Scientific Reports

https://doi.org/10.1038/s41598-026-36068-2

Article in Press

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Received: 11 July 2025

Accepted: 9 January 2026

Published online: 22 January 2026

Cite this article as: Kot E., Skimina E., Pietras T. *et al.* Dietary patterns and emotion dysregulation in borderline personality disorder and eating disorders as a shared mechanism underlying symptom severity. *Sci Rep* (2026). https://doi.org/10.1038/s41598-026-36068-2

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Dietary patterns and emotion dysregulation in borderline personality disorder and eating disorders as a shared mechanism underlying symptom severity

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Abstract

Borderline personality disorder (BPD) and eating disorders (EDs) are often comorbid and share a core feature of emotion dysregulation (EDys). While diet has been linked to mental health, its relationship with EDys and symptom severity in these groups remains understudied. This study investigated dietary intake in BPD, EDs, and their comorbidity, and examined whether EDys mediates the relationship between diet and symptom severity. Female inpatients with BPD (n = 40), ED (n = 22), and BPD with comorbid ED (BPD+ED; n = 37), along with healthy controls (HCs; n = 37) completed Food Frequency Questionnaire (FFQ-6), Emotion Dysregulation Scale (EDS), and clinical self-report measures. Dietary patterns differed between groups. Clinical groups consumed sources of omega-3 polyunsaturated fatty acids and Mediterranean diet (MD) foods less frequently than HCs. EDys fully mediated the link between dietary patterns and symptom severity in most models. The mediation was partial when omega-3 intake predicted ED severity in the ED group.

Women with BPD and BPD+ED showed poorer diet quality, especially regarding omega-3 and MD-aligned foods. EDys mediated the association between low-quality diet and symptom severity, suggesting a

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transdiagnostic mechanism. Nutritional interventions may positively influence emotion regulation, thereby reducing the risk of developing and maintaining symptoms of BPD and EDs.

Keywords*:* borderline personality disorder, eating disorders, diet, food frequency, omega-3 fatty acids, Mediterranean diet

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1. Introduction

Borderline personality disorder (BPD) is a severe psychiatric condition affecting approximately 1.8% of the global population[1]. Characterized by emotional dysregulation (EDys), impulsivity, unstable interpersonal relationships, and a distorted self-image, BPD frequently co-occurs with other mental disorders, such as mood disorders, anxiety disorders, substance use disorders, and eating disorders (EDs)[2]. BPD is frequently associated with nonsuicidal self-injury (NSSI) and a significantly elevated risk of suicide[3].

1.1. Nutrition and Physical Health in BPD: Beyond Psychopathology

The implications of BPD extend beyond mental well-being, influencing overall physical health. Individuals with BPD face a greater risk of developing non-communicable chronic diseases (NCDs), including cardiovascular conditions, type 2 diabetes, metabolic syndrome, and gastrointestinal disorders[4–6]. Furthermore, longitudinal analyses show that individuals in long-term remission from BPD symptoms engage in fewer unhealthy behaviors and have a lower incidence of NCDs compared to those with BPD[7]. Chronic stress and lifestyle-related factors are thought to contribute to this increased susceptibility[5,8], alongside maladaptive emotion regulation patterns that may disrupt physiological systems such as the hypothalamic–pituitary–adrenal (HPA) axis[9].

Lifestyle behaviors, including physical activity, sleep, smoking, and dietary habits, are increasingly recognized as crucial in both mental[10] and physical health[11]. Poor diet quality has been linked to depression,

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schizophrenia, and anxiety[12,13]. Conversely, adherence to dietary patterns associated with positive health outcomes, like the Mediterranean diet (MD), which emphasizes fruits, vegetables, fish, whole grains, and beneficial fats, has shown promise in reducing the risk of mental disorders[14] and improving their symptoms[15]. The MD offers anti-inflammatory properties and supports gut health. It also reduces the intake of saturated fatty acids (SFAs) and helps optimize the omega-6 to omega-3 polyunsaturated fatty acids (PUFAs) ratio. These features are associated with reduced risks of type 2 diabetes, metabolic disorders, and certain cancers[16,17], as well as improved cardiometabolic markers, including a reduction in cardiovascular events by approximately 30%[14,18,19].

Although research on the relationship between dietary patterns and personality disorders is limited, existing evidence suggests that neuroticism and alexithymia are linked to poor dietary habits, including low consumption of fruits and vegetables and high intake of sweets and SFA-rich foods[20]. There is a lack of comprehensive studies on how specific dietary components affect BPD symptoms. However, nutrient-based interventions, such as omega-3 supplementation, have shown potential in alleviating BPD symptoms, particularly impulsivity and EDys[21,22]. Additionally, supplementing with EPA, an omega-3 fatty acid, has been found to reduce aggression in BPD patients[23].

Interesting data emerge from the study comparing vitamin D levels in individuals who attempted suicide to those with depression who did not attempt suicide and a healthy control group. Individuals with a history of

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suicide attempts showed significantly lower vitamin D levels compared to the other groups. Considering vitamin D's role in the nervous and skeletal systems, and reports linking BPD with reduced bone density[24], a hypothesis could be proposed regarding the relationship between suicidal behaviors in this group and their nutritional status. In another study, higher levels of visceral fat were observed in patients with major depression and co-occurring BPD compared to those with major depression without BPD[25]. Additionally, a 10-year longitudinal study found that cumulative BMI in BPD patients was associated with a diagnosis of two or more NCDs[26].

1.2. Intersecting Psychopathologies: BPD, Eating Disorders, and the Role of Emotion Dysregulation

Empirical evidence suggests a relationship between BPD and obesity[26,27]; however, a systematic review by Gerlach et al.[28] highlights that this association is not unequivocal and may be influenced by co-occurring binge eating disorder (BED). Notably, EDs are highly prevalent in BPD, with up to 61% of hospitalized patients meeting diagnostic criteria. BPD frequently co-occurs with bulimia nervosa (28%; BN), the binge-purge subtype of anorexia nervosa (25%; AN), BED (12%), and restrictive AN (10%)[29–31].

The significant symptom overlap and frequent co-occurrence of BPD with EDs suggest shared underlying mechanisms, leading researchers to adopt a transdiagnostic perspective. According to the biosocial model of BPD, EDys is central to the onset and maintenance of BPD symptoms, often manifesting in maladaptive behaviors like non-suicidal self-injury

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(NSSI), risky behaviors, and disordered eating[32,33]. Furthermore, research suggests that BPD may mediate the relationship between traumatic experiences and the development of EDs, with body image disturbances and NSSI increasing the likelihood of EDs onset[34].

EDys is also an important transdiagnostic characteristic in EDs[35], involving deficits such as heightened emotional intensity, lower acceptance of emotions, reduced emotional awareness and clarity, and increased reliance on dysfunctional emotion regulation strategies[36,37]. Some authors consider EDys a key factor maintaining eating pathology[38]. Interestingly, behaviors like binge eating or self-induced vomiting may indicate impulsivity in BPD, while not being sufficient for diagnosing a co-occurring EDs[39]. Among patients with AN and BN, higher levels of EDys are associated with greater severity of ED symptoms[40]. In patients with BPD, abnormal eating behaviors may serve as emotion regulation strategies and are potentially linked to EDys, warranting further investigation in scientific studies.

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1.3. Aims and Hypotheses

Given the established association between dietary pattern and the risk of NCDs[11], the elevated risk of NCDs in patients with BPD[4–6], and the lack of research on dietary pattern in this population, the aim of this study was to evaluate dietary intake in individuals with BPD. In light of evidence linking the MD and omega-3 PUFAs to a reduced risk of mental disorders and improvements in psychiatric symptom severity[19], we also assessed the intake frequency of a) foods frequently consumed in MD, b) sources of omega-3 PUFAs, and the intake of c) foods associated with

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unhealthy dietary patterns. Due to the prevalence of abnormal eating behaviors in BPD and the high comorbidity of EDs in this population, dietary patterns were compared not only to healthy controls (HCs) but also to clinical control (CC) groups: BPD patients with comorbid EDs and ED patients without BPD. We hypothesized that individuals with BPD would exhibit less healthy dietary pattern compared to HCs and distinct dietary pattern relative to CC. Specifically, we expected the BPD group to show higher consumption of foods rich in simple carbohydrates and saturated fats, and lower intake of foods typical of the MD, including omega-3–rich products, compared to HCs. Moreover, we hypothesized that the CC groups would display generally lower intake of both MD and omega-3–rich foods, as well as high-fat/high-sugar foods, relative to HCs, and lower consumption of foods deviating from the MD pattern compared to the BPD group.

The role of EDys in both BPD and ED psychopathology was outlined, along with evidence supporting the efficacy of omega-3 PUFAs supplementation in improving symptoms, including EDys, in BPD patients[22], and the protective role of the MD against ED development[41,42]. Considering that EDys is recognized as a contributing factor to the onset and maintenance of BPD and ED symptoms[32–34], a secondary aim was to investigate the mediating role of EDys in the relationship between dietary pattern and the severity of clinical symptoms in BPD and EDs. We hypothesized that EDys would mediate the association between dietary pattern and symptom severity in the following way: less frequent consumption of products typical for MD and of foods rich in omega-3

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PUFAs as well as more frequent consumption of foods deviating from the MD pattern would be related to greater EDys, which in turn would be associated with greater severity of BPD and EDs symptoms.

Additionally, we examined whether BPD and ED diagnoses moderate the relationships among dietary pattern, EDys, and disorder-specific symptoms. Specifically, we expected that the relationship between dietary pattern and EDys would be similar across all participants, whereas the effects on symptom severity would emerge primarily within the relevant clinical groups. That is, dietary pattern and EDys would predict BPD symptoms primarily in participants with a BPD diagnosis, and ED symptoms primarily in participants with an ED diagnosis. Mediation hypotheses are illustrated in Figure 1 and moderation hypotheses are illustrated in Figure 2.

[Insert Figure 1 here]

[Insert Figure 2 here]

2. Methods

2.1. Participants

Due to the predominance of women among individuals diagnosed with BPD (75%) and ED (90% in AN and BN)[2], only female participants aged 18–50 of Polish origin were recruited. The final sample consisted of 136 women, following the exclusion of five individuals due to increased symptom severity (in the HC group) or incomplete data (in all groups). Participants were divided into four groups: women diagnosed with BPD

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according to ICD-10 and DSM-5 (n = 40), women with EDs, including AN, BN, and BED (n = 22), women with comorbid BPD and ED (n = 37), and HC women (n = 37). Patients were recruited from the inpatient unit of the Institute of Psychiatry and Neurology in Warsaw, Poland, while HC participants were primarily university students matched for age.

Inclusion criteria required written informed consent, at least primary education, normal intellectual functioning, Polish as a native language, and adequate verbal communication skills. Exclusion criteria included neurodevelopmental and neurological disorders, brain injury, substance dependence, severe metabolic or diet-related illnesses, and psychiatric conditions such as schizophrenia spectrum disorders, mania, or psychosis. HC participants were additionally screened for BMI outside the 18.5–25 kg/m² range and any history of psychiatric disorders. A detailed characterization of the study groups is presented in Table 1.

[Insert Table 1 here]

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2.2. Procedure

Study approval was obtained from the Bioethical Committee at the Institute of Psychiatry and Neurology in Warsaw, Poland (no. 10/2020). Subjects read the study description and signed the informed consent sheet prepared in concordance with the current version of the Declaration of Helsinki.

This study is part of a larger project that also assessed sleep and physical activity over seven days, though these results are not included here. The focus of this analysis is on dietary patterns, EDys, and clinical symptoms. Clinical interviews were conducted using the SCID-5-PD in

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clinical groups and SCID-5-SPQ[43] in the HC group to exclude personality disorders. Additionally, selected health indicators were assessed during the study (see Table 1), including body mass index (BMI) and waist-hip ratio (WHR). Blood pressure, resting heart rate, and pulse pressure were calculated as the average of two measurements. Participants in clinical groups were examined within the first three weeks of hospitalization. All participants completed a battery of measures described below.

Although the study design and hypotheses were formulated prior to data collection, they were not preregistered.

2.3. Measures

2.3.1. Dietary intake

Dietary intake was assessed using the Food Frequency Questionnaire (FFQ-6)[44], which evaluates the consumption frequency of 62 food groups over the past 12 months. Responses are recorded on an ordinal scale and converted into a semi-quantitative scale based on daily intake frequency (e.g., *never* = 0 times/day, *several times per month* = 0.1 times/day, *daily* = 1 time/day). The FFQ-6 has demonstrated internal validity through test-retest reliability and has been used to identify dietary patterns in previous studies[44,45]. At the time of data collection, the 62-item version of the FFQ-6 was the most widely used in Poland. However, a revised 72-item version has since been introduced[46], offering expanded food group coverage for future studies.

In accordance with the recommendations of the questionnaire's authors, we aggregated the 62 food groups into 25 broader categories and calculated mean daily consumption frequencies[47]. Additionally, for

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the purpose of this study, we computed dietary indices relevant to mental health, including: Omega-3 fatty acid intake index, based on the summed frequency of consumption of vegetable oils, nuts, seeds, and fish. MD adherence index, calculated separately for a) foods recommended in MD (e.g., dairy, whole grains, fish, fruits, vegetables) and b) those advised to be consumed in limited amounts (e.g., refined grains, processed meats, sweets).

2.3.2. Emotion dysregulation

EDys was assessed using the Difficulties in Emotion Regulation Scale (DERS)[48,49], a 36-item self-report questionnaire measuring difficulties in regulating negative emotions. The scale comprises six subscales: Lack of Emotional Awareness, Lack of Emotional Clarity, Impulse Control Difficulties, Difficulties Engaging in Goal-Directed Behavior, Non-Acceptance of Emotional Responses, and Limited Access to Emotion Regulation Strategies.

Participants responded using a 5-point Likert scale, with higher scores indicating greater difficulties. Both the original and Polish versions of the DERS have demonstrated good reliability and validity. In this study, the total score and subscale scores were used to assess emotion regulation difficulties. Internal consistency in our sample was satisfactory (Cronbach's α: BPD = 0.81, ED = 0.75, BPD+ED = 0.83, HC = 0.86).

2.3.3. Severity of Borderline Personality Disorder Symptoms

The Borderline Personality Disorder Checklist (BPD Checklist)[50] is a self-report tool assessing BPD symptom severity over the past month.

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Unlike the SCID-5-PD, which evaluates lifetime symptom presence, the BPD Checklist measures current symptom intensity.

The questionnaire consists of 47 items reflecting DSM-IV/DSM-5 BPD criteria[50,51]. Responses are rated on a 5-point Likert scale (from *not at all* to *very much*), with scores ranging from 47 to 235, where higher values indicate greater symptom severity. The scale provides both an overall symptom severity score and scores across nine subscales: Abandonment, Interpersonal Relationships, Identity, Impulsivity, Self-mutilation/Parasuicide, Mood Instability, Emptiness, Anger, and Dissociation.

Recent validation of the Polish version confirmed its high reliability[51], though normative data for the Polish population are not yet available. The original validation in American clinical and non-clinical samples suggested a score ≥100 indicates significant BPD symptoms, while ≤67 reflects remission [52]. The internal consistency in this study was high (Cronbach's α: BPD = 0.94; BPD+ED = 0.92; ED = 0.93; HC = 0.97).

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2.2.4. Severity of Eating Disorder Symptoms

The Eating Attitudes Test (EAT-26)[53,54] is a self-report questionnaire assessing disordered eating behaviors. It includes four subscales: Social Pressure, Dietary Restraint, Bulimia, and Food Preoccupation. EAT-26 is used as a screening tool to identify individuals at risk of developing EDs and as a research measure of ED symptom severity.

The questionnaire consists of two parts (A and B), with Part A comprising 26 items measuring ED symptoms. Responses are given on a

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6-point Likert scale (*never–always*), with scores ranging from 0 to 78. Higher scores indicate greater severity of disordered eating behaviors. A total score of ≥20 suggests clinically relevant ED symptoms. The Polish version has demonstrated good psychometric properties (Cronbach's α = 0.80 in a nonclinical sample)[54]. In this study, high reliability was confirmed (BPD: α = 0.90; ED: α = 0.88; BPD+ED: α = 0.92; HC: α = 0.86).

2.2.5. Severity of Anxiety and Depression Symptoms

The Hospital Anxiety and Depression Scale (HADS)[55,56,57] is a 14-item self-report questionnaire designed to measure psychological distress. It consists of two 7-item subscales: Anxiety (HADS-A) and Depression (HADS-D). Originally developed for rapid mental health assessment in hospital settings, HADS is also widely used as a screening tool for psychological distress in the general population and in research assessing anxiety and depression severity in clinical populations.

Participants rated their experiences over the past week using a 4-point Likert scale (0–3), with total scores ranging from 0 to 21 for each subscale. Higher scores indicate greater symptom severity. The Polish version of HADS has demonstrated good reliability (Cronbach's α = 0.81 for HADS-A and 0.80 for HADS-D)[57]. In the present study, reliability was confirmed across groups (BPD: HADS-A α = 0.82, HADS-D α = 0.84; BPD+ED: HADS-A α = 0.88, HADS-D α = 0.83; ED: HADS-A α = 0.82, HADS-D α = 0.88; HC: HADS-A α = 0.70, HADS-D α = 0.81).

2.3. Statistical Analyses

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Statistical analyses were carried out with IBM SPSS Statistics 29 software[53]. Before performing between-group comparisons, assumptions for parametric methods were checked. Chi-square tests (χ^2) were used to verify group equivalence, with no significant differences in group sizes ($\chi^2(3) = 5.82$, $p = .120$). Normality of data distributions was assessed using skewness and kurtosis values, with values exceeding 2.00 indicating non-normal distribution, prompting the use of the Kruskal-Wallis test. For normally distributed data, Levene's test assessed homogeneity of variances. Significant results ($p < .05$) led to Kruskal-Wallis testing; otherwise, ANOVA was applied. Post-hoc analyses were conducted using Bonferroni correction for both ANOVA and Kruskal-Wallis tests. Effect sizes were calculated using eta squared (η^2), with thresholds for small (.01–.05), medium (.06–.13), and large (≥.14) effects [54].

Spearman’s rank correlation was used to examine relationships between variables due to non-normal data distributions. Correlations were calculated on the whole sample to verify assumptions for mediation analysis regarding relationships between variables included in the model[55]. Effect sizes were interpreted as follows: rs = 0.10–0.29 (weak), rs = 0.30–0.49 (moderate), rs ≥ 0.50 (strong)[56].

Mediation and moderated mediation analyses were performed using the PROCESS macro[57], with models 4 and 15 for testing direct, indirect, and total effects. Bootstrapping (10,000 samples) was used to generate 95% confidence intervals (CI) for indirect effects. Mediation effects were considered significant if the confidence intervals did not contain zero.

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Moderated mediation was assessed by testing interaction effects between the moderator and mediator, and between the moderator and independent variable.

A post-hoc sensitivity power analysis conducted using the 'WebPower' package[58] in R (α = .05, power = .80) indicated that the minimum detectable effect sizes were η^2 = .08 and rho = .24. The Monte Carlo Power Analysis for Indirect Effects shiny app by Schoemann et al.[59] with 1000 replications and 20,000 draws per replication, indicated power between .89 and .98 for indirect effects in this study. The InteractionPoweR shiny app by Finsaas et al.[64] based on R package by Baranger et al.[65] with 1000 simulations indicated power .66 for the interaction effect found in this study.

3. Results

3.1. Between-Group Differences in Clinical Symptoms Severity and Dietary Patterns

The between-group differences in symptoms severity analyzed in ANOVA and Kruskal-Wallis test are presented in Table 2. HCs had significantly lower levels of all symptoms and EDys than clinical groups. The ED group differed significantly from BPD in BPD total score and anger but not in impulsivity. However, the ED group revealed lower level of impulsivity than BPD+ED. CCs had higher levels of ED symptoms than BPD group. Clinical groups did not differ significantly in depression and anxiety. ED group had lower level of EDys than BPD+ED.

[Insert Table 2 here]

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The between-group differences in dietary patterns are presented in Table 3. BPD group consumed vegetable fats, fruits, dried and processed legumes, and nuts and seeds significantly less frequently, as well as sugar-sweetened and energy drinks significantly more frequently than HCs. Patients with EDs and those with BPD+ED diagnosis consumed cheese, vegetable fats, red meat and game, and alcohol, significantly less frequently than HCs. What is more, patients with BPD+ED diagnosis consumed eggs and egg-based dishes, nuts and seeds, white meat, and fish less frequently than HCs. Patients with BPD consumed butter and cream more often than patients with BPD+ED. BPD and HC groups consumed alcohol more frequently compared to patients with EDs. HCs and CC groups consumed sources of omega-3 PUFAs more often than patients with BPD. Moreover, HCs consumed products typical for MD more frequently than BPD and BPD+ED groups. Effects were medium to large, ranging from .06 to .16.

[Insert Table 3 here]

3.2. Correlations Between Dietary Patterns, Emotion Dysregulation, and Clinical Symptoms

The results of Spearman's rank correlations are presented in Supplementary Table S1. Intake of both Omega-3 PUFAs and products typical for MD correlated significantly and negatively with EDys and all clinical symptoms, including BPD (main score, impulsivity, and anger), EDs symptoms, as well as anxiety and depression. Consumption frequency of products that should be consumed only occasionally in the MD (Anti-MD) correlated significantly and positively with EDys and anger

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(a symptom of BPD). All effects were small to moderate, ranging from .19 to .40. Because the index of anti-MD diet did not correlate significantly with clinical symptoms, this variable was not used as a predictor in mediation models presented in the next section.

3.3. Mediation and Moderated Mediation Models

We tested eight models in total: four mediation models (see Figure 1) and four moderated mediation models (see Figure 2).

Table 4 presents the results of models examining the severity of BPD symptoms as predicted by Omega-3 PUFAs (Model A) and adherence to the MD (Model C), with EDys as a mediator. Models B and D tested whether the indirect and direct effects in Models A and C, respectively, were moderated by BPD diagnosis.

[Insert Table 4 here]

In Model A, Omega-3 PUFAs significantly predicted EDys: $R^2 = .11$, $F(1, 134) = 16.92$, $p < .001$. BPD symptom severity was significantly predicted by Omega-3 PUFAs together with EDys: $R^2 = .57$, $F(2, 133) = 88.19$, $p < .001$. The indirect effect through EDys was significant and the direct effect was not, indicating full mediation. Model B did not reveal any significant interaction.

In Model C, MD significantly predicted EDys: $R^2 = .07$, $F(1, 134) = 10.61$, $p < .001$. MD together with EDys significantly predicted BPD symptom severity: $R^2 = .57$, $F(2, 133) = 89.85$, $p < .001$. Again, the indirect effect was significant and the direct effect was not, indicating full mediation. Model D did not reveal any significant interaction.

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Table 5 presents the results of models examining the severity of EDs symptoms as predicted by Omega-3 PUFAs consumption (Model E) and adherence to the MD (Model G), with EDys as a mediator. Models F and H tested whether the indirect and direct effects in Models E and G, respectively, were moderated by EDs diagnosis.

[Insert Table 5 here]

Omega-3 PUFAs together with EDys significantly predicted the severity of EDs symptoms: $R^2 = .28$, $F(2, 132) = 26.23$, $p < .001$. The indirect effect in Model E was significant, while the direct effect was not, indicating full mediation. Model F revealed a significant interaction between Omega-3 PUFAs consumption and EDs diagnosis. Comparison of conditional direct effects showed that the direct effect of Omega-3 PUFAs consumption on EDs severity was significant among patients with an ED diagnosis, but not among women without the diagnosis. Therefore, for individuals with an ED diagnosis, the observed mediation was only partial.

In Model G, the severity of EDs symptoms was significantly predicted by MD together with EDys: $R^2 = .27$, $F(2, 132) = 24.29$, $p < .001$. The indirect effect was significant, while the direct effect was not, indicating full mediation. Model H did not reveal any significant interaction.

4. Discussion

4.1. Between-Group Differences in Dietary Pattern

The findings partially confirmed less healthy dietary pattern among patients with BPD compared to HC, and a distinct pattern compared to CC. More frequent consumption of sugar-sweetened beverages and

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energy drinks—sources of simple carbohydrates—was observed in the BPD group relative to HCs. These products have been linked to a higher risk of type 2 diabetes, obesity, and cardiometabolic diseases[60–62], which may have clinical relevance given the higher values of nutritional status indicators, resting heart rate, and pulse pressure observed in our BPD sample. In light of previous evidence suggesting an increased risk of metabolic syndrome and cardiovascular diseases in this population[5,8], the present results indicate that sweetened and energy drinks may represent a potential dietary risk factor. However, longitudinal studies assessing dietary intake and health outcomes in individuals with BPD are warranted.

Patients with BPD showed less frequent intake of fruits, legumes, nuts, and seeds compared to HCs, suggesting a reduced dietary fiber supply from these sources. No differences were found in the intake of specific fiber categories between patient groups. Previous studies have linked high fiber intake to a lower risk of depression[63], which may be particularly relevant given the high severity of depressive symptoms observed across all clinical groups in our study. The underlying mechanisms may involve the gut microbiota and oxidative stress[63,64], and our findings indirectly align with reports of gut microbiota disturbances in BPD and EDs[65–67]. Considering the established associations between dietary fiber intake and reduced risk of type 2 diabetes, cardiovascular disease, obesity, and certain cancers[68–72], as well as the increased prevalence of these conditions in individuals with BPD[4,8,27,73], the

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potential protective role of fiber intake in this group warrants further investigation.

A higher frequency of butter and cream consumption was observed in patients with BPD compared to those with BPD+ED, with no significant differences in the intake of other SFA-rich products between the BPD, HC, and CC groups. This may suggest a relatively higher intake of SFAs from these specific sources in individuals with BPD without comorbid ED, rather than a generally higher intake of SFAs. One possible explanation could be the dietary restrictions typically associated with EDs, which often involve the avoidance of high-calorie foods such as butter and cream[74]. Given that BMI and WHR values were higher in the BPD group, future studies should investigate whether this dietary difference contributes to differences in nutritional status, although no conclusions can be drawn based on the current findings alone.

Patients with BPD reported a lower frequency of consumption of foods rich in omega-3 PUFAs compared to HCs, as well as a different intake pattern relative to CC. This finding aligns with previous evidence supporting the efficacy of omega-3 PUFA supplementation in reducing BPD symptoms such as impulsivity and aggression[21,22,75], and with studies indicating a link between low dietary omega-3 intake and aggressive behavior in children and adolescents—a potential risk factor for the development of BPD[76,77]. Considering the critical role of omega-3 PUFAs in the structure and functioning of the central nervous system, particularly in emotional regulation[78,79], these results highlight the need

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for further research into the relationship between omega-3 intake and emotional functioning in individuals with BPD.

Patients with BPD and BPD+ED showed a lower frequency of consumption of foods typical for the MD compared to HC, and a different consumption pattern compared to CC. Although direct studies on diet in BPD are lacking, this finding aligns with reports linking adherence to the MD with a lower risk of mental disorders and reduced symptom severity[14,80]. Moreover, greater efficacy of the MD has been confirmed in improving cardiovascular risk factors such as blood pressure, insulin sensitivity, and lipid profile, as well as reducing oxidative stress and inflammation[81], which play roles in the pathogenesis of mental disorders[82,83]. Elevated markers of inflammation and oxidative stress have been observed in patients with BPD[24,84], and antioxidant supplementation shows benefits in symptom reduction[22]. These results highlight the need for further research on the impact of diet, especially the MD, on the course of BPD.

Patients with BPD did not differ from HC and CC in the frequency of consumption of foods rarely consumed in the MD (potentially unhealthy). This finding suggests that, in individuals with BPD, insufficient intake of beneficial nutrients may be a more significant issue than excessive consumption of components discouraged in the diet.

4.2. Associations Between Dietary Patterns, Emotion Dysregulation, and BPD Symptoms

The identified association between the frequency of consuming foods typical of the MD and products rich in omega-3 PUFAs and the severity of

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BPD symptoms is indirectly supported by findings indicating the effectiveness of omega-3 PUFAs supplementation in reducing symptoms of this disorder. Furthermore, the observed link between frequent intake of omega-3-rich foods and lower severity of BPD symptoms aligns with the results of a meta-analysis on the impact of omega-3 supplementation on functioning in individuals with BPD. These studies have shown that omega-3-based interventions may particularly reduce EDys and impulsivity[22].

Consistently with these findings, mediation analysis demonstrated that EDys fully explained the relationship between the frequency of consumption of MD-typical foods and omega-3 sources and the severity of BPD symptoms. This suggests that a health-promoting dietary pattern may be associated with lower BPD symptom severity primarily through the reduction of difficulties in emotion regulation. This assumption is supported by studies showing the beneficial effects of the MD on nervous system functioning—both in cognitive and emotional domains—as well as its association with a lower risk of developing mental disorders[85]. In light of the key role of EDys in the development and maintenance of BPD symptoms[33], it is plausible that nutrition supporting psychological functioning may influence the mechanisms underlying BPD symptomatology. However, these findings do not allow for causal inference. To verify this hypothesis, randomized controlled trials are needed to evaluate the effects of MD-based dietary interventions on emotional functioning and clinical symptoms in individuals with BPD.

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Moderated mediation analysis did not reveal a significant moderating effect of BPD diagnosis—neither in the relationship between diet and BPD symptom severity nor in the association between EDys and BPD symptoms. These results suggest that the identified mediation mechanism operates similarly regardless of BPD diagnosis. In other words, a health-promoting dietary pattern may be linked to lower levels of BPD-related traits both in clinical and non-clinical populations, with EDys acting as a potential mediator. In reference to the biosocial theory of BPD development[32,33], these findings are consistent with a psychopathological model in which maladaptive dietary patterns—characterized by a recurrent behavioral pattern inconsistent with the MD and poor in omega-3 sources—may contribute to increased difficulties in emotion regulation, which in turn underlie BPD symptoms. As suggested by the moderated mediation results, such a mechanism may be relevant not only among individuals with a BPD diagnosis—potentially sustaining the disorder—but also among those without the diagnosis, contributing to the development of BPD-related difficulties.

4.3. Associations Between Dietary Patterns, Emotion Dysregulation, and ED Symptoms

A relationship was found between low frequency of consumption of foods typical of the MD and sources of omega-3 PUFAs and higher severity of ED symptoms. The direction of these associations aligns with current knowledge on ED psychopathology, which includes both food restriction and episodes of loss of control over eating[86], as well as with

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studies indicating a protective role of adherence to the MD against the development of ED symptoms[42].

Mediation analysis revealed that EDys fully mediated the relationship between the frequency of consuming MD-typical foods and omega-3-rich products and the severity of ED symptoms. While previous studies have reported omega-3 PUFA deficiencies in individuals with ED[87,88], findings regarding the effectiveness of supplementation in improving symptoms remain inconsistent[89,90]. A meta-analysis by Satogami et al.[97] associated omega-3 supplementation with benefits in weight normalization but found no significant effects on ED or mood symptoms. The present results suggest that the relationship between omega-3 intake and ED symptoms may be indirect and operate through reduced difficulties in emotion regulation, consistent with earlier findings[92].

One possible explanation is that diets lacking anti-inflammatory and antioxidant nutrients may adversely affect central nervous system functioning, leading to greater EDys and, consequently, more severe ED symptoms. This mechanism is consistent with previous research on MD adherence, and theories of EDys highlight the role of oxidative stress and inflammation in its development[93]. However, confirmation of this model requires studies including biomarkers of inflammation and oxidative stress.

In the moderated mediation analysis, no significant moderating effect of ED diagnosis was observed on the relationship between MD food consumption and ED symptoms or between EDys and ED symptoms. This

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suggests that the mediating mechanism operates similarly regardless of ED diagnosis. A different pattern emerged for omega-3 PUFA intake, where ED diagnosis significantly moderated the link between dietary intake and ED symptoms. The direct association was present only in the clinical group, suggesting that in individuals without ED, omega-3 consumption was a predictor of ED symptom severity only indirectly through EDys. Among those diagnosed with ED, however, omega-3 intake may relate to symptom severity both directly and indirectly through its effect on emotion regulation.

The observed interaction suggests a specific role of ED-related pathophysiological mechanisms in the association between omega-3 intake and ED symptoms, differentiating this clinical group from individuals without a diagnosis. Both EDys and EDs symptoms have been found to be linked to alterations in oxidative stress and inflammatory markers (independently of ED and BPD diagnoses), but there also appears to be a ED-specific alteration in inflammation [94]. Thus, it may explain a disorder-specific link between omega-3 acids consumption (which has anti-inflammatory and antioxidant properties), EDys and EDs in the ED group, but not HC. Additionally, the distorted food perception and low fat preference in the diet among ED patients (including anorexia nervosa) may contribute to a declaration of low PUFAs consumption in this group[95]. Those hypotheses require further testing in experimental studies with larger clinical samples. The use of complex, multifactorial statistical models may also help determine whether a co-occurring BPD

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diagnosis influences the pattern of associations between omega-3 intake, EDys, and ED symptomatology.

4.4. Limitations

The results concerning dietary patterns should be interpreted with caution. As the study included hospitalized patients, their diet during participation was based on hospital-provided meals. Thus, dietary intake was assessed retrospectively and subjectively, without prospective methods such as food diaries or weighed food records, which could offer more precise nutrient intake estimates and comparisons with population norms. Nevertheless, the FFQ used is a widely accepted tool for nutritional studies requiring simple and time-efficient methods[96].

Additionally, sodium intake—an important cardiovascular risk factor[97]—was not assessed. Another limitation stems from the heterogeneity of the ED group. While AN, BN, and BED share transdiagnostic features[98], differences in nutritional status and symptom profiles likely influence dietary habits across ED types. Therefore, comparisons involving the CC groups should be interpreted with caution and refer to EDs as a whole rather than specific diagnoses[74].

Given the cross-sectional nature of our study, causal direction cannot be determined. Although our models assume a pathway from dietary patterns to symptom severity via EDys, alternative models remain plausible. For instance, symptom severity may influence EDys, which in turn could shape dietary patterns in patients. This reversed pathway is consistent with existing literature indicating that symptoms of mental disorders can contribute to maladaptive health behaviors, including poor

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dietary choices[10]. While our proposed models are grounded in theoretical frameworks such as the biosocial model of BPD[32], future research employing longitudinal or experimental designs (e.g., dietary interventions) is necessary to test these pathways and evaluate alternative explanations.

Importantly, some effects found in this study did not had sufficient power, including group differences with $\eta^2 < .08$, rho correlations < .24 and the interaction effect. Hence, these findings should be interpreted with caution. While a post hoc power analysis was conducted, we acknowledge that a priori power calculations are preferred; future studies should replicate these findings in larger samples based on a priori power analysis.

Also, this is a single-centre, observational study with non-random sampling, on a relatively small group. Thus, no conclusions on any causal relationships can be drawn and the generalizability of the findings is limited.

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4.5. Future directions

To date, no studies have been published on dietary patterns among patients with BPD, making the present findings an important starting point for further investigation. Confirmation of these results in future studies—ideally using prospective methods—is warranted. Although retrospective assessment has limitations, it provides a valuable source of data, as demonstrated in studies involving oncology patients and general population samples in the context of anxiety and depression risk[99,100].

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Prospective research, in turn, constitutes a cornerstone of evidence-based nutrition[101].

4.6. Conclusions

Patients with BPD are characterized by a less healthy dietary pattern compared to HCs, involving more frequent consumption of selected dietary sources of simple carbohydrates as well as less frequent intake of selected sources of dietary fiber, omega-3 PUFAs, and foods typical of the MD. Compared to CC, patients with BPD do not differ significantly in the consumption frequency of most of these selected food groups, except for a higher intake of SFA-rich butter and cream and lower intake of omega-3 PUFAs sources.

Mediation analysis showed that EDys mediates the relationship between dietary pattern and the severity of BPD symptoms and ED symptoms, consistent with the transdiagnostic understanding of EDys in these conditions. Moreover, these associations were observed both in clinical and HC groups, suggesting that these mechanisms may operate independently of psychiatric diagnosis.

Direct associations between dietary pattern and the severity of ED symptoms were found only in individuals diagnosed with ED, which may be due to the fact that non-normative eating behaviors constitute core symptoms of these disorders.

Acknowledgments. We are deeply grateful to all participants who took part in this study. We also thank the staff of the Department of Neuroses,

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Personality Disorders, and Eating Disorders, Institute of Psychiatry and Neurology, for their support during data collection.

Funding. The publication was funded by the National Institute of Geriatrics, Rheumatology and Rehabilitation, the Medical University of Lodz, the SWPS University Research Development Fund, and the Warsaw University of Life Sciences.

Author Contributions. EK contributed to the conception and design of the study, data curation, methodology, and project administration, and drafted the manuscript. EK and ES conducted the formal analysis and investigation. ES, LM, JGO, and TP supported the conceptualization and contributed to writing, review, and editing. All authors approved the final version of the manuscript.

Data Availability Statement. The datasets used and analyzed during the current study are available from the corresponding author upon request.

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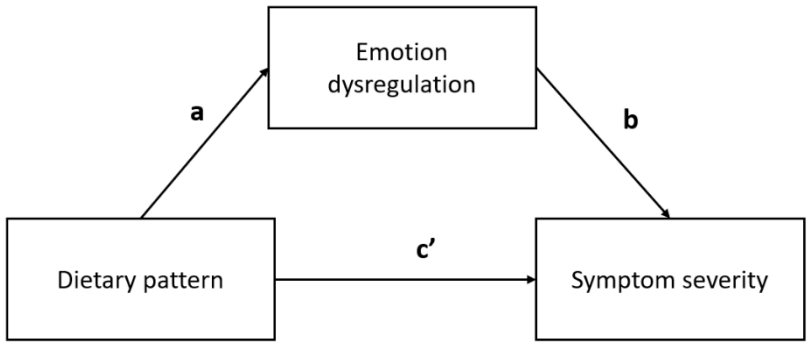
Figure 1

Mediation Models Tested in This Study



Note. Dietary pattern meant Mediterranean diet or sources of omega-3 PUFAs. Emotion dysregulation meant the general score of the Difficulties in Emotion Regulation Scale. Symptom severity meant severity of borderline personality disorder symptoms or severity of eating disorders symptoms.

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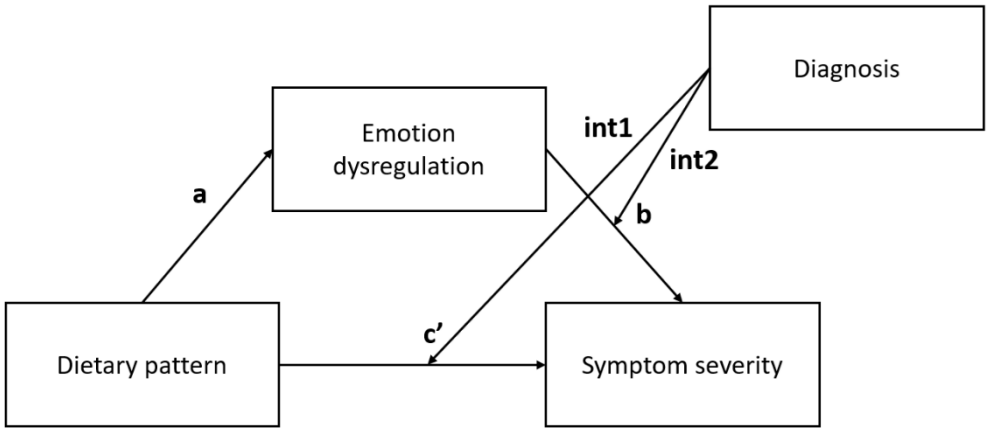
Figure 2

Moderated Mediation Models Tested in This Study



Note. Dietary pattern meant Mediterranean diet or sources of omega-3 PUFAs. Emotion dysregulation meant the general score of the Difficulties in Emotion Regulation Scale. Symptom severity meant severity of borderline personality disorder (BPD) symptoms or severity of eating disorders (ED) symptoms. Diagnosis meant BPD or ED diagnosis.

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Table 1

Group Characteristics Including Between-group Differences Based on ANOVA with Bonferroni Correction and Kruskal–Wallis test with Dunn–Bonferroni Post-hoc Test

Variable	BPD (n = 40)		BPD+ED (n = 37)		ED (n = 22)		HC (n = 37)		F or H	η^2	Post hoc
	M	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*			
Age	26.27	7.37	24.03	5.61	28.45	7.31	27.35	6.10	F = 2.58	.06	ns
BMI	26.29	6.05	23.69	8.01	21.37	7.69	21.24	2.24	H = 23.24***	.15	BPD > ED, HC, BPD+ED
WHR	0.78	0.06	0.77	0.06	0.76	0.05	0.74	0.04	H = 14.02**	.08	BPD > HC, ED, BPD+ED
Heart rate	84.20	11.64	76.07	9.82	72.36	11.02	76.32	9.89	F = 7.35***	.14	BPD > BPD+ED, HC
Systolic blood pressure	118.93	13.56	112.96	13.88	112.75	14.13	114.57	8.89	F = 1.84	.04	ns
Diastolic blood pressure	71.09	10.47	70.00	11.82	70.04	10.49	69.68	8.62	F = 0.13	.003	ns
Pulse pressure	47.84	9.06	42.96	6.18	42.70	5.25	44.89	4.32	F = 4.45**	.09	BPD > BPD+ED

Note. BPD = women diagnosed with borderline personality disorder; BPD+ED = women diagnosed with comorbid borderline personality disorder and eating disorders; ED = women diagnosed with eating disorders; HC = healthy controls; BMI = body mass index; WHR = waist-hip ratio.

** $p < .01$. *** $p < .001$. ns = not statistically significant.

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Table 2

Between-group Differences in Symptoms Severity and Emotion Dysregulation Based on ANOVA with Bonferroni Correction and Kruskal–Wallis test with Dunn–Bonferroni Post-hoc Test

Variable	BPD (n = 40)			BPD+ED (n = 37)			ED (n = 22)			HC (n = 37)			F or H	η^2	Post hoc
	M	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*			
BPD: Total score	128.6	31.2	129.0	136.9	28.8	139.0	95.6	26.5	96.5	64.4	13.7	60.0	H = 81.75***	.60	BPD, BPD+ED > ED > HC
BPD: Impulsivity	14.7	5.3	13.5	16.9	5.1	17.0	12.9	3.5	11.5	10.2	1.6	10.0	H = 49.89***	.36	BPD, BPD+ED, ED > HC; BPD+ED > ED
BPD: Anger	9.1	3.8	9.0	9.9	3.6	10.0	6.6	2.7	6.0	5.2	1.0	5.0	H = 48.59***	.35	BPD, BPD+ED > ED, HC
EAT-26	12.70	11.79	10.0	36.43	17.79	42.0	33.73	15.89	36.5	4.16	4.06	3.0	H = 74.32***	.53	BPD+ED, ED > HC, BPD
HADS: Anxiety	15.1	4.4	15.5	15.1	4.4	15.0	12.5	5.1	14.0	6.5	3.2	6.0	F = 34.98***	.45	BPD, BPD+ED, ED > HC
HADS: Depression	11.6	5.0	11.0	10.1	5.3	10.0	8.7	4.7	9.0	2.4	2.1	2.0	H = 59.24***	.43	BPD, BPD+ED, ED > HC
DERS	123.40	21.69	125.0	130.30	17.98	131.0	109.23	24.29	108.5	76.65	19.73	75.0	F = 49.61***	.53	BPD, BPD+ED, ED > HC; BPD+ED > ED

Note. BPD = women diagnosed with borderline personality disorder; BPD+ED = women diagnosed with comorbid borderline personality disorder and eating disorders; ED = women diagnosed with eating disorders; HC = healthy controls. DERS = total score of the Difficulties in Emotion Regulation Scale. EAT-26 = total score of the Eating Attitudes Test. BPD = the Borderline Personality Disorder Checklist. HADS = the Hospital Anxiety and Depression Scale.

*** $p < .001$.

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Table 3

Between-group Differences in Consumption Frequency (Times/Day) Based on ANOVA with Bonferroni Correction and Kruskal–Wallis test with Dunn–Bonferroni Post-hoc Test

Food group	BPD (n = 40)			BPD+ED (n = 37)			ED (n = 22)			HC (n = 37)			F or H	η^2	Post hoc
	M	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*			
Sugar, sweets, and snacks	1.8	1.4	1.3	1.8	1.8	1.6	1.4	1.5	1.0	1.4	1.4	0.9	$H = 2.42$	.004	ns
Milk, fermented milk beverages, and cottage cheese	1.0	0.8	0.8	1.0	1.0	0.7	1.2	1.0	1.1	1.1	0.7	1.1	$F = 0.45$	.01	ns
Sweetened dairy products	0.4	0.6	0.1	0.4	0.7	0.1	0.4	0.5	0.1	0.2	0.3	0.1	$H = 2.30$	.005	ns
Cheese	0.3	0.3	0.1	0.3	0.5	0.1	0.3	0.3	0.1	0.5	0.4	0.6	$H = 13.67$**	.08	BPD+ED. ED < HC
Eggs and egg-based dishes	0.3	0.3	0.1	0.2	0.3	0.1	0.2	0.3	0.1	0.5	0.3	0.6	$F = 5.16$**	.11	BPD+ED < HC
Breakfast cereals	0.3	0.4	0.1	0.3	0.4	0.1	0.2	0.3	0.1	0.1	0.3	0	$H = 2.24$	.005	ns
Whole grain cereal products	0.7	0.7	0.6	0.9	0.9	0.7	1.0	0.9	0.8	1.0	0.6	1.1	$F = 1.10$	.02	ns
Refined cereal products	0.8	0.6	0.7	0.7	0.7	0.7	0.8	0.8	0.6	0.7	0.5	0.7	$F = 0.14$	.003	ns
Butter and cream	0.7	0.6	0.6	0.4	0.6	0.1	0.5	0.5	0.1	0.5	0.6	0.6	$H =13.95$**	.08	BPD+ED < BPD
Other animal fats	0.02	0.09	0	0.03	0.2	0	0.01	0.02	0	0.002	0.01	0	$H = 2.06$	.007	
Vegetable fats	0.4	0.3	0.1	0.3	0.4	0.1	0.3	0.4	0.1	0.8	0.5	0.6	$H =$ 24.04***	.16	BPD. BPD+ED. ED < HC
Margarine, mayonnaise, and dressings	0.3	0.4	0.1	0.2	0.4	0.03	0.2	0.5	0.02	0.2	0.3	0.1	$H = 3.86$	.007	ns
Fruits	0.7	0.6	0.6	0.9	0.7	0.6	1.0	0.6	0.8	1.1	0.7	1.0	$H = 8.35$*	.04	BPD < HC
Vegetables (excluding potatoes)	0.9	0.7	1.0	1.0	0.8	1.0	1.1	0.7	1.0	1.3	0.6	1.0	$F = 2.11$	.05	ns
Dried and processed legumes	0.1	0.2	0.03	0.3	0.4	0.1	0.2	0.4	0.1	0.3	0.3	0.1	$H = 9.19$*	.05	BPD < HC
Potatoes	0.3	0.3	0.1	0.3	0.3	0.3	0.3	0.4	0.1	0.2	0.2	0.1	$H = 0.66$	.02	ns
Nuts and seeds	0.3	0.4	0.2	0.5	0.7	0.1	0.6	0.9	0.2	0.7	0.5	0.7	$H = 11.82$**	.07	BPD. BPD+ED < HC
Processed meat products	0.5	0.6	0.2	0.5	1.2	0	0.3	0.7	0	0.5	0.6	0.2	$H = 10.09$*	.05	ns
Red meat and game	0.2	0.4	0.03	0.2	0.7	0	0.03	0.05	0	0.2	0.2	0.1	$H =19.23$***	.12	BPD+ED. ED < HC

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Food group	BPD (n = 40)			BPD+ED (n = 37)			ED (n = 22)			HC (n = 37)			F or H	η^2	Post hoc
	M	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*	*M*	*SD*	*Mdn*			
White meat	0.2	0.3	0.1	0.1	0.4	0.02	0.1	0.2	0.03	0.3	0.3	0.1	H = 10.71*	.06	BPD+ED < HC
Fish	0.1	0.2	0.1	0.2	0.7	0.1	0.1	0.1	0.1	0.2	0.3	0.1	H = 13.37**	.08	BPD+ED < HC
Fruit and vegetable juices	0.4	0.4	0.1	0.5	1.0	0.1	0.3	0.5	0.1	0.3	0.4	0.1	H = 4.86	.01	ns
Sugar-sweetened and energy drinks	0.5	0.7	0.1	0.5	0.7	0.1	0.3	0.4	0.04	0.1	0.4	0.03	H = 11.50**	.06	BPD > HC
Alcohol	0.3	0.4	0.1	0.2	0.5	0.1	0.1	0.2	0	0.2	0.3	0.2	H = 19.79***	.13	BPD. HC > ED; BPD+ED < HC
Sources of omega-3 PUFAs	0.8	0.5	0.8	1.0	1.4	0.4	1.0	1.2	0.6	1.7	0.9	1.7	H = 23.99***	.16	BPD < BPD+ED. ED. HC
Mediterranean diet	5.1	1.8	5.0	5.7	4.3	3.9	6.2	3.3	5.0	7.7	2.6	7.4	H = 19.66***	.13	BPD. BPD+ED < HC
Anti-Mediterranean diet	3.3	2.0	3.0	3.2	3.7	1.8	2.5	2.5	2.3	2.7	2.0	2.1	H = 4.33	.01	ns

Note. BPD = women diagnosed with borderline personality disorder; BPD+ED = women diagnosed with comorbid borderline personality disorder and eating disorders; ED = women diagnosed with eating disorders; HC = healthy controls; PUFAs = polyunsaturated fatty acids; Anti-Mediterranean diet = total consumption frequency of products that should be consumed only occasionally in the Mediterranean diet.

* p < .05. ** p < .01. *** p < .001. ns = not statistically significant

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Table 4

Results of Mediation and Moderated Mediation Models of Relationship Between Dietary Patterns and Symptoms of Borderline Personality Disorder

Model	Effect	*B*	β/R2ch	*SE*	*t*	*p*	BootCI/CI
A	a	−9.26	−0.33	2.25	−4.11	< .001	[−13.72; −4.81]
	b	1.01	0.77	0.08	12.69	< .001	[0.86; 1.17]
	ab	−9.38	−0.26	2.79	3.36		[−15.41; −4.46]
	c'	1.23	0.03	2.21	0.56	.578	[−3.14; 5.60]
	c	−8.15	−0.22	3.08	−2.64	.009	[−14.24; −2.05]
B	Int_1	0.58	0.0001	4.06	0.14	.887	[−7.46; 8.61]
	Int_2	−0.09	0.0007	0.18	−0.51	.614	[−0.45; 0.27]
C	a	−2.50	−0.27	0.77	−3.46	.001	[−4.01; −0.98]
	b	0.97	0.73	0.08	12.48	< .001	[0.82; 1.12]
	ab	−2.42	−0.20	0.79	3.06		[−4.05; −0.98]
	c'	−0.95	−0.08	0.72	−1.32	.189	[−2.36; 0.47]
	c	−3.37	−0.28	1.01	−3.33	.001	[−5.37; −1.37]
D	Int_1	0.27	0.0001	1.35	0.20	.840	[−2.39; 2.94]
	Int_2	−0.08	0.0005	0.18	−0.42	.675	[−0.43; −0.28]

Note. N = 136. Model A = mediation model of the relationship between omega-3 PUFAs and BPD symptom severity. Model B = Mediation from Model A moderated by BPD diagnosis (1 = yes, 0 = no). Model C = mediation model of the relationship between Mediterranean diet and BPD symptom severity. Model D = Mediation from Model C moderated by BPD diagnosis (1 = yes, 0 = no). BootCI = 95% Bootstrap Confidence Interval with 10,000 samples. CI = confidence interval. a = effect of dietary pattern on mediator variable. b = effect of mediator on symptom severity when dietary pattern was controlled. c = total effect of dietary pattern on symptom severity. c’ = direct effect of dietary pattern on symptom severity when mediator was controlled. ab = indirect effect. Int_1 = interaction between dietary pattern and moderator (BPD diagnosis). Int_2 = interaction between mediator (emotion dysregulation) and moderator (BPD diagnosis).

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Table 5

Results of Mediation and Moderated Mediation Models of Relationship Between Dietary Patterns and Symptoms of Eating Disorders (EDs)

Model	Effect	*B*	β/R2ch	*SE*	*t*	*p*	BootCl/CI
E	a	−9.23	−0.33	2.26	−4.09	< 0.001	[−13.71; −4.76]
	b	0.30	0.47	0.05	6.01	< 0.001	[0.20; 0.40]
	ab	−2.75	−0.16	0.93	2.96		[−4.87; −1.23]
	c'	−2.47	−0.14	1.37	−1.80	0.074	[−5.18; 0.24]
	c	−5.22	−0.30	1.45	−3.59	< 0.001	[−8.10; −2.35]
F	Int_1	−5.75	0.03	2.14	−2.68	0.008	[−9.98; −1.51]
	Int_2	0.04	0.001	0.08	0.46	0.645	[−0.13; 0.21]
	c' no-ED	1.18		1.77	0.67	0.506	[−2.32; 4.68]
	c' ED	−4.57		1.21	−3.79	< 0.001	[−6.95; −2.18]
G	a	−2.49	−0.27	0.77	−3.23	0.002	[−4.01; −0.96]
	b	0.32	0.50	0.05	6.51	< 0.001	[0.23; 0.42]
	ab	−0.79	−0.14	0.28	−2.82		[−1.42; −0.30]
	c'	−0.29	−0.05	0.45	−0.63	0.527	[−1.18; 0.61]
	c	−1.08	−0.18	0.50	−2.17	0.032	[−2.07; −0.10]
H	Int_1	−0.65	0.003	0.72	−0.89	0.374	[−2.08; 0.79]
	Int_2	0.10	0.004	0.09	1.11	0.270	[−0.08; 0.27]

Note. N = 136. Model E = mediation model of the relationship between omega-3 PUFAs and EDs symptom severity. Model F = Mediation from Model E moderated by EDs diagnosis (1 = yes, 0 = no). Model G = mediation model of the relationship between Mediterranean diet and EDs symptom severity. Model H = Mediation from Model G moderated by EDs diagnosis (1 = yes, 0 = no). BootCI = 95% Bootstrap Confidence Interval with 10,000 samples. CI = confidence interval. a = effect of dietary pattern on mediator variable. b = effect of mediator on symptom severity when dietary pattern was controlled. c = total effect of dietary pattern on symptom severity. c’ = direct effect of dietary pattern on symptom severity when mediator was controlled. ab = indirect effect. Int_1 = interaction between dietary pattern and moderator (EDs diagnosis). Int_2 =

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interaction between mediator (emotion dysregulation) and moderator (EDs diagnosis).

No-ED = women without EDs

diagnosis. ED = women with EDs diagnosis

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