	25.09 ± 4.53	0.09	0.16
BMI (M ± SD)	23.17 ± 8.08	21.12 ± 2.71	**0.002**	0.25
Diagnosis (n, %)				
AN	91 (32.26%)			
BN	66 (23.40%)			
BED	63 (22.34%)			
*OSFED**	52 (18.43%)			
(M ± SD) FOFM-AE	30.15 ± 14.93	12.49 ± 8.32	**<0.001**	1.27
FOFM-FAB	20.89 ± 9.78	12.03 ± 6.47	**<0.001**	0.95
FOFM-FC	34.03 ± 15.72	14.94 ± 8.24	**<0.001**	0.74
EAT 40-Total	36.66 ± 23.89	11.03 ± 9.61	**<0.001**	1.17
EDI-3-Risk	53.84 ± 23.82	25.53 ± 18.91	**<0.001**	1.23
EDI-3-GPM	105.08 ± 44.60	61.28 ± 33.93	**<0.001**	1.02
		Fisher's F		
Occupation				
Student (n, %)	161 (57.09%)	42 (61.76%)	0.07	V = 0.08
Employed (n, %)	87 (30.85%)	21 (30.88%)		
Unemployed (n, %)	20 (7.09%)	1 (1.47%)		
Retired (n, %)	7 (2.48%)	0		
Education				
Middle school (n, %)	69 (24.46%)	3 (4.41%)	**<0.001**	V = 0.19
High school (n, %)	118 (41.84%)	30 (44.11%)		
Degree (n, %)	87 (30.85%)	35 (51.47%)		
		Chi squared		
Marital status				
Single (n, %)	243 (86.17%)	64 (94.11%)	0.11	V = 0.08
In a relationship (n, %)	31 (10.99%)	3 (4.41%)		

Notes. Significant values in bold.
AN = anorexia nervosa; BED = binge-eating disorder; BMI = body mass index; BN = bulimia nervosa; EAT-40 = Eating Attitudes Test; EDI-3 = Eating Disorder Inventory General Psychological Maladjustment; FOFM-AE = Fear of Food Measure-Anxiety about Eating; FOFM-FAB = Fear Of Food Measure–Food Avoidance Behaviors; FOFM-FC = Fear of Food Measure–Feared Concerns; M = mean; OSFED = Other specified feeding or eating disorders; SD = standard deviation.
* *The OSFED group (N = 52) was composed of the following subtypes: atypical anorexia nervosa (n = 22; 42.3%), low-frequency or short-duration bulimia nervosa (n = 12; 23.1%), low-frequency or short-duration binge-eating disorder (n = 9; 17.3%), purging disorder (n = 6; 11.5%), and night eating syndrome (n = 3; 5.8%).*

Table 2
Means and Standard Deviations of Control and ED Groups in FOFM Subscales

Measure	AN ($n = 28$)	BN ($n = 29$)	BED ($n = 36$)	OSFED ($n = 29$)	Controls ($n = 94$)
FOFM-AE	34.04 ± 17.05	37.10 ± 13.84	20.61 ± 11.61	22.31 ± ± 11.60	11.95 ± 8.238
FOFM-FAB	23.00 ± 10.14	24.83 ± 11.38	13.72 ± 6.81	17.62 ± 8.69	11.45 ± 6.063
FOFM-FC	34.54 ± 16.80	37.59 ± 17.58	25.86 ± 11.94	25.28 ± 11.58	14.96 ± 8.368

with medium to large effect sizes in all FOFM subscales compared to outpatients with BED and OSFED. Similarly, patients with BN reported significantly higher scores with medium to large effect sizes than patients with BED and OSFED in all FOFM subscales. Ultimately, patients with BED reported medium effect size differences with patients with OSFED in food avoidance behaviors.

EARLY CHANGES IN FEAR OF FOOD AND ED-RELATED MEASURES IN ED OUTPATIENTS

To address the second aim of the study—examining early modifications in fear of food assessed through the FOFM (anxiety about eating, food avoidance behaviors and feared concerns), in ED symptomatology assessed through the EAT-40 (total score) and the EDI-3-risk, and in ED-related

Table 3
Post-Hoc Comparisons Using Bonferroni Between Groups in FOFM Subscales Showing Mean Differences and Effect Sizes (η^2)

	AN ($n = 81$)	BN ($n = 60$)	BED ($n = 62$)	OSFED ($n = 51$)	Controls ($n = 63$)
FOFM-AE					
AN	1	−0.40 (0.00)	14.70^{+} (0.19)	10.48^{+} (0.07)	23.44^{+} (0.30)
BN		1	15.10^{+} (0.17)	10.88^{+} (0.06)	23.83^{+} (0.28)
BED			1	−4.22 (0.01)	8.74** (0.05)
OSFED				1	−12.96^{+} (0.09)
Controls					1
FOFM-FAB					
AN	1	1.12 (0.00)	11.38^{+} (0.18)	5.81** (0.05)	13.01^{+} (0.23)
BN		1	10.26^{+} (0.13)	4.69* (0.03)	11.89^{+} (0.18)
BED			1	−5.56* (0.04)	1.64 (0.00)
OSFED				1	7.20^{+} (0.06)
Controls					1
FOFM-FC					
AN	1	−0.96 (0.00)	−11.78^{+} (0.08)	9.31** (0.05)	23.80^{+} (0.28)
BN		1	−12.74^{+} (0.08)	10.27^{+}(0.05)	24.76^{+} (0.27)
BED			1	−2.47 (0.00)	12.01^{+} (0.07)
OSFED				1	14.48^{+} (0.09)
Controls					1

Notes. Mean differences shown.
AN, anorexia nervosa; BED, binge-eating disorder; BN, bulimia nervosa; FOFM-AE, Fear Of Food Measure-Anxiety about Eating; FOFM-FAB, Fear Of Food Measure-Food Avoidance Behaviors; FOFM-FC, Fear Of Food Measure-Feared Concerns; OSFED, Other specified feeding or eating disorders
* $p < .05$.
** $p < .01$.
$^{+}$ $p < .001$.

psychopathology assessed through the EDI-3-GPM by mid-treatment—*t*-test for paired samples were conducted in a subsample of patients ($n = 79$) who concluded mid-treatment. In this subgroup, patients met criteria for AN ($n = 35$), BN ($n = 16$), BED ($n = 14$), and OSFED ($n = 14$). The mean age of this sample at T0 was 25.95 ± 9.31 years. At baseline, patients who completed midtreatment did not differ significantly from non-completers in terms of BMI. However, completers showed a significantly longer illness duration ($t = 2.34$, $p = 0.02$), higher fear of food as measured by both the FOFM-AE ($t = 2.25$, $p = 0.02$) and FOFM-FAB ($t = 2.11$, $p = 0.03$), and a trend toward greater overall ED symptomatology ($t = 2.56$, $p = 0.01$). Significant changes between T0 and T1 in all FOFM subscale scores were observed. All ED symptomatology and ED-related psychopathology scales, specifically EAT-40-total, EDI-3-risk, and EDI-3-General Psychological Maladjustement, showed significant reductions. See Table 4.

RELATIONS BETWEEN FOFM CHANGES AND EAT AND EDI-3 CHANGES

To address the third aim of the study and test whether changes in FOFM subscales (ΔFOFM-AE = FOFM-AE score at T1 − FOFM-AE score at T0; ΔFOFM-FAB = FOFM-FAB score at T1 − FOFM-FAB score at T0; ΔFOFM-FC = FOFM-FC score at T1 − FOFM-FC score at T0) were associated with changes in ED symptomatology assessed through the EAT total score (ΔEAT = EAT-total score at T1 − EAT-total score at T0) and the EDI-3-risk subscale (ΔEDI-3-risk = EDI-3-risk score at T1 − EDI-3-risk score at T0) and changes in ED-related psychopathology assessed through the EDI-3-GPM (ΔEDI-3-GPM = EDI-3-GPM score at T1 − EDI-3-GPM score at T0), three linear regression analyses were performed respectively entering ΔEAT-total score (model 1), ΔEDI-3-risk (model 2) and ΔEDI-3-GPM (model 3) as dependent variables. Overall, all three regression models were statistically significant.

Model 1 explained 45% of the variance in EAT-total score changes ($R^2 = 0.45$, $F(73,3) = 21.75$, $p < 0.001$), with the most significant predictor being ΔFOFM-AE, followed by ΔFOFM-FAB (Table 5). Model 2 explained 35% of the variance in ΔEDI-3-risk ($R^2 = 0.35$, $F(73,3) = 15.03$, $p < 0.001$), with the most important predictor being changes ΔFOFM-AE, followed by changes in ΔFOFM-FC

Table 4
T-Test for Paired Samples in ED Outpatients ($n = 78$)

Measure	T0 M ± SD	T1 M ± SD	Statistic t (df)	p
FOFM-AE	33.24 ± 15.17	25.90 ± 14.01	$t(78) = 5.399$	<.001
FOFM-FAB	22.80 ± 10.04	19.24 ± 8.87	$t(78) = 5.608$	<.001
FOFM-FC	36.12 ± 16.14	29.95 ± 14.81	$t(77) = 9.491$	<.001
EAT-40-Total	42.33 ± 23.94	27.72 ± 22.18	$t(77) = 7.600$	<.001
EDI-3-Risk	54.67 ± 22.93	40.35 ± 22.23	$t(77) = 6.431$	<.001
EDI-3-GPM	107.09 ± 44.34	88.76 ± 50.68	$t(77) = 4.652$	<.001

Notes: df, Degrees of Freedom; EAT-40, Eating Attitudes Test 40; EDI-3, Eating Disorder Inventory-3-General Psychological Maladjustment; FOFM-AE, Fear of Food Measure-Anxiety about Eating; FOFM-FAB, Fear Of Food Measure-Food Avoidance Behaviors; FOFM-FC, Fear Of Food Measure-Feared Concerns; M, Mean; p, p-value of statistical significance; T0, baseline; T1, 12 sessions; SD, Standard Deviation.

Table 5
Regression Model Coefficients

Predictors	B	ES B	b	$t(p)$
Model 1 (outcome: ΔEAT-40-total score)				
ΔFOFM-AE	0.73	0.16	0.51	**4.43 (<0.001)**
ΔFOFM-FAB	0.57	0.22	0.31	**2.53 (0.01)**
ΔFOFM-FC	0.056	0.16	0.04	0.34 (0.73)
Model 2 (outcome: ΔEDI-3-risk)				
ΔFOFM-AE	0.57	0.20	0.35	**2.83 (0.006)**
ΔFOFM-FAB	0.16	0.28	0.07	0.55 (0.58)
ΔFOFM-FC	0.50	0.20	0.37	**2.48 (0.01)**
Model 3 (outcome: ΔEDI-3-GPM)				
ΔFOFM-AE	1.03	0.36	0.36	**2.88 (0.005)**
ΔFOFM-FAB	0.21	0.51	0.43	0.43 (0.66)
ΔFOFM-FC	0.61	0.36	0.26	1.69 (0.09)

Notes: EAT-40, Eating Attitudes Test 40; EDI-3, Eating Disorder Inventory-3-General Psychological Maladjustment; FOFM-AE, Fear of Food Measure-Anxiety about Eating; FOFM-FAB, Fear Of Food Measure-Food Avoidance Behaviors; FOFM-FC, Fear Of Food Measure-Feared Concerns.

(Table 5). Model 3 explained 34% of the variance in ΔEDI-3-GPM ($R^2 = 0.34$, $F(73,3) = 14.24$, $p < 0.001$), with the only significant predictor being ΔFOFM-AE (Table 5).

EARLY BMI RESTORATION AND ASSOCIATION BETWEEN FOFM CHANGES AND BMI CHANGES IN UNDERWEIGHT SUBGROUP

A sub-group of $n = 43$ patients in the study sample was underweight at baseline, including $n = 35$ patients with AN and $n = 8$ patients with OSFED. An early significant increase in BMI was observed from baseline (T0) to mid-treatment (T1) in this subgroup, as reported in Supplementary Table S1.

Regression analysis run in this sub-group to evaluate whether changes in FOFM subscales between T0 and T1 predicted changes in BMI between T0 and T1 did not yield statistically significant results, as reported in Supplementary Table S2.

Discussion

This study aimed to examine the emotional, behavioral, and cognitive components of FOFM in an ED outpatient sample spanning all four major ED diagnoses (AN, BN, BED and OSFED). Additionally, the study aimed to investigate early preliminary changes in fear of food components following an outpatient CBT-based treatment integrated with NRC and to assess whether changes in fear of food at mid-treatment were associated with changes in ED symptomatology and overall psychological changes beyond ED-specific symptoms. In line with the present study, previous findings on fear of eating (Steinglass et al., 2012), and more specifically on the construct of fear of food (Levinson & Byrne, 2015), have shown that ED patients exhibit significantly greater food-related anxiety, associated food avoidance behaviors, and feared concerns compared to controls.

Moreover, following previous findings showing limited differences in ED fears between ED diagnoses (Brown & Levinson, 2022), in the current study FOFM components did not differ significantly between AN and BN groups. However, compared to patients with BED and OSFED, both patients with AN and BN exhibited significantly higher levels of anxiety about eating and food avoidance behaviors, with medium to large effect sizes, especially in comparison to patients with BED. Differences in feared concerns were also observed but to a lesser degree. These findings align with previous research suggesting that fear of food, while considered a transdiagnostic feature across EDs (Levinson & Byrne, 2015), may be more specifically associated with a drive for thinness (a core characteristic of AN and BN) rather than with disorders, such as BED, in which

binge-eating is central and typically not accompanied by the same level of weight- and shape-related concerns (Levinson et al., 2017).

Despite the higher fear of food observed in AN and BN, patients with BED in our sample exhibited significantly higher scores in anxiety about eating and feared concerns compared to controls. Thus our findings not only confirm the well-established association between fear of food and AN/BN symptoms, but also suggest that this construct may also be oserved in BED as well, although to a lower degree than AN and BN. This interpretation is consistent with previous studies showing that individuals with BED report greater fear of weight gain compared to individuals with overweight or obesity but without a BED diagnosis (Bullock et al., 2021) and levels of such fears comparable to those observed in other ED diagnoses (Melisse & Dingemans, 2025). Moreover, weight/shape concern has been identified as a prodromal symptom in the onset of BED (Stice et al., 2021), further underscoring its clinical importance. Notably, having a lower BMI or being weight suppressed, common in patients with AN and BN respectively, has been linked in the literature to higher food-related anxiety (Levinson et al., 2019), which may explain the higher FOFM scores observed in AN and BN patients in our study.

The heightened anxiety about eating observed in AN and BN compared to other ED subgroups might stem from a fear of losing control over eating—either a subjective or objective core characteristic of these disorders—which is less prominent in BED (Fairburn & Garner, 1986; Pyle et al., 1991; Wilfley et al., 2000). While BED is indeed characterized by a loss of control over eating, typically resulting in binge-eating episodes, the fear of losing control is more closely linked to concerns about weight gain or becoming fat, which is a core features of AN and BN, but a more heterogeneous characteristic in BED patients. More specifically, while weight- and shape-related concerns appear to be a key diagnostic feature for AN and BN but not for BED (American Psychiatric Association, 2022), a substantial proportion of BED patients nevertheless experience significant body image disturbances to the point that authors suggest that these symptoms should be included as a severity specifier (Grilo, 2013; Grilo et al., 2019; Lewer et al., 2016), which explains why BED patients still scored higher than controls in all FOFM subscales. Therefore, the present findings may reflect the heterogeneity of body image experiences within BED samples.

Additionally, longitudinal data suggest that DSM-defined AN and BN diagnoses may represent different phases of a single ED continuum (Eddy et al., 2008; Strober et al., 1997), with similar emotional, cognitive, and behavioral psychopathological aspects, such as anxiety, food preoccupations, and dietary restraint (Elran-Barak et al., 2015; Levinson & Byrne, 2015; Wilfley et al., 2000) explaining the lack of differences. However, this lack of differences between patients with AN and BN in fear of food might also indicate that the FOFM might not be sufficiently sensitive to detect meaningful clinical distinctions between these diagnostic groups. Further research is needed to evaluate whether the FOFM adequately reflects diagnostic distinctions or whether it primarily captures shared features across ED presentations.

Differences between patients with AN and BN and between patients with BED and OSFED were less pronounced on the scale of feared concerns. This might imply that rumination and worry, recognized as transdiagnostic features across EDs (Startup et al., 2013), may indeed contribute to cognitive dietary restriction by reinforcing persistent preoccupations with food and eating. These cognitive patterns could drive individuals to constantly monitor and limit dietary intake, creating a shared behavioral framework that bridges diagnostic categories and highlights similarities across all EDs. Overall, the lower levels of fear of food observed in patients with BED and OSFED compared to AN and BN may reflect other problematic food-related aspects specific of these ED diagnoses. For instance, BED has been reconceptualized as comparable to substance use disorders, sharing behavioral traits such as craving, desire thinking, and using substances (or binge eating) for emotion regulation (Gearhardt et al., 2013; Schulte et al., 2016; Spada et al., 2016). In OSFED, given its diagnostic heterogeneity, food-related fears might serve diverse psychological functions beyond strictly weight-related fears, such as social judgment or losing control (Butler et al., 2023). However, while difficulties related to emotion regulation, craving, or food neophobia may be relevant therapeutic targets for some individuals with OSFED, our results highlight that OSFED patients also report elevated fear of food compared to controls, underscoring the need for comprehensive treatment strategies that address both food-related anxiety and broader psychopathological mechanisms. Within this diagnostically heterogeneous category, the FOFM may serve as a valuable clinical tool to detect residual or atypical fear profiles that might otherwise be overlooked by standard assessments and help guide more

individualized therapeutic strategies for OSFED patients.

Finally, patients with BED and OSFED, similar to those with AN and BN, did not differ significantly in terms of FOFM, except for food avoidance behaviors, in which patients with BED resulted in being less impaired than patients with OSFED. This result reflects previous findings showing that avoidance behaviors in patients with BED are mostly endorsed in terms of body-avoidance behaviors (e.g., avoiding wearing clothes that make them particularly aware of their body) rather than food-related behaviors (Grilo et al., 2005; Reas et al., 2005). Moreover, it should also be noted that all ED subgroups (e.g., AN, BN, BED, and OSFED) still scored significantly higher than controls on FOFM subscales. This indicates that patients with BED and OSFED still experience clinically relevant levels of eating-related anxiety, even if their fear profiles differ from those observed in AN and BN.

Regarding the longitudinal results observed in the present study, only a third of participants completed 4 months of treatment. Notably, the majority of patients who completed the midtreatment showed significantly longer illness duration, consistent with existing literature supporting that illness duration predicts greater treatment completion and lower dropout rates (Mannan et al., 2021). Furthermore, in our sample, the majority of treatment completers had a diagnosis of AN and showed higher baseline levels of eating-related anxiety compared to those who discontinued. In particular, elevated fear of food, both in terms of anxiety about eating and food avoidance behaviors, appeared to characterize treatment completers. This finding may indicate that patients with higher levels of eating-related anxiety are more motivated to remain engaged in treatment, possibly because such symptoms are perceived as particularly distressing and therefore drive treatment-seeking behavior. Conversely, patients with lower levels of fear of food might not experience the same urgency to adhere to treatment, which could partially explain their higher attrition rates. Although preliminary, this result highlights the potential clinical utility of assessing fear of food at intake, as it may inform risk of dropout and guide early interventions aimed at sustaining engagement in care.

In terms of improvements, significant reductions by mid-treatment were observed across all fear of food components, in line with previous studies observing FOFM changes following exposure therapy interventions (Butler et al., 2024; Levinson & Byrne, 2015). Consistent with the literature (Butler et al., 2024; Levinson & Byrne, 2015), in the present study anxiety about eating exhibited the greatest changes at mid-treatment, followed by feared concerns, with large effect sizes, while food avoidance behaviors demonstrated smaller changes. These results align with the initial goals of CBT protocols for EDs, which prioritize symptom reduction to build motivation and confidence in therapy (Don et al., 2019). The more modest reductions in food avoidance behaviors may reflect the entrenched nature of these behaviors in EDs. Food avoidance behaviors, often resistant to treatment, may persist beyond successful intervention and remain present even in remitted or recovered patients, representing residual symptoms (Tomba et al., 2019). This highlights the potential need for enhanced exposure exercises early in therapy, as exposure has been shown to effectively target avoidance behaviors (Linardon et al., 2016). Furthermore, these findings may be partially attributed to the small sample size in these preliminary analyses. Although preliminary, these findings suggest that administering the FOFM at the beginning of treatment and readministering it at midtreatment to detect changes in fear of food may help clinicians to develop more appropriate treatment planning tailored to the patients' specific needs over the course of therapy. This approach could be particularly valuable in cases where specific FOFM dimensions, such as behavioral avoidance, results in impairment.

The present findings also showed that early changes in fear of food are associated with early changes in ED specific and overall psychological changes beyond ED-specific symptoms. Specifically, anxiety about eating emerged as the most significant predictor across regression models, predicting both EDI-3 risk and EAT-total scores, as well as EDI-3 general psychological maladjustment scores. While previous studies showed that fear of food predicted ED-specific symptomatology in ED clinical populations (Levinson et al., 2017; Levinson & Byrne, 2015), this is the first study showing that changes in fear of food are associated with changes also in psychological changes beyond ED-specific symptoms and that these changes not only affect cognitive and behavioral aspects specific to ED symptomatology, but also general psychopathology. The role of fears in ED treatment planning, and specifically of anxiety about eating, has been observed in the literature regarding ED. A study using network analysis highlighted how a similar construct, food-related anxiety, would be a central symptom working as a bridge between several ED symp-

toms, such as food avoidance, adherence to food rules, and excessive exercise (Levinson & Williams, 2020). In line with this evidence, the novel finding of the present study can enrich the existing literature on fear of food in ED patients as it suggests that addressing this specific feature during treatment may have beneficial effects on the overall psychological functioning of ED patients.

Ultimately, while a small sub-group of underweight ED patients showed BMI restoration at mid-treatment, early changes in FOFM subscales were not found to predict early changes in BMI in this sub-group. This may suggest that reduction in fear of food may not necessarily accompany early weight restoration. Furthermore, it may also indicate that therapeutic progress in addressing fear of food and related avoidance behaviors may reflect early cognitive-emotional changes that require more than 12 sessions of CBT and 12 sessions NRC before being mirrored by substantial weight restoration. However, this result may also be due to the small sample size of the subgroup used for this analysis and its limited statistical power, and should therefore be interpreted with caution.

CLINICAL IMPLICATIONS AND FUTURE DIRECTIONS

These findings extend the understanding of the fear of food beyond eating-related symptoms, highlighting its broader impact on patients' psychological maladjustment and providing useful insights for the treatment of EDs, which should be optimized within a multidisciplinary framework that integrates CBT and nutritional rehabilitation (Crone et al., 2023).

Anxiety about eating—identified as the most significant predictor of improvements in both ED-specific and general psychopathology—should be prioritized in early interventions. Cognitive-behavioral interventions, particularly exposure-based techniques, can be useful in reducing food- and eating-related anxiety, thus fostering more comprehensive recovery outcomes (Butler et al., 2024; Levinson & Byrne, 2015). Additionally, given the higher levels of food-related anxiety and avoidance behaviors observed in patients with AN and BN, individualized interventions that directly address these fears may be especially beneficial for these groups. Importantly, patients with BED and OSFED also report elevated food-related anxiety compared to controls, with differences with AN and BN reflecting greater heterogeneity in their clinical presentations rather than the absence of such concerns. Therefore, interventions targeting food-related fears are likely relevant across all EDs as they likely represent a transdiagnostic feature (Levinson & Byrne, 2015), although clinicians should pay attention to tailoring these interventions accordingly to the specific fear profiles and clinical characteristics of each ED diagnosis

Importantly, taking into account patients' food-related fears alongside their nutritional needs—through structured, personalized meal plans—may support recovery by reducing cravings, enhancing satiety, and minimizing binge-purge cycles (Abbate-Daga et al., 2007; Garner & Garfinkel, 1979), consistent with APA Clinical Practice Guidelines (Crone et al., 2023). The persistence of food avoidance behaviors despite partial improvement in other symptoms further underscores how FOFM may serve as a clinically useful tool to identify residual areas of food-related anxiety that are not yet fully addressed by current treatment strategies. In this regard, the FOFM has the potential to guide and refine available exposure interventions by detecting persistent deficits in food-related anxiety. Given the heterogeneity and shared features of EDs, flexible, transdiagnostic treatment models are essential. To refine such approaches, further longitudinal research with larger samples is needed, especially studies employing network models to explore the role of fear of food as a potential bridge symptom.

LIMITATIONS

The reported findings should be interpreted in light of the study's limitations. The first is the absence of a formal interrater reliability coefficient (e.g., Cohen's kappa) for the ED diagnoses. Although each diagnosis was independently formulated by both a board-certified psychiatrist and a licensed clinical psychologist using the Structured Clinical Interview for DSM-5 (First et al., 2016) and discrepancies were resolved through diagnostic consensus, the lack of a quantitative estimate of diagnostic agreement may limit the assessment of diagnostic precision. Nevertheless, consensus-based diagnostic procedures conducted by experienced clinicians are commonly employed in clinical research and considered acceptable when standardized diagnostic criteria are applied (First et al., 2016). Another important limitation concerns the heterogeneity of the OSFED group, of which 42% received a diagnosis of atypical AN (AAN). While research has consistently shown that AAN patients present with levels of eating disorder psychopathology comparable to, or even exceeding, those observed in AN (Fitterman-Harris et al., 2024), in our study diagnoses were

established through structured clinical interviews and validated self-report questionnaires conducted at intake in the referral center. While we retained the original diagnostic categorizations as determined by clinicians, future studies with larger samples could aim to subgroup AAN together with AN, and low-frequency/short-duration BN together with BN, allowing for a more precise characterization of symptom patterns.

In addition, our sample lacks gender diversity, as only female participants were included. This limitation is common in ED research, given that the majority of individuals affected by these disorders are female. However, future studies should include mixed-gender samples to explore potential gender differences in fear of food. Moreover, the lack of racial and ethnic diversity in the sample—composed entirely of participants who self-identified as White/European—limits the generalizability of the findings to more diverse populations. Future research should include more ethnically diverse samples to better understand how fear of food may manifest across cultural contexts. Additionally, although our overall sample size met the requirements established by prior power analyses, fewer than half of the participants completed the mid-treatment assessment and were therefore included in the preliminary analyses of early changes in FOFM subscales and ED symptomatology. The study was characterized by a substantial attrition rate, with only 28% of participants completing the midtreatment assessment. This dropout is consistent with previous literature on ED populations, which reports attrition rates ranging widely from approximately 20% to over 70% in outpatient treatments (Fassino et al., 2009). Attrition in ED research is multifactorial and influenced by psychological distress, ambivalence toward treatment, avoidance behaviors, as well as logistical and organizational challenges. Although the longitudinal analyses were intended as preliminary and exploratory, the reduced sample size may limit the generalizability and representativeness of the findings. Furthermore, the heterogeneity of the sample limits the generalizability of these findings to specific ED diagnoses. Similarly, although BMI change is often considered a clinically relevant outcome, particularly for underweight and weight suppressed ED patients, it was not included as an outcome variable in all patients taking part in this study due to the diagnostic heterogeneity of the sample. Given that treatment goals regarding weight differ substantially across ED subtypes, BMI change would not provide a consistently interpretable measure of therapeutic progress in such a mixed ED sample. Still, a supplementary exploratory regression analysis was conducted within an underweight subgroup of patients to examine the predictive role of early changes in fear of food on early changes in BMI. Although no significant association emerged, this analysis was based on a small subgroup and was underpowered, limiting the interpretability of the results. Future research with larger samples and increased statistical power is needed to better clarify the temporal and causal relationships between fear of food reduction and weight restoration in underweight or weight suppressed ED patients.

In conclusion, this study reinforces the central role of fear of food as a transdiagnostic construct in EDs, carrying critical implications for treatment. By addressing core phobic components early in therapy, clinicians can enhance treatment efficacy, promote sustained recovery, and reduce the risk of residual symptoms persisting beyond initial therapeutic interventions.

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